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ERP correlates of complex human decision making in a gambling paradigm: Detection and resolution of conflict

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Abstract

The present event-related potential study investigated the correlates of decision making in relation to the amount of response conflict. In a gambling paradigm, response conflict was introduced by giving participants the option to either gamble or pass. Second, the odds and gains in each trial were manipulated to make the decision to gamble or pass determined or underdetermined. Underdetermined trials included an extra conflict. The N2 was modulated by the mere presence of conflict. In contrast to both conflict monitoring and inhibition theories for N2, these results suggest that an enhancement in N2 reflects the mere detection of conflicting alternatives. The P3 showed a fronto-central increase in amplitude in trials including two forms of response conflict compared to trials including only one conflict. These findings suggest that P3 reflects part of the conflict resolution processes.

Descriptors: N2, P3, Conflict, Normal volunteers, Prefrontal

Making a decision requires monitoring and integrating potentially conflicting information (Krawczyk, 2002). Some situations show high response conflict, as the tendency for a prepotent but incorrect response has to be overridden by the correct response (Botvinick, Braver, Barch, Carter, & Cohen, 2001; Ridderinkhof, Ullsperger, Crone, & Nieuwenhuis, 2004). Response conflict can also occur when one has to make an underdetermined choice between equally compelling responses that activate different incompatible response pathways (Botvinick et al., 2001). The brain region most commonly associated with monitoring conflict is the anterior cingulate cortex (ACC; Bush, Luu, & Posner, 2000; Carter et al., 1998; van Veen & Carter 2002a). This region is assumed to signal lateral prefrontal cortical areas to engage cognitive control in order to reach a final decision (Cohen, Botvinick, & Carter, 2000; Kerns et al., 2004; MacDonald, Cohen, Stenger, & Carter, 2000). However, the translation of conflict monitoring into adjustments in cognitive control is not well understood (Botvinick, Cohen, & Carter, 2004).

Conflict and the implementation of control are commonly studied with fMRI. The coarse time resolution of this imaging technique makes it difficult, however, to study online adjustments in cognitive control. Some studies tried to solve this by taking the preceding trial into account (Botvinick, Nystrom, Fissell, Carter, & Cohen, 1999; Carter et al., 2000; Kerns et al., 2004). Others chose the high temporal resolution provided by event-related brain potential (ERP) measurements (Nieuwenhuis, Yeung, van den Wildenberg, & Ridderinkhof, 2003; van Veen & Carter 2002b). However, most studies so far manipulated the presence of conflict in an all-or-nothing manner, by using conditions that generate either little or high amounts of conflict. A more direct link between signal modulation and conflict can be obtained by manipulating the amount of conflict gradually. Such a manipulation is more consistent with the conflict monitoring theory for ACC that assumes increasing ACC activity in relation to increasing conflict (Barch, Braver, Sabb, & Noll, 2000; Botvinick et al., 2001).

Here we manipulate the amount of conflict by superimposing conflict related to the appropriateness of the prepotent response on conflict resulting from the underdeterminedness of responding. These manipulations were applied within a gambling paradigm aimed at winning as many points as possible by guessing the location of a hidden token. To generate a first type of response conflict, participants were given the opportunity not to gamble (i.e., to pass), which requires the need to override the dominant, because rewarding, gambling response. A second type of response conflict was introduced by manipulating the attractiveness of the trial. This made the choice to gamble or pass either determined or underdetermined. The choice is determined in highly attractive (high odds and gains) and unattractive (very low gains) trials, because the appropriate response is indicated by the

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stimulus (the gains and odds). In trials with intermediate odds and gains, on the other hand, both the gamble and pass responses are coactivated, making the choice for a response underdetermined. If a participant then decides to pass, conflict inherent to overriding the dominant gambling response will be superimposed on conflict elicited by the underdeterminedness of the trial.

Most studies that used gambling paradigms studied aspects of feedback and reward processing (e.g., Dunning & Hajcak, 2007; Elliott, Friston, & Dolan, 2000; Gehring & Willoughby, 2002; Hajcak, Moser, Holroyd, & Simons, 2007; Hewig et al., 2007; Yeung, Holroyd, & Cohen, 2005; Yeung & Sanfey, 2004). The decision phase itself was studied by few and only with fMRI (Ernst et al., 2004; Rogers et al., 1999, 2004). Here we measure the electrophysiological activity of the brain during decision making to disentangle neural processes related to conflict.

