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Psychophysiological Mechanisms of Interindividual Differences in Goal Activation Modes During Action Cascading

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Our daily life is characterized by multiple response options that need to be cascaded in order to avoid overstrain of restricted response selection resources. While response selection and goal activation in action cascading are likely driven by a process varying from serial to parallel processing, little is known about the underlying neural mechanisms that may underlie interindividual differences in these modes of response selection. To investigate these mechanisms, we used a stop–change paradigm for the recording of event-related potentials and standardized low resolution brain electromagnetic tomography source localizations in healthy subjects. Systematically varying the stimulus onset asynchrony (the temporal spacing of "stop" and "change" signals), we applied mathematical constraints to classify subjects in more parallel or more serial goal activators during action cascading. On that basis, the electrophysiological data show that processes linking stimulus processing and response execution, but not attentional processes, underlie interindividual differences in either serial or parallel response selection modes during action cascading. On a systems level, these processes were mediated via a distributed fronto-parietal network, including the anterior cingulate cortex (Brodman area 32, BA32) and the temporo-parietal junction (BA40). There was a linear relation between the individual degree of overlap in activated task goals and electrophysiological processes.

Keywords: anterior cingulate cortex, P3, response selection, stop–change paradigm, temporo-parietal junction

Introduction

Our daily life is characterized by multiple response options that seek access to restricted resources. To cope with this, it is important to cascade different actions on response options. From a computational neuroscientific perspective, response selection processes are frequently conceptualized as functions of fronto-striatal networks (Redgrave et al. 1999; Bar-Gad et al. 2003; Plenz 2003; Humphries et al. 2006; Schroll et al. 2012), which have been underlined by studies in basal ganglia disorders (e.g., Beste et al. 2009; Cameron et al. 2010; Willemssen et al. 2011; Beste et al. 2012; Ravizza et al. 2012). Other concepts suggest that complex multicomponent behavior is mediated via a "multiple demand system" (MD system), encompassing areas in the frontal and parietal cortex (Duncan 2010). Classical cognitive models (aka central bottleneck models) assume that response selection cannot concurrently operate on more than one response option (Pashler 1994; review: Meyer and Kieras 1997). However, a number of results from experimental psychology suggest that the selection of actions and goal activation in situations requiring action cascading does not necessarily rely on a central bottleneck that implies a serial processing of the different responses (e.g., Sommer et al. 2001; Oberauer and Kliegl 2004; Verbruggen et al. 2008; for review: Wiu and Liu 2008; Miller et al. 2009). Rather, the processing of different responses and the activation of task goals may be performed in a "strategic nature" (e.g. Meyer and Kieras 1997). This is also proposed in the concept of "threaded cognition" (Salvucci and Taatgen 2008), an integrated theory of "multitasking". According to this account, 2 or more tasks can be performed at once, as long as a complex multicomponent goal can be represented as different threads of processing that are coordinated by a processing resource and are executed by other resources (e.g. motor or perceptual resources; Salvucci and Taatgen 2008).

Along these lines, the activation of intended outcomes in situations where action cascading is necessary is driven by some form of nondeterministic processing that can be implemented in either a serial or a parallel fashion (Verbruggen et al. 2008). Even though there is a lot of experimental psychological evidence showing that response selection mechanisms vary along a continuum from more serial to more parallel processing (e.g. Miller et al. 2009), the physiological mechanisms that differ between subjects displaying a more serial or a more parallel mode of response selection are not known.

To investigate this question, we quantified the degree to which 2 consecutive actions are selected on a continuum ranging from serial to parallel processing in a stop–change paradigm (Verbruggen et al. 2008). This paradigm is a hybrid of a stop-signal paradigm and a psychological refractory period (PRP) paradigm (Verbruggen et al. 2008). Subjects are required to stop an ongoing response and then shift to an alternative response. This shift is signaled either at the same time as the stop process, or with a short delay. Verbruggen et al. (2008) varied the delay (stimulus-onset asynchrony, SOA) between the stop and change stimuli and estimated (on the basis of reaction time data) in how far the stop and the change processes overlap. The degree of overlap was estimated by calculating the slope of the SOA-reaction times (RTs) function, that is, the function that describes how the RT on the change stimulus varies depending on the delay between stop and change stimuli. A steeper slope of this function reflects more overlapping of the stop and change processes (c.f. Verbruggen et al. 2008; refer Materials and methods section for details). Here, we used this mathematical constraint to classify subjects as a "serial mode group" and a "parallel mode group" of action cascading. On the basis of this classification, we constrained analysis of electrophysiological (EEG) data and source localization using standardized low resolution brain electromagnetic tomography (sLORETA).

It has been suggested that central executive and working memory processes play a pivotal role in tasks imposing multiple demands, as they map a stimulus on the appropriate

response (e.g., Huestegge and Koch 2010; Oberauer and Bialkova 2011; Schroll et al. 2012) and/or activate different goals involved in action cascading (e.g. Verbruggen et al. 2008). A component of the event-related potential (ERP) that is assumed to reflect working memory processes (review: Polich 2007) and, in choice reaction tasks, an intermediate process between stimulus evaluation and responding (Falkenstein et al. 1994a, 1994b; Verleger et al. 2005) is the parietal P3. Confirming these hypotheses, several studies provide evidence that the P3 is modulated by experimental variations in dual-task situations (i.e. PRP paradigms; e.g., Brisson and Jolicoeur 2007; Sigman and Dehaene 2008). Yet, some results also point toward the relevance of attentional processes in dual- or multitasking situations (Brisson and Jolicoeur 2007). We therefore examine the possible effects of more serial and more parallel goal activation during action cascading in subjects from attentional processes (P1 and N1 ERP) to later processes reflected by the P3. We assume that especially the P3 ERP component yield differences between processing groups, as classified by the SOA-RT function. Since fronto-striatal networks, including the anterior cingulate cortex (ACC), play an important role in response selection (Bar-Gad et al. 2003; Plenz 2003; Botvinick et al. 2004; Humphries et al. 2006), variations at the ERP level, due to the group classification on the basis of the SOA-RT function, may emerge due to the modulation of ACC activity. However, besides the ACC, the inferior parietal cortex that has been shown to be involved in the chaining of actions (e.g. Chersi et al. 2011) is part of the MD system, which mediates multicomponent behavior (Duncan 2010). Based on these considerations, we expect that differences in the P3 ERP between groups (more serial vs. more parallel mode of response selection) are related to differential modulations in frontal and parietal areas, as to be revealed via sLORETA.

Materials and Methods

Participants

In total, 24 subjects ($N = 24$) between 20 and 30 years of age (15 females) participated in the study. All subjects had normal or corrected-to-normal vision, had normal hearing level, no history of neurological and psychiatric diseases, were right-handed, and received course credits or financial compensation for their participation. The study was approved by the ethics committee of the medical faculty of the Ruhr-Universität Bochum. All subjects gave written informed consent before the study protocol was conducted. The study accords to the Declaration of Helsinky.

