NeuroImage 96 (2014) 95–105

Contents lists available at ScienceDirect

NeuroImage

journal homepage: www.elsevier.com/locate/ynimg

Trial-by-trial fluctuations in CNV amplitude reflect anticipatory adjustment of response caution

Udo Boehm [a,*], Leendert van Maanen [b,1], Birte Forstmann [b], Hedderik van Rijn [a]

[a] Department of Experimental Psychology, University of Groningen, 9712 TS Groningen, The Netherlands
[b] Cognitive Science Center Amsterdam, University of Amsterdam, 1018 VZ Amsterdam, The Netherlands

ARTICLE INFO

Article history:
Accepted 25 March 2014
Available online 1 April 2014

Keywords:
CNV
EEG
Linear ballistic accumulation
Response caution
Slow wave potentials
Speed-accuracy tradeoff

ABSTRACT

The contingent negative variation, a slow cortical potential, occurs when humans are warned by a stimulus about an upcoming task. The cognitive processes that give rise to this EEG potential are not yet well understood. To explain these processes, we adopt a recently developed theoretical framework from the area of perceptual decision-making. This framework assumes that the basal ganglia control the tradeoff between fast and accurate decision-making in the cortex. It suggests that an increase in cortical excitability serves to lower response caution, which results in faster but more error prone responding. We propose that the CNV reflects this increased cortical excitability. To test this hypothesis, we conducted an EEG experiment in which participants performed the random dot motion task either under speed or under accuracy stress. Our results show that trial-by-trial fluctuations in participants' response speed as well as model-based estimates of response caution correlated with single-trial CNV amplitude under conditions of speed but not accuracy stress. We conclude that the CNV might reflect adjustments of response caution, which serves to enhance quick decision-making.

Introduction

Events in the environment often have a natural order to them where events that are of significance to a human decision maker are embedded in a stream of predictable but non-significant events. Numerous studies have shown that decision makers can use knowledge of warning stimuli preceding significant events to prepare efficiently for the upcoming event, especially if only limited time is available to process the event (e.g., Leuthold et al., 2004; Mulckhuyse and Theeuwes, 2010; Praamstra, 2006; Wühr and Kunde, 2008).

The first description of the neural signature of the processes underlying this preparation effect, the contingent negative variation (CNV), was provided by Walter et al. (1964). They observed a slow buildup of a negative-going EEG potential if a warning stimulus preceded a response stimulus while no such build-up was observed if no warning was given. Despite numerous replications of the CNV effect, it is still unclear what the underlying cognitive processes are (e.g., Kononowicz and Van Rijn, 2014; Van Boxtel and Böcker, 2004).

Here we adopt a recently developed framework from the area of perceptual decision-making that can coherently account for the CNV effect. It suggests that decision makers adjust to stronger environmental limitations on decision time by trading off faster decisions for lower decision accuracy, which should be reflected in more negative CNV amplitudes.

Forstmann et al. (2008) suggested that the tradeoff between speeded and accurate deciding (Bogacz et al., 2010; Wickelgren, 1977), often referred to as response caution (Forstmann et al., 2008, 2010), is implemented by a brain circuitry involving cortical areas, the basal ganglia, and the thalamus. If speeded decisions are emphasized, optimal performance requires low response caution, promoting excitatory cortical input to the basal ganglia. As cortical processing starts to favor one course of action over another, the input nuclei of the basal ganglia (i.e., the striatum) become activated, leading to an inhibition of the basal ganglia's output nuclei (i.e., the globus pallidus interna and the substantia nigra pars reticularis). This releases the thalamus from its usual inhibited state and allows it to excite the cortex, thus facilitating the execution of the action that is being prepared.

Interestingly, modulations of cortical excitability, as suggested by Forstmann et al.'s (2008) model, might give rise to slow cortical EEG potentials such as the CNV (Birbaumer et al., 1990; Elbert, 1990). If quick decisions are required, the thalamus might partially depolarize cortical neurons to increase the speed at which a response option is selected. This suggests that, on average, an emphasis on speed, and hence lower response caution, should be associated with more negative CNV amplitudes. Moreover, under speed stress, trial-by-trial fluctuations in CNV amplitude should be associated with fluctuations in response caution.

* Corresponding author at: Department of Experimental Psychology, University of Groningen, Grote Kruisstraat 2/1, 9712TS Groningen, The Netherlands.
E-mail addresses: u.bohm@rug.nl (U. Boehm), l.vanmaanen@uva.nl (L. van Maanen), buforstmann@gmail.com (B. Forstmann), hedderik@van-rijn.org (H. van Rijn).
[1] LvM is now at Department of Psychological Methods, University of Amsterdam, The Netherlands.

http://dx.doi.org/10.1016/j.neuroimage.2014.03.063
1053-8119/

Suggestions that the CNV might reflect the degree to which humans are prepared to take action quickly have been made early on in the long-standing history of research on the CNV (e.g., Hillyard, 1969; Loveless and Sanford, 1974; Näätänen, 1970). Indeed, Loveless and Sanford (1974) suggested that the CNV reflects adjustments of a response criterion that determines how much sensory information decision makers gather before engaging in a decision. In their experiment they used speed instructions to manipulate how fast participants responded and found that under conditions of high speed stress, average response times were lower and average CNV amplitudes were higher. Similar results were reported by McAdam et al. (1969) and Loveless and Sanford (1973). Gaillard and Näätänen (1973) as well as Hillyard (1969) further showed that under speed stress, also on a single-trial basis higher CNV amplitudes are associated with shorter response times.