Methods

Participants

Data were recorded from 10 right-handed participants. All participants had normal or corrected-to-normal vision, were free of neurological or psychiatric disorders, and gave written informed consent. Due to technical problems, data from 2 participants were excluded from the analyses. Therefore analyses were restricted to 8 participants (age range: 19–35 years; 4 women). After a practice run of 20 trials, all participants completed nine runs of the gambling paradigm, each comprising 50 trials. After we pruned the data for EEG analysis, this resulted in an average of 430 trials ($SD = 14$) per participant.

Gambling Paradigm

Paradigm. Participants were asked to engage in a computer game and motivated to earn as many points as possible. Each trial started with a central stimulus consisting of a horizontal bar divided into two colored parts, each side indicating the probability of an imaginary token being hidden underneath (e.g., 30% blue–70% yellow; see Figure 1). The proportions of each colored part in relation to the total bar could range from 5%–95% to 50%–50%. Participants could choose the side on which they thought the token was hidden by pressing the corresponding left or right response button with their respective hand. Points could be won or lost depending on the correctness of the participants' guess. The number of points that could be won was indicated above the bar and was randomly chosen on each trial ranging from 10 to 100 points. The amount of points that could be lost was shown below the bar and was systematically linked to the proportional division of the bar. The most ambiguous proportions (50%–50%) were coupled with the highest losses (100, 90, or 80 points). To win as many points as possible, participants had to make gambles. However, on each trial they were given the opportunity to opt out of gambling (i.e., to pass) whenever they felt insecure about the trial. All they had to do was withhold the key-press response and wait until the stimulus disappeared after 4 s. Passing resulted in a small 20-point reward. After each trial participants were informed about the result of that trial, and their total score was updated and shown. The probability of winning or losing was coupled to the proportional division of the bar. All participants were given 100 points to start with and were verbally motivated to gather as many points as possible.

We ensured that enough pass trials were gathered by implementing three task features. First, we covertly manipulated the real probability of winning on a trial (as opposed to the proportional division of the bar) depending on the number of gambles a participant made before that trial. We wanted at least 33% of all trials to be pass trials. Therefore, when a gamble was made in more than 66% of the preceding trials, the actual probability of winning on the current trial was covertly decreased, thus increasing the chance of losing in order to discourage the participant from gambling on the next trials. On the other hand, when a participant gambled in less than 33% of the trials (i.e., passing too much), the probability of winning on the current trial was covertly increased to encourage the participant to gamble on the next trials. More specifically, this manipulation was achieved by multiplying the size of the chosen side with the ratio between the observed percentage of passing and the wanted percentage of passing (i.e., 33%). As such, passing in more than 33% of the preceding trials results in a ratio exceeding 1; this in turn results in a virtual increase of the chosen side. Accordingly, passing in less than 33% of the preceding trials results in a ratio smaller than 1, decreasing the size of the chosen side upon multiplication. Second, when participants gambled, the next stimulus was made