Task

To examine response selection on a continuum between serial and parallel processing, we adapted the "stop–change paradigm" by Verbruggen et al. (2008), since this allows an estimation of the degree of nondeterministic serial or parallel processing. This paradigm represents a procedural bridge between "PRP" and "stop-signal" paradigms (Verbruggen et al. 2008). The task was presented using "Presentation" software (Neurobehavioural Systems, Inc.). The experiment is structured as follows and shown in Figure 1.

The target stimuli were 4 vertically arranged circles (8 mm diameter) separated by 3 horizontal lines (line thickness: 1 mm and width: 8 mm), which served as reference lines. The distance between the edge of a circle and a reference line was 12 mm. All stimuli had a vertical viewing angle of 8°. Target stimuli and reference lines were framed by a white rectangle (20 × 96 mm, line thickness of 1 mm). In the first picture of every trial, the potential target stimuli (4 empty

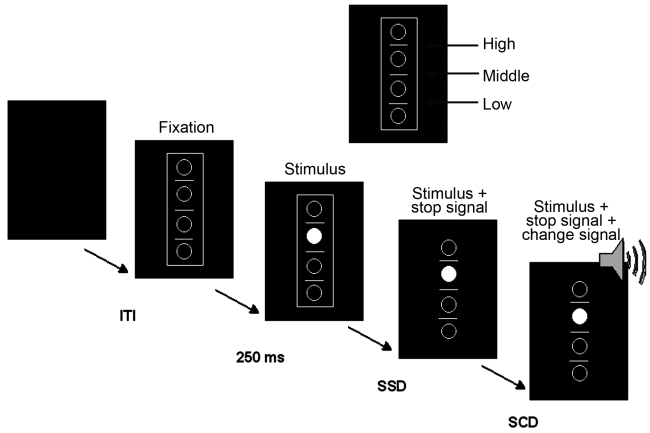

Figure 1. Schematic illustration of the applied stop–change paradigm (modified from Verbruggen et al. 2008).

circles) separated by the 3 reference lines were presented within the white rectangle. After 250 ms, one of the circles was filled with white color (GO1 stimulus). In the GO1 condition, participants were instructed to judge whether this white-filled circle (target) was located above or below the middle reference line. In the GO1 condition, participants responded by pressing the right outer key with the right middle finger (for "above" judgments) or the right inner key using the right index finger (for "below" judgments) on a response paddle with 4 keys. All stimuli remained visible until either the participant responded or a time frame of 2500 ms had elapsed. If no "stop signal" (a red rectangle replacing the usual white rectangle framing stimuli and reference lines; denoted by gray color in Fig. 1) was presented, the GO1 trial ended at this point. The intertrial interval was 900 ms. In 30% of all trials, a stop signal was presented. In these cases, a reaction toward the GO1 stimulus had to be inhibited and a new task ("GO2") had to be executed afterwards, which will be explained below. The "stop-signal delay" (SSD) was initially set to 450 ms and modified by means of a "staircase procedure" (Verbruggen et al. 2008) in order to obtain a 50% probability of successfully interrupted GO1 responses. If a participant fulfilled the requirements of both successfully inhibiting the GO1 response in face of a stop signal and correctly reacting to the subsequent GO2 stimulus, the SSD for the next "stop–change trial" was prolonged by 50 ms. In case at least 1 of these 2 operations failed, the SSD was shortened by 50 ms. The GO2 task was a new judgment following the "stop signal" (which persisted until the end of the trial including the presentation of the GO2 stimulus). In order to set a new reaction goal for the GO2 part of the trial, a sine tone presented via headphones served as a "change signal". There were change signals at 3 different pitches [low (300 Hz), middle (900 Hz), and high (1300 Hz) tones] (presented at 75 dB sound pressure level), indicating which of the 3 lines replaced the middle reference line if previously set by the GO1 section of the trial. These auditory stimuli were presented via headphones. In case, the change signal was a low tone, the low line became the new reference line. Following the same logic, the middle tone encoded the middle reference line, while the high tone represented the upper reference line. For the GO2 task, participants responded either by pressing the left outer key using the left middle finger (for "above" judgments), or by pressing the left inner key using the left index finger (for "below" judgments). All 3 reference lines were in effect equally often. The participants were instructed to always respond as fast and accurately as possible. Trials in which only a GO1 response was required and where stopping and changing to another response was required were randomly intermixed. Moreover, it was not predictable whether the change signal was presented at the same time as the stop signal, or with 300-ms SOA. Furthermore, the pitch of the tone signaling the change was not predictable. As the pitch of the tone (in relation to the varying spatial position of the visual stimuli) was also not predictable, it is impossible for the subjects to predict with which finger the alternative response on the change stimuli should be given. All this

prevents that preparatory effects in the motor system bias the results. The experiment consisted of a total of 864 trials that were presented within approximately 25 min.

Modulating the Continuum of Serial and Parallel Processing in Action Cascading

In line with Verbruggen et al. (2008), we introduced different lengths of "stop–change delays" (SCDs) in order to modulate response selection processes on a serial–parallel continuum in the stop–change paradigm mentioned above. One SCD had a SOA of 0 ms (i.e., onset of the stop signal and change signal occurred simultaneously), the other SCD had a SOA of 300 ms (i.e., onset of the change signal occurred 300 ms after the onset of the stop signal). Stochastic variation of trial types (GO1, and STOP + GO2 with SCD 0 and 300) results in a mixture of trials in which the GO1 response is sometimes not inhibited before the change signal is presented, while in other occasions the GO1 response is inhibited (Verbruggen et al. 2008). Using this SOA manipulation, it is possible to calculate the slope of RTs across SOAs describes how the RT on the change stimulus varies depending on the delay between stop and change stimuli. We calculated the slope value using the equation:

$$\text{slope} = \frac{\text{GO2 RT}_{\text{SOA 300}} - \text{GO2 RT}_{\text{SOA 0}}}{\Delta\text{SOA}}$$

In this context, RTs refer to the change signal response (i.e., GO2 response). The rationale behind this is as follows (c.f. Verbruggen et al. 2008): The local slope of the SCD function at a given delay (SCD 0 and 300) reflects the probability that the first process (STOP process) has not finished and overlaps with the following process (GO2; Schwarz and Ischebeck 2001; Verbruggen et al. 2008). If the STOP process has not finished, it was shown that the slope approximates −1. If it has finished, it was shown that the slope is close to 0 (Verbruggen et al. 2008). Obtaining a mean slope value in between 0 and −1 hence suggests that the initiation of some (but not all) of the GO2 responses sometimes occurred before the termination of the inhibitory process of stopping the GO1 response. Hence, the steeper the mean slope, the more likely it is that the stop process had not finished at the time the GO2 response was initiated. In other words, in case of more serial processing, the slope of the SOA-RT function is flatter (closer to 0) than in the case of a more parallel processing mode (closer to −1). In the SOA-300 condition, all subjects are forced to serially perform both the stop and change processes, simply because the temporal gap between the stop and the change stimuli imposes a cascaded task order. Therefore, RTs should be highly similar between more serial and more parallel processing groups in this condition. However, things lie differently in the SOA-0 condition. Here, the simultaneous presentation of the stop and change signals yields the possibility of 2 different processing modes: A more serial and a more parallel processing mode. Virtually, all response selection (bottleneck) models allow parallel and serial processing (Miller et al. 2009). Because response selection depends on a restricted resource, the processing mode may differentially affect RT in the SOA-0 condition. As long as subjects vary in the RTs of the SOA-0 condition, the serial/parallel categorization can be made. Calculating the individual slope for each subject across the experimental trials yielded a value denoting the degree to which this subject is inclined toward either a more serial or a more parallel processing mode. In the current study, we used this parameter to split (median split) the cohort into 2 subgroups, denoting either a more serial or a more parallel processing mode. However, according to Verbruggen et al. (2008), it is not possible to distinguish between nondeterministic serial processes and parallel processing based on the RT slope value (c.f. Verbruggen et al. 2008 for a detailed discussion on this issue). We only use the terms "more serial" or "more parallel" just for the sake of simplicity.