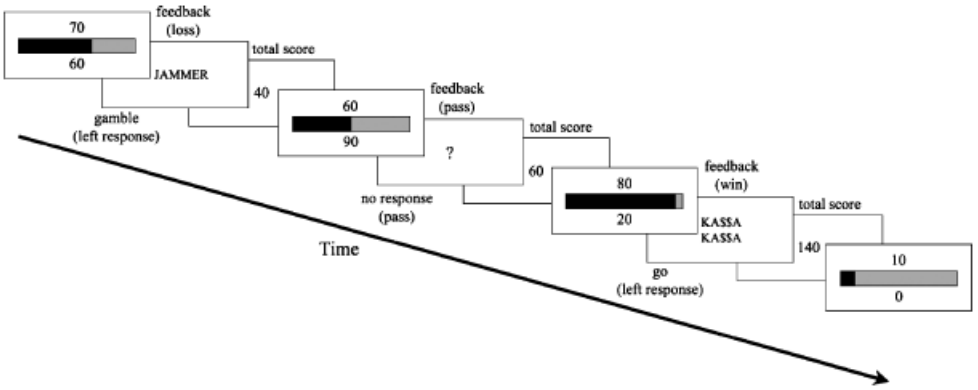

Figure 1. Stimulus sequence of the gambling paradigm. On each trial participants were shown a proportionally divided colored bar (blue and yellow). Participants could gamble on the location of a hidden token. A correct gamble was rewarded the points shown above the bar; an incorrect gamble resulted in a loss of the points shown below the bar. Participants could also choose not to gamble and settle for a small reward (20 points), as shown in Trial 2 of this sequence. After each stimulus bar feedback was given and the total score was updated and shown. (Jammer: "Too bad"; Ka$$a Ka$$a: "ka-ching").

more unfavorable by decreasing the proportional division of the stimulus bar (except on trials with a 50% yellow–50% blue bar). On the other hand, when no gamble was made the next stimulus was made more favorable by increasing the proportional division of the bar (e.g., 60%–40% to 80%–20%). Third, the pass reward was set to 20 points and participants were clearly instructed that "on some trials it is best to pass." This proved very effective in ensuring an acceptable number of pass trials. In pilot runs with a pass reward of only 10 points and no emphasis on the option to pass, the number of inhibitions was about 10% (results for these runs are not shown here). This increased to 40% after including the higher reward and specific instructions. Only the instruction feature was known by the participants. They were not informed about the first (manipulated chance of winning) and second (better/worse next trial) features.

Although there are no instructions emphasizing a speeded response, making a gamble is considered the dominant response in this paradigm. First, participants have to gamble in order to get the biggest rewards. Second, the pilot runs showed only 10% inhibitions when the option to pass was not stressed during the instructions and the reward for passing was limited to 10 points, illustrating the participants' urge to gamble in the presented paradigm. A similar urge to respond is also hypothesized in the go trials of a go/no-go task, even when they are less frequent compared to no-go trials (Nieuwenhuis et al., 2003). Finally, verbal comments during the task by participants who chose to gamble in a trial where only 10 or 20 points could be won indicated that their response was impulsive and that they would rather have passed in that trial (e.g., "Oops, I should have let that one pass!").

Trial grouping. Based on our paradigm and the conflict theory of Botvinick (Botvinick et al., 2001), we dissociated four trial groups. We dissociated trials that always lead participants to a gamble, trials that always lead to a pass, trials that sometimes resulted in a gamble, and finally trials that sometimes resulted in a pass. The behavior of the participants in the different trials was used to delineate these groups more precisely.

As expected the behavioral results confirmed the existence of trials that always resulted in a gamble response (see Figure 2). These trials are characterized by a favorable proportional division of the stimulus bar and a gain over 20 points. Based on the behavioral results, we included all trials with a proportion of 80% or higher and a gain exceeding 20 points in this trial condition. These trials were labeled GO trials and do not elicit a response conflict because the proportional division ($\geq$80%) and points shown on the stimulus ($\geq$30) are clear incentives toward the dominant gamble response. The second condition included the trials with a gain of only 10 or 20 points, regardless of the proportional division of the colored bar. In these trials gambling was always disadvantageous, as participants were certain of a 20-point gain when passing. These trials elicit conflict by clearly signaling the participant to override the dominant gamble response. Therefore these trials were labeled NOGO trials. Figure 2 shows that participants did indeed refrain from gambling in these trials, as only 11% lead to a gamble (these trials were not included in any analysis). Finally, we expected some trials to be more difficult in deciding on the most favorable response, as the stimulus shown in these trials was not clearly pro gambling, with a high proportion and large gain, or pro inhibition, with a gain of only 10 or 20 points. These trials are underdetermined, as it is not immediately clear to the participants which response should be given (Botvinick et al., 2001). The final decision regarding the most appropriate response in such trials will be guided by the interpretation of different kinds of information such as previous experiences, the gain or gain/loss ratio, the total score, or the moment during the task (e.g., at the beginning or near the end). These trials included trials with a proportional division between 50% and 75% and a gain exceeding 20 points (Figure 2a, gray zone). Based on the chosen response, both a GAMBLE and a PASS trial condition were defined. As such, an identical trial was sometimes categorized as GAMBLE but other times as PASS depending on the decision of the participant. In both these trial groups response conflict is elicited due to the trial being underdetermined. Moreover, compared to the GAMBLE trials, which result in the dominant gambling response, the PASS trials included an additional conflict, as these involve the decision to override the dominant gambling response.

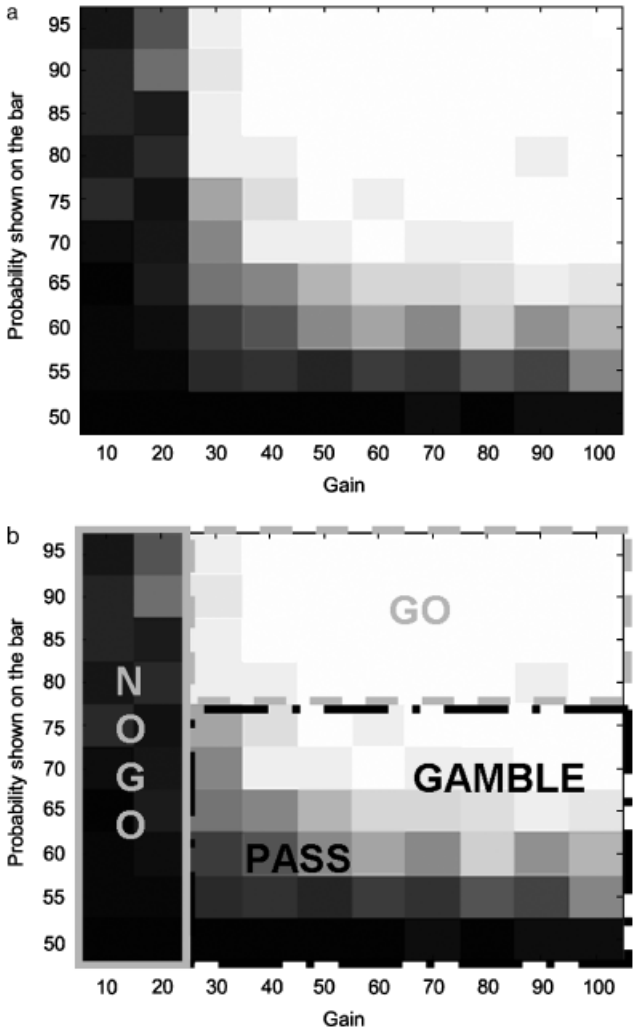

Figure 2. Graphical representation of the action of the participants in each typical trial (a). Part b shows the subsequent trial grouping. Darker squares indicate a higher percentage of inhibitions in that trial type (black = 100% pass); lighter squares indicate a higher percentage of gambles in that trial type (white = 100% gamble). Trials are ordered according to proportional division of the stimulus bar (Y-axis) and the amount of points that could be won (X-axis). *N* trials: GO: 817; NOGO: 596; GAMBLE: 1019; PASS: 920.

To summarize, our paradigm included trials without a response conflict (GO), trials with conflict related to the inappropriateness of the dominant response (NOGO), trials eliciting

conflict by being underdetermined (GAMBLE), and finally trials eliciting conflict by being underdetermined and by the inappropriateness of the dominant response (PASS).

Electrophysiological Recordings

We recorded the electroencephalogram (EEG) using 19 Ag/AgCl electrodes applied to the scalp of the participants using the standard 10-20 system of electrode placement: Fp1, Fp2, F3, F4, F7, F8, C3, C4, T3, T4, P3, P4, T5, T6, O1, O2, Fz, Cz, and Pz (Schwarzer GmbH, Germany). A ground electrode was placed on the forehead above the nose. Additionally two electrodes were placed on the outer canthi (HEOG) and two above and below the right eye (VEOG) to detect horizontal and vertical eye movements. All electrodes were referenced to physically linked left and right mastoids and all electrode impedances were kept below 5 kΩ. Sampling rate was 1000 Hz with an analog pass band of 0.095–70 Hz. Data were filtered off-line using a 30-Hz digital low-pass filter. Off-line analysis of the data, including removal of eye movement artifacts, was performed using the EEGLAB v4.515 toolbox (Delorme & Makeig, 2004) under Matlab v7.0 (Mathworks, Natick, MA). EEG epochs of 1500 ms synchronized on the onset of the gambling stimulus were extracted off-line with an additional 400 ms baseline. Eye movement artifacts were removed from the data using ICA (Jung et al., 2000).