It may be argued that it is problematic to artificially split the continuum from more serial to more parallel processing into 2 groups. Therefore, we also ran a second approach where we do not split the continuum into 2 groups, but use the slope value as a continuous regressor in correlation analyses.

EEG Recording and Analysis

EEG was recorded from 65 Ag–AgCl electrodes using a QuickAmp amplifier (Brain Products, Inc.) at standard scalp positions against a reference electrode located at FCz. The sampling rate was 1000 Hz, which was down-sampled offline to 256 Hz. All electrode impedances were kept <5 kΩ. Data processing involved a manual inspection of the data to remove technical artifacts. After manual inspection, a band-pass filter ranging from 0.5 to 20 Hz (48 db/oct) was applied. After filtering, the raw data were inspected a second time. To correct for periodically recurring artifacts (pulse artifacts, horizontal and vertical eye movements), an independent component analysis (Infomax algorithm) was applied to the unepoched data set. Afterwards, the EEG data were segmented according to the 4 different conditions. Segmentation was applied with respect to the occurrence of the stop signal (i.e., stimulus-locked). Visual ERPs (due to the stop signal) and auditory ERPs (due to the change signal) were evaluated. Automated artifact rejection procedures were applied after epoching: Rejection criteria included a maximum voltage step of >60 μV/ms, a maximal value difference of 150 μV in a 250-ms interval, or activity <0.1 μV. Then, the data were current source density (CSD)-transformed (Perrin et al. 1989) in order to eliminate the reference potential from the data. A second advantage of the CSD transformation is that it serves as a spatial filter (Nunez and Pilgreen 1991), which makes it possible to identify electrodes that best reflect activity related to cognitive processes. After CSD transformation, the baseline correction was performed. For the baseline correction we choose a time window from −900 till −700 ms and not a baseline prior to the presentation of the stop stimulus, since we wanted to have a "real" prestimulus baseline that was well before the presentation of the GO1 stimulus. Based on this stimulus-locking procedure, the P1, N1, and P3 ERPs were quantified [For inhibitory control processes, the (Nogo)-N2 occurring with a latency of 200–300 ms after the inhibitory signal (e.g. Falkenstein et al. 1999; van Boxtel et al. 2001) has frequently been analyzed. In the current paradigm, a Nogo-N2 like component is evident in the SOA-300 condition (Fig. 4). However, in the SOA-0 condition, this component is not detectable due to the simultaneously occurring change processes. As the (Nogo)-N2 is therefore not quantifiable in all conditions, the (Nogo)-N2 is not included in the analysis.], based on the scalp topography; that is, electrodes used for data quantification were selected in a data-driven manner. Electrodes were first chosen on the basis of visual inspection of the scalp topography. As the scalp topography showed a bilateral pattern of activation for the different ERP components. Because of this bilateral pattern electrodes at both sides of the scalp were quantified, even though there is no reason to assume lateralizations in the effects. According to this, the visual P1 and N1 were measured at electrodes PO7 and PO8 (P1: 0–140 ms and N1: 150–250 ms), the auditory N1 at C5 and C6 (0–500 ms), and the P3 at Cz and Pz (200–600 ms). To verify the choice of these electrodes, the following validation procedure was run: For each ERP component, a search interval was defined (noted above), in which the component is expected to be maximal. After this, we extracted the mean amplitude within each of these search intervals at each of the 65 electrode positions. This was done after CSD transformation of the data, because the CSD transformation has the effect of a spatial filter that accentuates scalp topography (Nunez and Pilgreen 1991), as can also be seen in comparison with ERPs and maps presenting the data on average reference (refer also Supplementary Material). Subsequently, we compared each electrode against an average of all other electrodes using Bonferroni-correction for multiple comparisons (critical threshold, $P = 0.0007$). Only electrodes that showed significantly larger mean amplitudes (i.e., negative for N1 potentials and positive for the P1 and P3 potentials) than the remaining electrodes were chosen. This procedure revealed the same electrodes as previously chosen on the basis of visual inspection of the scalp topography plots. The ERP components were quantified relative to the prestimulus baseline. All components were quantified in peak amplitude and latency on the single-subject level. In case of the P3, the peak-to-peak amplitude was used, since the negativity before the P3 was differently large for the different groups (refer Fig. 4). (Data analysis was repeated using the mean amplitude at the above mentioned and validated electrode positions. The effects obtained were identical to the reported data analysis on the peak amplitudes.)

Source Localization

Source localization was carried out on ERPs showing differences between the serial and parallel processing groups. Source localization was conducted using sLORETA (Pascual-Marqui 2002). sLORETA gives a single linear solution to the inverse problem based on extracranial measurements without a localization bias (Pascual-Marqui 2002; Marco-Pallarés et al. 2005; Sekihara et al. 2005). sLORETA has been validated in simultaneous EEG/functional magnetic resonance imaging studies (Vitacco et al. 2002). For sLORETA, the intracerebral volume is partitioned in 6239 voxels at 5-mm spatial resolution, and the standardized current density at each voxel is calculated in a realistic head model (Fuchs et al. 2002) using the MNI152 template (Mazziotta et al. 2001). In the present study, the voxel-based sLORETA images were compared between groups using the sLORETA-built-in voxel-wise randomization tests with 2000 permutations, based on statistical nonparametric mapping. Voxels with significant differences ($P < 0.05$, corrected for multiple comparisons) between groups were located in the MNI brain, and Brodman areas (BAs) as well as coordinates in the MNI brain were determined using the sLORETA software (www.unizh.ch/keyinst/NewLORETA/sLORETA/sLORETA.htm). The comparison of sLORETA images between groups was based on the original ERPs in the time domain on the basis of scalp voltages. sLORETA was applied on P3 ERP data, since only the P3 revealed differences between processing groups and a linear correlation between individual slope values (refer Results section).