Specific analysis methods are indicated in the results section. Geisser–Greenhouse corrections were used when appropriate.

Results

Behavioral Data

As expected, behavioral results showed that participants were significantly slower in the GAMBLE compared to the GO trials, 1016 versus 781 ms, $t(7) = 7.81$, $p < .001$, supporting the difference in the amount of conflict and difficulty between these two trial conditions. There are no reaction time data for trials resulting in a pass, as no response had to be made in these trials.

ERP Data

Visual inspection of the grand-average ERP waveforms shown in Figure 3 showed an enhanced frontal negativity, N2, around 280 ms after stimulus onset in all but the GO trials. A repeated measures ANOVA with conditions and electrodes (all frontal and central electrodes were included) as within-subject variables for the mean amplitude between 240 and 320 ms after stimulus onset yielded a main effect of condition, $F(3,21) = 4.05$, $p < .05$, $\varepsilon = .61$. Post hoc comparisons confirmed that there was no difference in N2 between the NOGO, PASS and GAMBLE trials, $p > .17$. However, these trials all elicited a more negative N2 compared to the GO trials, GO vs.versus NOGO: $F(1,7) = 3.61$, $p = .09$; GO versus GAMBLE: $F(1,7) = 6.1$, $p < .04$; GO versus PASS: $F(1,7) = 7.7$, $p < .03$. A significant Conditions × Electrodes interaction indicated that the difference between conditions was maximal at electrodes Fz, F4, and Fp2, $F(33,231) = 3.32$, $p < .01$, $\varepsilon = .16$. At Fz, all comparisons between each condition and the GO trials reached significance, GO versus NOGO: $F(1,7) = 6.24$, $p < .04$; GO versus GAMBLE: $F(1,7) = 8.14$, $p < .02$; GO versus PASS: $F(1,7) = 7.64$, $p < .03$. Figure 4 shows headplots for the NOGO, GAMBLE and PASS trials at 270 ms after stimulus onset.

The P3 peak observed between 400 and 500 ms after stimulus onset also showed condition related modulations (Figure 3). The largest differences were observed at the fronto-central electrodes. For the statistical analyses P3 was quantified as the mean amplitude between 340 and 550 ms after stimulus onset. A repeated measures ANOVA with conditions and the three midline electrodes (Fz, Cz, and Pz) as within-subject variables yielded main effects of electrodes, $F(2,14) = 16.71$, $p < .01$, $\varepsilon = .99$, and conditions, $F(3,21) = 5.26$, $p < .02$, $\varepsilon = .69$, and a significant Conditions × Electrodes interaction, $F(6,42) = 11.13$, $p < .01$,

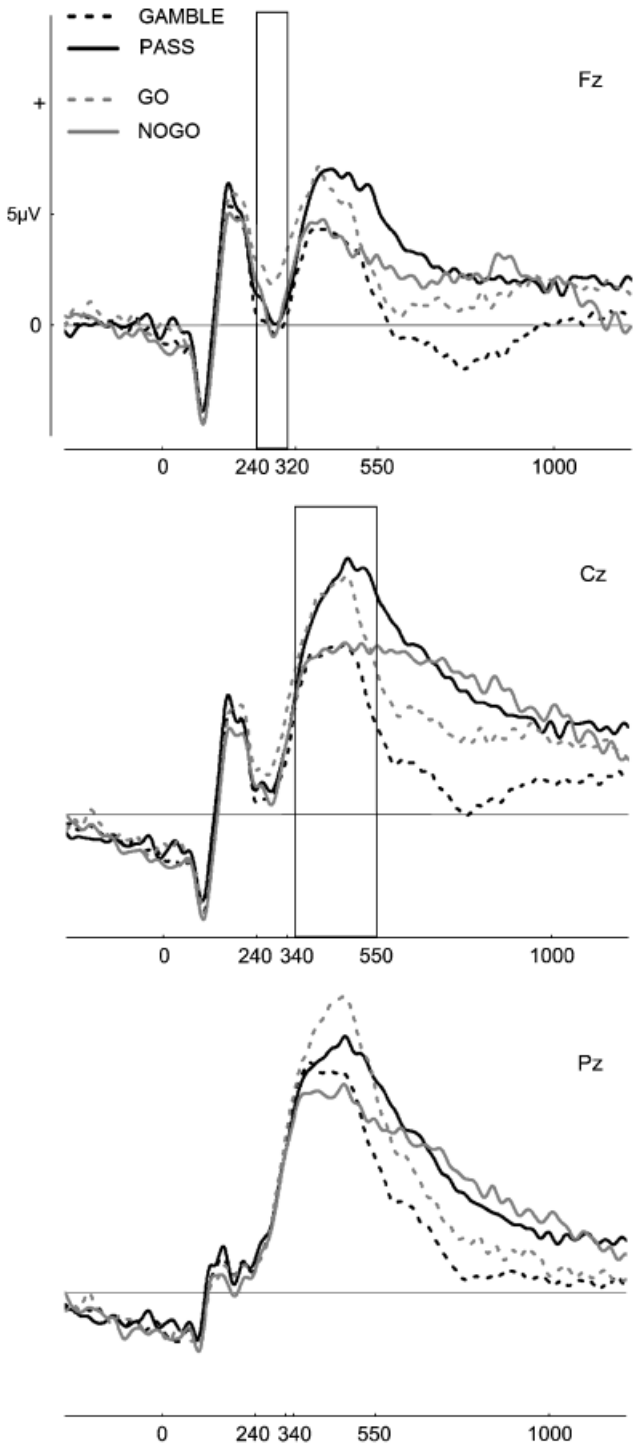

Figure 3. Grand-average ERP waveforms for each of the four trial conditions at the midline electrodes Fz, Cz, and Pz. At Fz the window for measuring the N2 peak is shown. At Cz the window for measuring P3 is shown. The X-axis shows the time in milliseconds relative to the onset of the gamble stimulus.

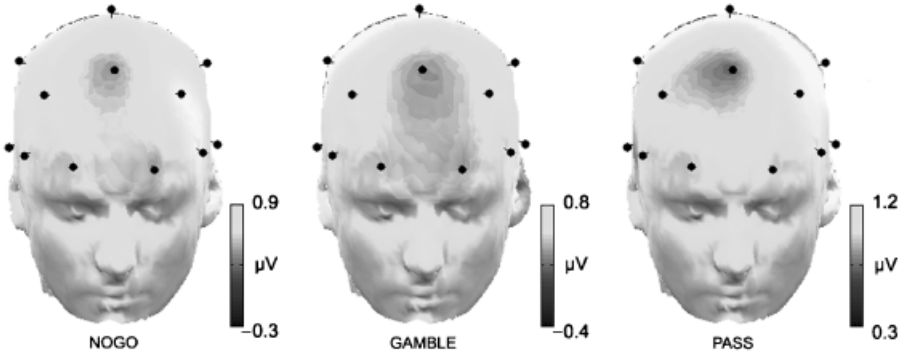

Figure 4. Scalp topographies for the N2 peak in the NOGO, GAMBLE and PASS condition. These head plots show the amplitude at 270 ms after onset of the gamble stimulus.

$\varepsilon = .49$. Post hoc pairwise comparisons (Tukey test, all significant p values $< .001$) revealed three main findings. First, there were no differences between the P3 amplitude of the NOGO and GAMBLE trials at the three midline electrodes, whereas both these conditions produced smaller amplitudes at each electrode compared to the PASS and GO trials. Second, the PASS trials showed a significantly higher P3 amplitude than the GO trials at electrode Fz. The same trend, albeit not significant, was observed at Cz ($p = .09$). However, the difference was reversed at Pz, with GO trials showing a higher P3 amplitude compared to the PASS trials. Third, the NOGO, GAMBLE and GO trials all showed an $Fz < Cz < Pz$ effect. This effect was not observed in the PASS trials; instead these trials showed an $Fz < Cz = Pz$ distribution. In addition, scalp maps of the difference in amplitude between the PASS and NOGO, GAMBLE, and GO trials, respectively, highlighted the higher fronto-central amplitude in the PASS trials compared to the three other trial conditions (see Figure 5).