Statistics

Behavioral data were analyzed using mixed and univariate analyses of variance (ANOVAs). In the mixed ANOVAs, the factor "SOA length" was the within-subject factor with 2 factor levels (i.e., SOA-0 and SOA-300). As the between-subject factor, the groups calculated on the basis of the median split (i.e., "serial progressing group" and "parallel processing group") were included in the ANOVAs as a 2-level factor. For the neurophysiological data, an additional within-subject factor ("electrode") with 2 factor levels was introduced where necessary, resulting in 2 within-subject factors (SOA length and electrode) and the between-subject factor. For the visual P1 and N1, the factor levels were electrodes PO7 and PO8, for the auditory N1 the factor levels were electrodes C5 and C6, and for the P3 the factor levels were electrodes Cz and Pz. The ERP components were quantified relative to the prestimulus baseline. When appropriate, the degrees of freedom were adjusted using Greenhouse-Geisser correction. All post hoc tests were Bonferroni-corrected. Kolmogorov–Smirnov tests revealed that all relevant variables were normally distributed (all $z < 0.5$; $P > 0.4$; 1-tailed). As a measure of variability, the standard error of the mean (SEM) is given.

Results

Behavioral Data

The mixed ANOVA revealed a significant main effect of "trial (GO1 trials without stop–change signals and GO2 trials on SCD 0 and 300)" ($F_{1,22} = 95.70$; $P < 0.001$; $\eta^2 = 0.81$). Post hoc pair-wise comparisons showed that RTs for all 3 trial conditions differed significantly from each other ($P < 0.001$). On average, subjects showed the shortest RTs in the GO1 trial (486.02 ± 73.00 ms) and the longest RTs for SOA-0 (1017.83 ± 270.37 ms). The mean duration of RTs for SOA-300 (906.61 ± 246.06 ms) ranged between those of the GO1 and SOA-0 trial. Calculating the slope of the RT-SOA function (c.f. Verbruggen et al. 2008) revealed a mean slope of −0.55 (0.07). The more parallel processing group (as defined by a median split) revealed a slope of −0.93 ± 0.26, while the more serial processing group revealed a slope of −0.17 ± 0.25. Necessarily, the 2 groups differed from each other ($F_{1,22} = 53.09$; $P < 0.001$; $\eta^2 = 0.71$). Within the serial processing group, RTs for SOA-0 were (980 ± 57 ms) and differed from RTs in the SOA-300 condition (929 ± 71 ms) ($t_{11} = 2.30$; $P < 0.05$). Within the parallel processing group, RTs for SOA-0 (1163 ± 88 ms) and SOA-300 (883 ± 72 ms) also differed from each other ($t_{11} = 12.53$; $P < 0.001$), with the SOA effect being stronger than in the serial processing group ($P < 0.05$). There was no effect of the factor "group" on RTs in GO1 trials ($F_{1,22} = 0.02$; $P = 0.90$; $\eta^2 = 0.001$). The mean stop-signal reaction time (SSRT) was 241.2 ms (16.1). The median splitted groups did not differ with respect to their SSRT ($P > 0.2$). There was also no correlation between the slope of the RT-SOA function and SSRT ($r = 0.2$; $R^2 = 0.4$; $P > 0.3$). The mean SSD was 203.3 ms (8). To account for a possible speed–accuracy trade-off between the groups, error rates on the GO2 stimulus were examined. There was no interaction between SOA length and group ($P > 0.4$). Moreover, there was no main effect group, or main effect SOA length (all $F < 0.5$; $P > 0.5$). This shows that group differences do not reflect a speed-accuracy trade-off.

Electrophysiological Data

Stimulus-locked ERPs for SOA-0 and SOA-300 are shown in Figures 2 and 3. The ERP traces and topographies depicted in these figures represent the group data.

P1

Due to the scalp topography of the visual P1 ERPs, electrodes PO7 (left hemisphere) and PO8 (right hemisphere) were chosen for analysis (Fig. 2*A*), since these electrodes were located in the center of the scalp positivities. The mixed ANOVA revealed a significant main effect of the factor SOA length ($F_{1,22} = 31.91$; $P < 0.001$; $\eta^2 = 0.59$), showing that the P1 amplitude was larger for SOA-0 (23.82 $\mu V/m^2$ ± 16.62) than for SOA-300 (17.38 $\mu V/m^2$ ± 14.62). In contrast, only a non-significant trend was found for the factor "electrodes" ($F_{1,22} = 4.00$; $P = 0.06$; $\eta^2 = 0.15$). The main effect of group and all interactions, including the group factor, did not show significant effects (all Fs < 1.55 and Ps > 0.23), showing that serial or parallel processing did not affect P1-related processes. There were also no effects of latency of the P1 peak (all $F < 1$ and $P > 0.2$).

Visual N1

Similar to the P1, the visual N1 was most pronounced at PO7 and PO8 (Fig. 2*A*). There was a significant main effect of electrode ($F_{1,22} = 16.71$; $P < 0.001$; $\eta^2 = 0.43$), indicating that following stimulus presentation, the amplitude of N1 was significantly more negative at electrode PO8 (−61.73 $\mu V/m^2$ ± 32.17) than at PO7 (−44.79 $\mu V/m^2$ ± 21.27). Even though there was no significant main effect of group ($F_{1,22} = 0.47$; $P = 0.50$; $\eta^2 = 0.02$) showing that serial and parallel processing did not affect N1-related processes, the mixed ANOVA revealed a significant interaction between electrodes and group ($F_{1,22} = 5.55$; $P = 0.03$; $\eta^2 = 0.20$). The difference in the N1 amplitude between the parallel and serial groups was larger for PO8 than for PO7, with PO8 showing a larger amplitude difference in the serial group (−70.09 $\mu V/m^2$ ± 36.44) than in the parallel group (−53.37 $\mu V/m^2$ ± 6.13). For PO7, this difference was smaller, but still the parallel group showed a lower amplitude (−46.19 $\mu V/m^2$ ± 21.80) than the serial group (−43.39 $\mu V/m^2$ ± 21.31) ($P < 0.01$). All other main effects and

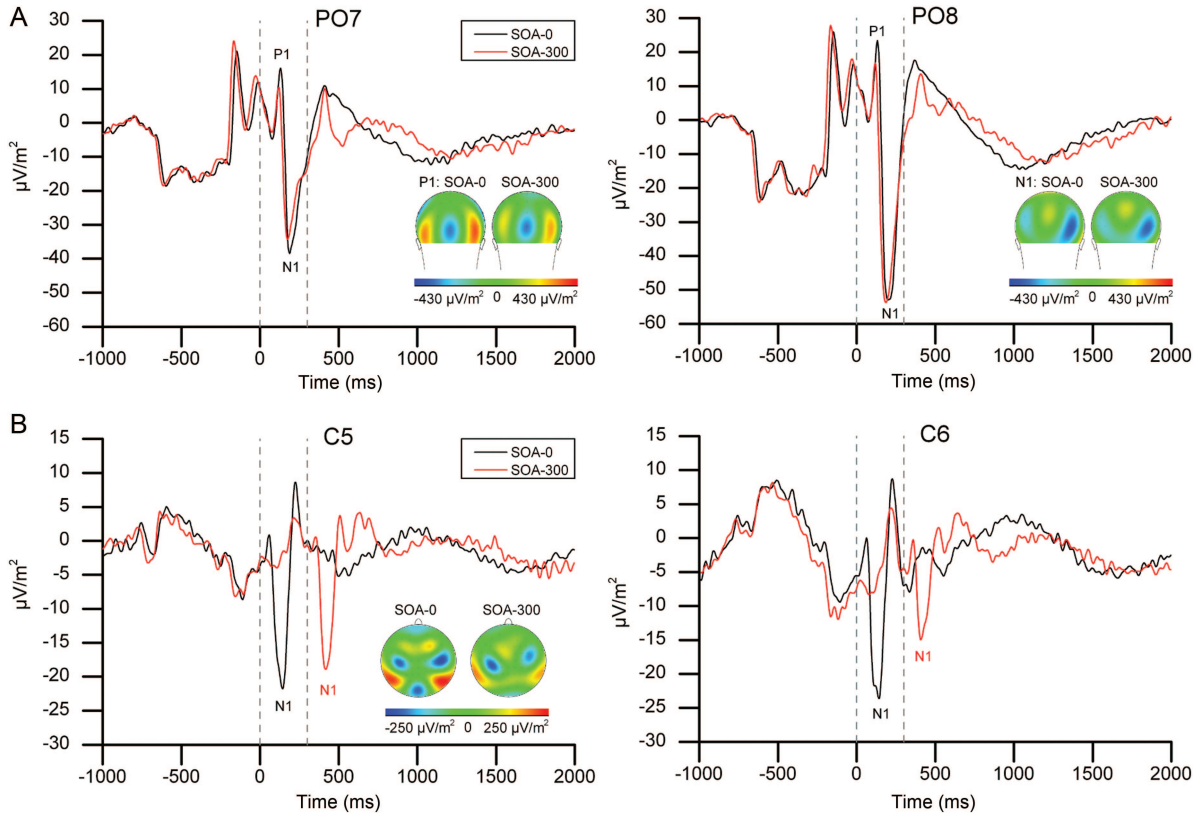

Figure 2. (*A*) ERPs locked to the occurrence of the stop signal (time point 0; first vertical dashed line) for the SOA-0 (black) and SOA-300 conditions (red). Electrodes PO7 and PO8 are shown. A clear visual P1 and visual N1 complex is seen at both electrodes, with the topographic maps revealing a clear P1 and N1 scalp topography. The scalp topographies depict the topography of the ERP at its peak. The second vertical dashed line denotes the time point of the auditory change stimulus in the SOA-300 condition. (*B*) ERPs locked to the occurrence of the stop signal (time point 0; first vertical dashed line) for the SOA-0 (black) and SOA-300 conditions (red). Electrodes C5 and C6 are shown. A clear auditory N1 complex is seen at both electrodes, with the topographic maps revealing N1-related negativities around electrodes C5 and C6. The scalp topographies depict the topography of the ERP at its peak.

interactions failed to reach significance (all Fs < 3.79 and Ps > 0.32). For N1 as well, there were no effects of latency (all $F < 1$ and $P > 0.2$).

Auditory N1

The topography of the auditory N1 was centered around the C5 and C6 electrodes, as shown by the scalp topographies (Fig. 2*B*). Accordingly, electrodes C5 and C6 were chosen for further analysis. There was a significant main effect of SOA length on the auditory N1 amplitude ($F_{1,22} = 6.07$; $P = 0.02$; $\eta^2 = 0.22$). The amplitude was more negative in the SOA-0 ($-28.52\ \mu V/m^2 \pm 13.47$) than in the SOA-300 ($23.90\ \mu V/m^2 \pm 12.65$) trials. Even though the main effect of electrodes failed to reach significance ($F_{1,22} = 0.17$; $P = 0.69$; $\eta^2 = 0.01$), the interaction electrodes × SOA was significant ($F_{1,22} = 16.58$; $P = 0.001$; $\eta^2 = 0.43$). This indicates that the different SOA durations had differential influences on the amplitudes on electrodes C5 and C6. At SOA-0, the difference in N1 amplitudes between electrode C6 ($-29.88\ \mu V/m^2 \pm 13.65$) and C5 ($-27.16\ \mu V/m^2 \pm 13.29$) was smaller than for SOA-300 (where C5 = $-26.13\ \mu V/m^2 \pm 13.85$ and C6 = $-21.68\ \mu V/m^2 \pm 11.45$). Post hoc *t*-tests revealed that this difference between C5 and C6 amplitudes was significant only for the SOA-0 condition (SOA-0: $P = 0.05$ and SOA-0: $P = 0.29$).

At electrode C6, the difference between the N1 amplitudes for SOA-0 ($-29.88\ \mu V/m^2 \pm 13.65$) and SOA-300 ($-21.68\ \mu V/m^2 \pm 11.45$) was significantly larger ($P = 0.05$) than at electrode C5. At electrode C5, the amplitudes in the different SOA conditions (SOA-0 = $-27.16\ \mu V/m^2 \pm 13.29$ and SOA-300 = $-26.13\ \mu V/m^2 \pm 13.85$) did not differ from each other ($P = 0.29$). The main effect of groups and all interactions was not significant (all Fs < 2.59 and all Ps > 0.12), again showing that the N1-related processes were not affected by serial and parallel processing modes.

P3

The P3 is given in Figure 3*A* for electrodes Cz and Pz for the SOA-0 and SOA-300 conditions. As can be seen in Figure 3*B*, the serial and parallel processing groups seem to differ in the amplitude of the P3 in both the SOA-0 and SOA-300 conditions. The ERP traces and topographies depicted in these figures represent the group data. The statistical analysis reveals the following:

The latency of the P3 for the SOA-0 condition was 261 ms (30) at electrode Cz and 384 ms (35) at electrode Pz ($P < 0.001$). For the SOA-300 condition, the latency of the P3 was 458 ms (31) at electrode Cz and 515 ms (29) at electrode Pz ($P < 0.001$). The amplitude of the P3 at electrode Cz differed significantly between SOA-0 ($39.97\ \mu V/m^2 \pm 19.16$) and SOA-300 ($29.84\ \mu V/m^2 \pm 12.05$) ($F_{1,22} = 28.04$; $P < 0.001$; $\eta^2 = 0.56$), but there was no effect for electrode Pz ($F_{1,22} = 1.04$; $P > 0.3$). Although the main effect for group did