Discussion

Our gambling paradigm and the high temporal resolution of ERPs enabled us to disentangle modulations related to the amount of conflict involved in decision making. An equally enhanced N2 was found in all conditions that included some form of response conflict. In contrast, an increase in the amplitude of P3 at the frontal and central electrodes was observed when including an extra response conflict compared to including only one response conflict.

In ERP studies, an N2 negativity is traditionally found in the no-go or inhibition trials of go/no-go and stop tasks, where it is thought to reflect a frontal inhibition process (Falkenstein, Hoormann, & Hohnsbein, 1999). However, recent studies reported an N2 also in infrequent go trials. This led to an alternative interpretation of N2, linking it also to response conflict as compared to mere response inhibition (Donkers & Van Boxtel, 2004; Nieuwenhuis et al., 2003). In line with this interpretation, we observed an enhanced N2 in all trials that included conflict either by requiring the participants to override the dominant gambling response (NOGO), by being underdetermined (GAMBLE), or by featuring both these conflicts (PASS), as compared to GO trials, which included no conflict. The equal enhancement of N2 in both the GAMBLE and NOGO trials indicates that there is no effect of the type of conflict on N2. The absence of an extra enhancement in the PASS trials, which feature both types of conflict superimposed, suggests that N2 is also not affected by the amount of conflict. The enhanced N2 in trials including conflict, without any modulation by either type or amount of conflict, warrants the conclusion that the observed enhancement in our N2 reflects the mere occurrence of conflict. Furthermore, the finding that GAMBLE trials, which do not result in inhibition but in a gamble response, equally enhanced the N2 confirms the recent hypothesis that N2 does not reflect mere response inhibition (Donkers & Van Boxtel, 2004; Nieuwenhuis et al., 2003). However, our finding that the N2 enhancement was unaffected by the amount of conflict contradicts the conflict monitoring theory of ACC, which states that increased conflict should lead to an increase in ACC activity (Barch et al., 2000; Botvinick et al., 2001).

The scalp distributions of N2 point to a medial-frontal source contributing to N2 in all three conflict conditions (Figure 4). Although these scalp distributions do not necessarily reflect the exact location of its underlying sources, the central position of the focus is compatible with a source in the medial prefrontal cortex. ERP source localization studies indeed identified ACC as a likely generator of the frontal N2 peak (Bekker, Kenemans, & Verbaten, 2005; Nieuwenhuis et al., 2003). However, the fact that the enhancement in N2 was also visible albeit weaker at the right hemisphere electrodes F4 and Fp2 suggests that other sources are contributing to our N2. For instance, sources in lateral orbitofrontal cortex might also contribute, supporting the hypothesis

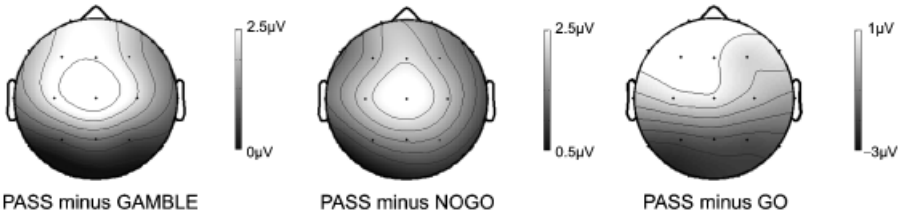

Figure 5. Scalp topographies for the difference in P3 amplitude between the PASS and GAMBLE (left), PASS and NOGO (middle), and PASS and GO (right) conditions. Plots show the difference between condition means across the time window of the P3 peak.

that N2 is linked to response inhibition (Bokura, Yamaguchi, & Kobayashi, 2001).

In light of the results described above, our N2 does not reflect pure response inhibition or conflict monitoring as defined by Botvinick et al. (2001). Instead our results suggest that N2 is related to the mere detection of conflicting alternatives, possibly reflecting an initial, premotor inhibition process (Falkenstein, 2006). Such a process could mean no more than the detection of the presence of conflicting alternatives, guiding decisions of whether it is worth acting or not (Rushworth, Walton, Kennerley, & Bannerman, 2004).