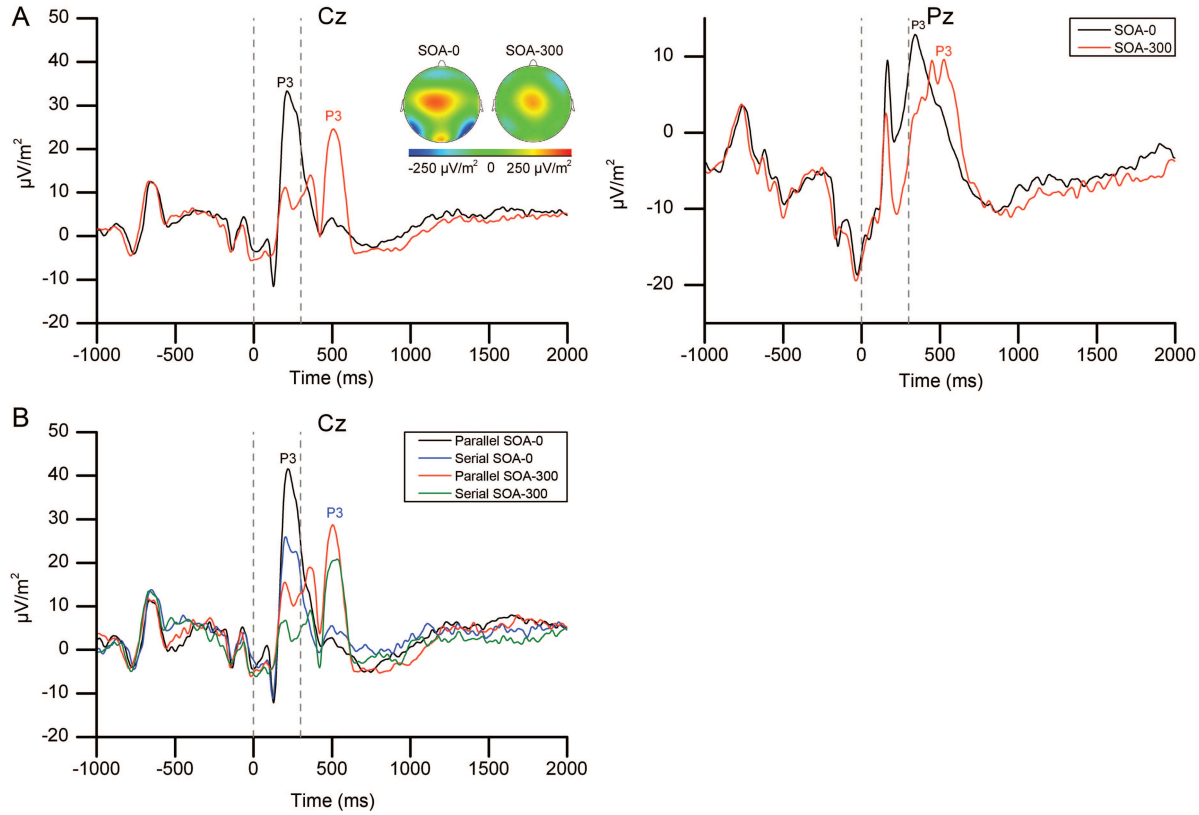

Figure 3. (*A*) ERPs locked to the occurrence of the stop signal (time point 0, first vertical dashed line) for the SOA-0 (black) and SOA-300 conditions (red). Electrodes Cz and Pz are shown. A clear P3 complex is seen at both electrode sites, with the topographic maps revealing P3-related positivities around electrode Cz. The scalp topographies depict the topography of the ERP at its peak. The second vertical dashed line denotes the time point of the auditory change stimulus in the SOA-300 condition. (*B*) ERPs at electrode Cz for the SOA-0 and SOA-300 conditions, separated for the more serial and more parallel processing subgroups.

not reach significance ($F_{1,22} = 3.57$; $P = 0.07$; $\eta^2 = 0.14$), the mixed ANOVA revealed a significant interaction for SOA length × group ($F_{1,22} = 6.63$; $P = 0.02$; $\eta^2 = 0.23$) for electrode Cz, but not for Pz ($F_{1,22} = 1.14$; $P > 0.29$). For electrode Cz, the P3 amplitudes in the SOA-0 condition seem to differ more between the serial (31.98 $\mu V/m^2 \pm 15.38$) and parallel groups (47.97 $\mu V/m^2 \pm 19.79$) than they did for SOA-300 (where serial = 26.77 $\mu V/m^2 \pm 13.29$ and parallel = 32.92 $\mu V/m^2 \pm 10.32$; refer Fig. 3*B*). This is supported by post hoc tests: A post hoc *t*-test revealed that the SOA-0 difference reached significance ($t_{22} = 4.21$; $P = 0.01$), while the P3 amplitudes in the SOA-300 condition did not differ between groups ($P > 0.3$). Yet, as can be seen in Figure 3*B* and for the SOA-300 condition, the potentials of the serial and parallel processing groups already seem to differ within a time frame of 200–400 ms after the stop signal (P3-like component). After quantification of the mean amplitude of this P3-like component for each subject within this time frame from 200 till 400 ms after the stop signal in the SOA-300 condition, the statistical analyses show that this difference was not statistically reliable ($P > 0.5$). The differences in P3 amplitudes between the groups were followed up in sLORETA analyses. The results are given in Figure 4.

Comparing the serial and parallel processing groups for the SOA-0 condition (i.e., contrasting the serial and parallel groups for the peak of the P3 in the SOA-0 condition) by using the built-in independent samples test revealed significant activation differences in the ACC (BA32) (refer Fig. 4*A*). The parallel processing group showed more activation of ACC areas (BA32) than the serial processing group. The ERP time-domain analysis revealed no differences between the serial and parallel processing groups in the SOA-300 condition. In addition, the sLORETA analysis using permutation tests to prevent false-positive activations was not able to find differences between the groups in particular brain areas. In this way, the pattern of the sLORETA analysis reflects the interaction in the P3 between "processing group" and "SOA condition" in the time-domain analysis on the ERPs. However, the difference in P3 amplitude between the SOA-0 and SOA-300 conditions was substantial as stated above. Therefore, sLORETA was calculated on this amplitude difference using the built-in dependent samples test. The results show that differences between the SOA-0 and SOA-300 conditions in the parallel processing group are due to activation differences in the temporo-parietal junction (TPJ) (BA40) (refer Fig. 5*B*), with this area being more activated in the SOA-0 than in the SOA-300 condition. Activation differences in the TPJ were also confirmed by an additional analysis in which we compared the SOA-0 and SOA-300 conditions across the whole cohort (refer Fig. 4*C*).

Correlation Analyses

As stated in the Materials and methods section, it may be problematic to split the continuum into 2 artificial serial and parallel processing groups. We therefore run a second approach, where we do not split the continuum into 2 subgroups, but

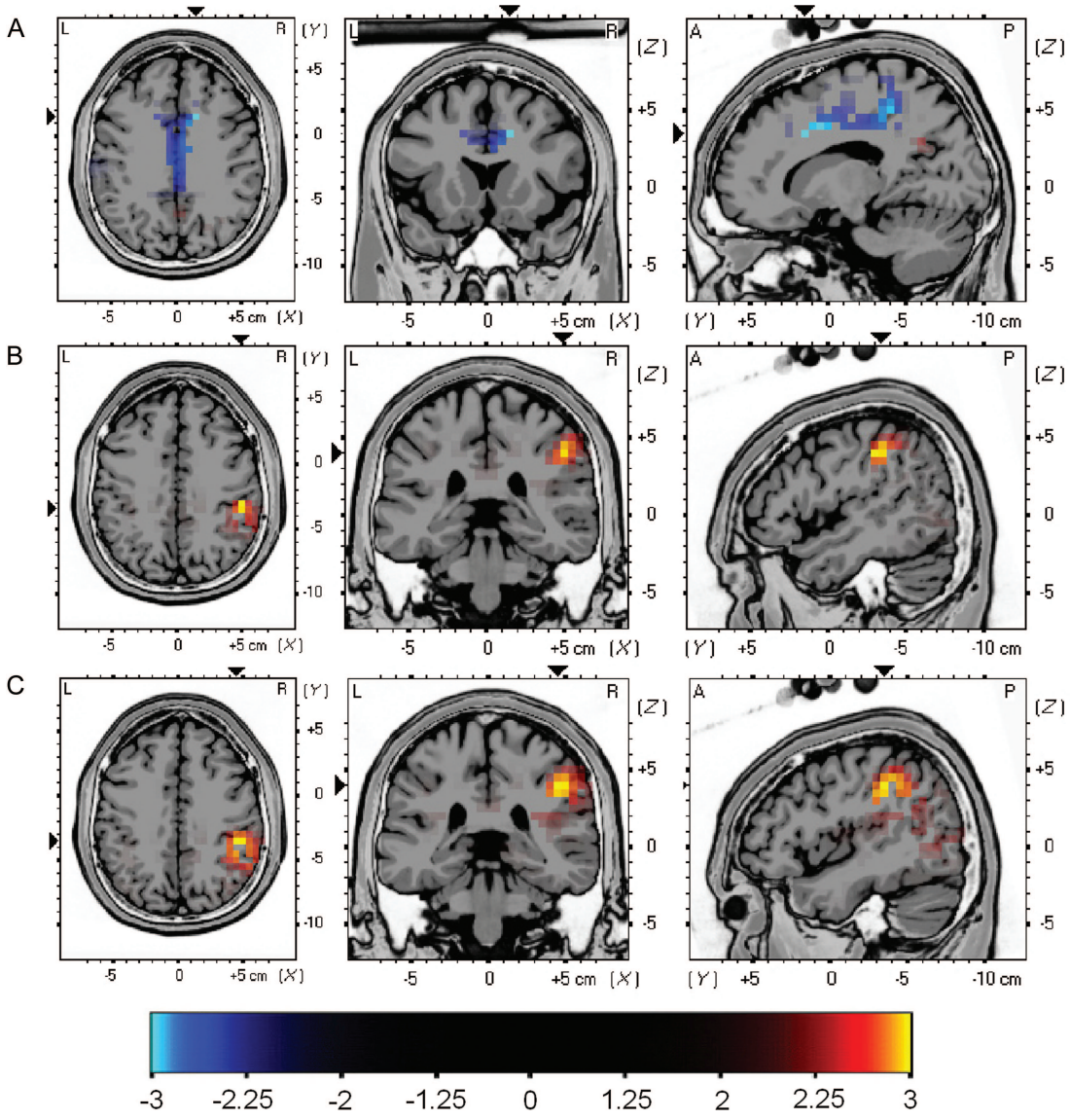

Figure 4. Results of the sLORETA analysis. (*A*) Contrast comparing the more serial and more parallel processing groups in the SOA-0 condition. (*B*) Contrast comparing the SOA-0 and SOA-300 conditions within the more parallel processing group. (*C*) Contrast comparing the SOA-0 and SOA-300 condition in the whole cohort. All pictures show group data. The sources depict the time point of the peak of the P3 within the interval 200–600 ms after stop-signal presentation. The color bar denotes the critical *t*-values (corrected for multiples comparisons using SnPM).

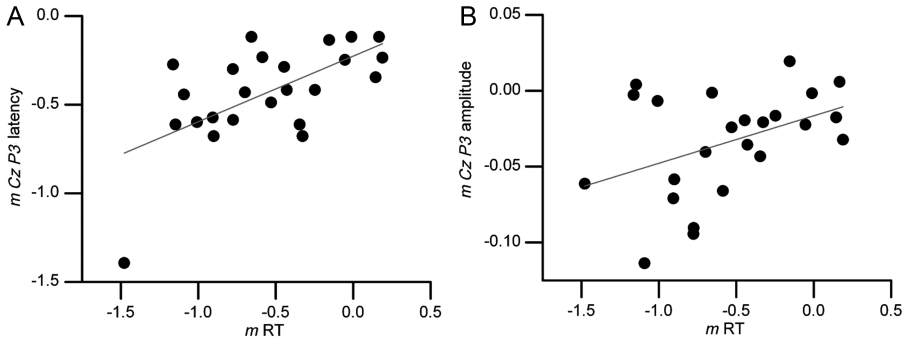

Figure 5. Scatterplots denoting the correlation between the slope of the SOA-RT2 function and the slope of the SOA-P3b peak latency (*A*) and amplitude (*B*) function in the whole cohort.

use the slope value as a continuous variable in correlation analyses. Due to the significant results for the P3 component, we used the latency of the amplitudes to calculate a slope value as previously done for the behavioral data. The results of the correlational analyses underline the pattern observed in the ANOVAs. The slope of the P3 peak latencies across SCD conditions at electrode Pz was significantly correlated with the slope of RTs across SCD conditions ($r = 0.62$, $R^2 = 0.38$; $P = 0.001$), as is shown by Figure 5*A*. The slope of the P3 peak amplitudes across SCD conditions at electrode Cz was also correlated with the slope of RTs across SCD conditions ($r = 0.42$, $R^2 = 0.16$; $P = 0.01$) (refer Fig. 5*B*). For the other ERP parameters (visual N1 and P1, auditory N1), there was no significant correlation ($r < 0.2$; $P > 0.5$), again underlining the specificity of the P3 results. The P3 amplitude at electrode Cz in the SOA-0 condition was highly positively correlated with the P3 amplitude in the SOA-300 condition. This positive correlation indicating that a higher P3 in the SCD 0 condition is related to a higher P3 amplitude in the SCD 300 condition was evident in the whole cohort ($r = 0.873$; $R^2 = 0.75$; $P < 0.001$), as well as in the parallel ($r = 0.943$; $R^2 = 0.88$; $P < 0.001$) and serial processing subcohorts ($r = 0.859$; $R^2 = 0.72$; $P < 0.001$).

An analysis using the SSRT and the amplitude and latency of the P3 in the SOA-0 and SOA-300 conditions did not show any reliable correlations between these parameters (all $r < 0.2$; $P > 0.6$), suggesting that the relation found is specific to the change processes and not to the stop process.

However, as can be seen in Figure 5, there is one outlier in the distribution, which may also distort the median split procedure applied above. Yet, when repeating the above-mentioned ANOVAs using the median splitted groups, the effects observed for the P3 ERP component across the more serial and more parallel processing groups remained the same. It therefore seems rather safe to say that the results obtained are unbiased with respect to this outlier. For the other ERP components (i.e., auditory and visual N1/P1), the effects were also unchanged. Overall, the correlation analysis showed systematic relations between the behavioral parameters and the P3, but not the other neurophysiological parameters. The results of the correlation analyses therefore underline the results pattern obtained in the ANOVAs, without artificially splitting a continuum into dichotomic groups.