The second major finding of this study is that the fronto-central amplitude of P3 varied with the amount of conflict present in a trial. The GAMBLE and NOGO trials both included only one type of response conflict. In the NOGO trials conflict was elicited by the inappropriateness of the dominant response and in the GAMBLE trials by the underdeterminedness of response selection. These two types of trials resulted in equal P3 amplitudes. As for N2, this result shows that the type of conflict included in a trial did not matter. The PASS trials, on the other hand, included both types of conflict simultaneously. In these trials a higher P3 was observed compared to both the GAMBLE and the NOGO trials, indicating that the amount of conflict included in a trial modulates the P3 peak amplitude.

Although the P3 observed in the GO trials also reached higher amplitudes compared to the GAMBLE and NOGO trials, clear differences could be observed in the amplitude distribution of the GO and the PASS trials, suggesting different underlying processes. The GO trials produced a P3 comparable to that observed in go trials of a go/no-go task. In go trials the P3 component reaches its maximum at electrode Pz, whereas the peak values are lower at Cz and even lower at Fz (Bruin & Wijers, 2002; Falkenstein, Koshlykova, Kiroj, Hoormann, & Hohnsbein, 1995). This pattern was present in the GO trials of the current study. In the PASS trials, however, the P3 amplitude measured at electrode Cz was equal to that measured at Pz. The P3 amplitude at Fz was indeed lower than that measured at Cz. Directly comparing the P3 amplitudes measured in the PASS and GO trials showed that, whereas at Pz the P3 amplitude was highest in the GO trials, the PASS trials reached significantly higher amplitudes at Fz and borderline higher at Cz. These comparisons support the interpretation that the absence of the gradual amplitude decrease from Pz to Fz in the PASS trials is rather due to increased amplitudes at Fz and Cz in these trials as opposed to a decrease in amplitude at electrode Pz. It at least underlines the differential frontal activation pattern observed in the PASS trials.

The spatial distribution of the conflict-related modulations in the P3 amplitude is more clearly visualized by mapping the distribution of the difference in amplitude between the PASS and GAMBLE trials, the PASS and NOGO trials, and the PASS and GO trials, respectively (Figure 5). These scalp maps further highlight that the P3 amplitude modulation in the PASS trials had a fronto-central distribution across the scalp. This distribution is compatible with conflict resolution sources in dorsolateral prefrontal cortex that are thought to increase control upon the detection of conflict (Cohen et al., 2000; Kerns et al., 2004; Ridderinkhof et al., 2004). Alternatively, this distribution, and the modulation related to the amount of conflict, is also compatible with conflict monitoring by sources in ACC.

Using the excellent time resolution of ERPs, we were able to show that before conflict is resolved it is first evaluated whether or not there are conflicts to resolve. This process is captured in the N2. The further processing of the detected conflict(s) is resembled in P3. However, the current results do not allow distinguishing whether the P3 peak represents conflict resolution by dorsolateral prefrontal cortex or conflict monitoring by ACC.

Finally it should be noted that an increase in the P3 amplitude at the frontal electrodes is generally linked to a lower probability of stimulus appearance (Courchesne, Hillyard, & Galambos, 1975). It is, however, unlikely that the current effects on the P3 amplitude are related to novelty, as the probabilities of the different conditions were comparable (NOGO, 17%; GO, 23%; PASS, 26%; GAMBLE, 28%). In fact, the NOGO trials had the lowest overall probability (17%), but showed the smallest P3. Our data also provide support for the notion that the higher P3 commonly observed in no-go trials of a go/no-go paradigm is not related to the absence of a response related negativity present in go trials (Verleger, Paehge, Kolev, Yordanova, & Jaśkowski, 2006). The P3 observed in the NOGO trials was lower compared to the P3 in the PASS trials, although in both trial conditions no response was required.

The opportunity to opt out of gambling proved to be a very useful manipulation in our paradigm. The fact that participants did not have to give any response when passing while the stimulus remained visible possibly created extra effort in maintaining that decision. This is opposed to the study of Magno, Foxe, Molholm, Robertson, and Garavan (2006), where choosing to reject a visual search trial effectively ended the trial, enabling participants to *avoid* any conflict and possible loss. Further research should consider including such manipulations to pass or reject a trial, as making a complex decision is associated with choosing and executing one option while leaving the other option(s) be.

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