Discussion

In the current study, we examined EEG mechanisms underlying a more serial, or a more parallel, mode of goal activation in action cascading using a stop–change paradigm, which allows a quantification of the degree of serial and parallel processing (Verbruggen et al. 2008). The stop–change paradigm is a hybrid of a classical stop paradigm and a PRP paradigm (Verbruggen et al. 2008). The behavioral data obtained are well in line with previous findings by Verbruggen et al. (2008) that revealed a mean slope of −0.61 in RTs (−0.55 (0.07) were obtained in our study). Overall, the behavioral data show that subjects act either on a nondeterministic serial response selection mode or a limited-capacity parallel response selection mode where most of the available resources are used for the stopping process (Verbruggen et al. 2008). A slope value of 0 would imply that the "stop" process had finished before the GO2 response was initiated. Obtaining a mean slope value of −0.55 thus suggests that the initiation of some (but not all) of the GO2 responses sometimes occurred before the termination of the inhibitory process of stopping the GO1 response. The data are unbiased with respect to a speed-accuracy trade-off.

The EEG data revealed differences in the P1, the auditory N1, and P3 ERP components between the 2 SOA conditions. All ERP components were stronger in the SOA-0 than in the SOA-300 condition. This finding is likely to reflect an overall intensification of cognitive effort. Yet, it is also possible that this reflects a multisensory enhancement effect. Although topographies between the ERP components are different, phase resetting (Shah et al. 2004; Lakatos et al. 2007; Kayser et al. 2008) may influence early sensory processing between modalities and could explain the main effect of "SOA." The auditory N1 was modulated by the length of the SOA at electrode C6, which is well in line with studies on multisensory integration also showing a SOA dependency of ERPs (e.g. Giard and Peronnet 1999; Foxe et al. 2000; Fort et al. 2002; Molholm et al. 2002, 2004; Murray et al. 2005). However, at this level of multisensory integration, there was no difference between the more serial and more parallel processing groups, which shows that, at least for the level of attentional processing, multisensory integration does not affect action cascading processes. Yet, it cannot be ruled out that multisensory integration mechanisms play a role for later stages on response selection (i.e., the P3). The results on the P3 at electrode Cz show that amplitudes differed significantly between the SOA-0 and SOA-300 conditions in the parallel group, there was only a trend in the serial group. In the parallel group, the amplitude of the P3 was lower in the SOA-300 than in the SOA-0 condition. In line with this interpretation, several studies using the PRP task have shown that experimental variations modulate especially the latency of the P3 component, which may reflect processing at a strategic central bottleneck (e.g. Brisson and Jolicoeur 2007; Sigman and Dehaene 2008). Matching the suggestion that the P3 reflects processes of a "strategic bottleneck" (e.g. Meyer and Kieras 1997), we found a substantial positive correlation between the slope of the RT on the GO2 stimulus and the slope of the P3 peak latency differences at electrode Pz. This suggests that the degree of nondeterministic parallel processing is directly reflected by EEG parameters, namely the latency of the P3. The slope of the SOA-RT function reflects the degree to which task goals are activated in a serial or in a more parallel fashion (Verbruggen et al. 2008). Therefore, the P3 latency effects reflect a direct EEG correlate of the degree of overlap in goal activation processes described above. The sLORETA analyses revealed that the difference in amplitudes in the critical SOA-0 condition between groups was due to a higher activation of the ACC (BA32) extending in the posterior cingulate cortex in the more parallel processing group. Within the parallel processing group, the sLORETA results further suggest that brain areas in the inferior parietal lobe (TPJ, BA40) extending in the posterior sulcus and supramarginal gyrus were more activated in the SOA-0 than in the SOA-300 condition. This picture was also evident, when examining differences between the conditions across the whole cohort. The TPJ has previously been reported to be related to modulations in the P3 component (e.g. Verleger et al. 1994). From a broader perspective, the TPJ has frequently been suggested

to play in role in the chaining of actions during response selection (e.g., Astafiev et al. 2006; Karch et al. 2010; Chersi et al. 2011). Especially, BA40 has been shown to play a critical role in dual-task performance, as it sustains executive control (Collette et al. 2005). Our sLORETA results suggest that the TPJ is especially important in situations in which stopping and change processes are triggered at once (i.e., SOA-0 condition) and hence in a situation where the chaining of actions is particular demanding. However, since the task used different stimuli to signal stop and change processes, it is possible that the involvement in the TPJ at least partly reflect multisensory integration processes related to the stop and change signal processing, since the TPJ has been shown to be involved in multisensory processing (Matsuhashi et al. 2004; Ionta et al. 2011). It will deserve further experiments to examine whether and how action cascading processes differ when to be cascaded actions are signaled within a single modality.

However, the concomitant ACC activation in the parallel processing group fits into the concept of a MD system (Duncan 2010). The MD system extends over a specific set of regions in the frontal and parietal cortex, encompassing dorsal anterior cingulate areas as well as areas in and around the intraparietal sulcus (Duncan and Owen 2000; Duncan 2010). The MD system is suggested to play an important role in complex multicomponent behavior (Duncan 2010). The task used for this study meets these requirements as subjects had to chain different actions. It is possible that, in subjects tending toward more parallel processing, the increased ACC activity in the SOA-0 condition reflects increased demands on action selection processes that have frequently been suggested to mediate by this area (e.g. Botvinick et al. 2004). Other results suggest that areas in the medial prefrontal cortex may be involved in the selection of tactics (Matsuzaka et al. 2012). It is therefore conceivable that the increased ACC activation in more parallel operating subjects may trigger the increased activation of the TPJ in order to chain the actions necessary to perform the tasks in parallel. This interpretation is in line with findings, suggesting that P3-related processes reflect a link between stimulus processing and the response (Verleger 1988; Falkenstein et al. 1994a, 1994b; Verleger et al. 2005; Polich 2007). In this context, the P3 has been related to the "decision" processes between stimulus evaluation and responding (Falkenstein et al. 1994a, 1994b; Verleger et al. 2005), which is related to the allocation of processing resources (Polich 2007). Yet, this interpretation does not foreclose that multisensory integration processes contribute to mechanisms reflected in differential TPJ activation in the groups.

The above discussion focuses on the role of the ACC and parietal cortex in the MD system. Yet, these areas are also involved in interference detection, response inhibition, and executive control. As we found no correlation between the P3 ERP parameters and SSRT as an indicator of inhibitory control in this paradigm, it is likely that the modulation observed at a neurophysiological level are more related to the cascading of actions in the sense of the MD-system conception and less related to the process of inhibitory control.

Summing-up our findings, it can be said that, out of all investigated ERP components, the P3 was the only component allowing for a clear dissociation of the parallel and serially processing groups. In contrast to this, the earlier attention-associated components (visual P1 and N1, auditory N1) did not display such marked differences between the 2 groups. This pattern of results is also corroborated by the regression analyses. The results pattern suggests that processes, which led to the serial/parallel differentiation of response selection, are unlikely to be in effect at earlier stages of attentional modality-specific stimulus processing. Rather, the differentiation seems to evolve during the later stage of multimodal stimulus analysis and –evaluation and integration and is mediated by structures constituting the MD system (i.e., ACC and TPJ) (Duncan 2010). The results suggest that interindividual differences in the processing mode used to cope with situations in which action cascading is necessary are mediated via a distributed fronto-parietal network modulating response control, but not attentional processes.

Supplementary Material

Supplementary material can be found at: http://www.cercor.oxfordjournals.org/.

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