Although these earlier studies were based on notions similar to the idea suggested by the framework adopted here, there are a number of important points that were not addressed. Firstly, all of the above studies used detection or choice response time tasks that did not include any response-relevant ambiguity in the stimulus. This minimizes the time spent on the actual decision process, thus emphasizing quick motor responses rather than quick decision-making. Secondly, all studies only investigated the relationship between CNV and response time data but ignored the inherent dependence between response time and response accuracy suggested by the speed-accuracy tradeoff (Bogacz et al., 2010; Wickelgren, 1977). Thirdly, although Loveless and Sanford (1974) suggested that the CNV reflects the adjustment of a response criterion, they did not specify how that criterion relates quantitatively to response time. Finally, none of the studies provided an account of the physiological processes linking the CNV to the decision makers' preparedness to respond quickly.

One alternative account of the CNV posits that the CNV reflects motor preparation, rather than the adjustment of response caution. A number of authors have suggested that higher CNV amplitudes are due to a more advanced calibration of the motor programs needed to execute a response (e.g., Grünewald et al., 1979; MacKay and Bonnet, 1990; Rohrbaugh and Gaillard, 1983; Ulrich et al., 1998; see Leuthold et al., 2004, for a review). For example, Ulrich et al. (1998) showed that as more parameters of the required motor response (e.g., direction and force of the hand movement) were specified in advance by a warning stimulus, more negative CNV amplitudes could be observed.

This explanation can be examined using motor-related EEG potentials. According to the motor preparation account of the CNV, participants' level of motor preparation might be higher under time pressure, leading to very fast responses. The lateralized readiness potential is assumed to be a genuine marker of motor processes (Coles, 1989; De Jong et al., 1988; Kutas and Donchin, 1980) and can be exploited to assess whether experimental effects are due to cognitive processes preceding the preparation of a motor response or are due to processes related to the motor response itself (Leuthold et al., 2004; Osman et al., 1995). In particular, the time between stimulus onset and the onset of the stimulus-locked LRP (S-LRP) reflects premotor processes whereas the time between the onset of the response-locked LRP and the execution of the motor response (LRP-R) is assumed to reflect motor processes (Leuthold et al., 2004). Consequently, if decreases in response time and accuracy under time pressure are due to advanced motor preparation, rather than adjustments of response caution, decreased response times should be associated with shortened LRP-Rs.

In the present study we tested the hypotheses that, on average, more negative CNV amplitudes are associated with lower response caution and that trial-by-trial fluctuations in CNV amplitude reflect fluctuations in response caution, as predicted by the neurobiological model adopted here. Moreover, we examined whether advanced motor preparation, rather than lower response caution, is responsible for shorter response times, as would be indicated by a shortening of LRP-Rs under speed pressure. We asked participants to perform a two-choice motion discrimination task where they had to decide whether a noisy stimulus slowly drifted to the left or to the right. We then related participants' response times to the CNV amplitude on a single-trial basis. Moreover, we used a mathematical model that combines response time and accuracy data to quantify response caution in a principled manner. We obtained model-based single-trial estimates of participants' response caution and used these estimates to predict the CNV amplitude on a trial-by-trial basis.

Materials and methods

Participants

Twenty-five students from the University of Groningen (17 female) participated in our experiment for partial course credit. Two of these participants were excluded from further analysis because of non-compliance with the task instructions and 3 other participants were excluded because of a high number of EEG artifacts. The mean age was 21.3 years (range 17–25) and all but one participant were right-handed. All participants had normal or corrected-to-normal vision and no known neurological conditions. Written informed consent was obtained from all participants before the beginning of the experimental session. Ethical approval for the study was given by the Department of Psychology's ethics committee (file no. 12026-E).

Task

Participants performed the random dot motion task, which is a standard task in neuroscience research with human and primate subjects (Britten et al., 1992). In this task, participants are shown a cloud of pseudo-randomly moving dots and have to decide whether the cloud is moving to the left or right. The cloud used here measured 250 pixels in diameter and consisted of 120 dots with a diameter of 3 pixels. A proportion of the dots, which depended on the coherence level for the individual participant determined in the psychometric task (see Psychometric task and coherence estimation section), moved coherently to the left or right by 1 pixel per frame whereas the other dots were randomly displaced by 1 pixel. Each frame was presented for 50 ms and the total number of frames was 30, meaning that the cloud moved for 1.5 s in total.

Each trial (Fig. 1) started with a black screen, the presentation duration of which was drawn from a discrete uniform distribution from 0.5 to 2 s in steps of 0.5 s. After that a cue consisting of the letters “AC” or “SP” appeared on the screen for 2 s, informing participants to either respond as accurately (accuracy trial) or as quickly (speed trial) as possible, respectively. This was followed by another black screen that was again presented for a randomly selected interval from a uniform distribution from 0.5 to 2 s. Subsequently, a fixation cross appeared for 2 s, indicating that the trial was about to start. A cloud of random dots appeared thereafter and stayed on the screen for 1.5 s, during which participants had to give a response. After that another black screen was presented for a randomly selected interval between 0.5 and 2 s. Finally, participants were given feedback on their performance depending on the trial type. On AC trials, the word “correct” appeared if the participant had given the correct response and the word “incorrect” was shown if the response was wrong. On SP trials, participants saw the words “in time” if they responded within 0.5 s of random dot onset; otherwise they saw the words “too slow”. The feedback “no answer” was given if participants did not respond within 1.5 s after random dot onset, irrespective of the trial type.

Apparatus and experimental procedure

Participants were seated in an electrically shielded, dimly lit room. They were asked to put their head on a chin rest at a viewing distance of 60 cm from the screen to ensure good quality of the EEG recordings (see below). The task was administered using the Presentation®

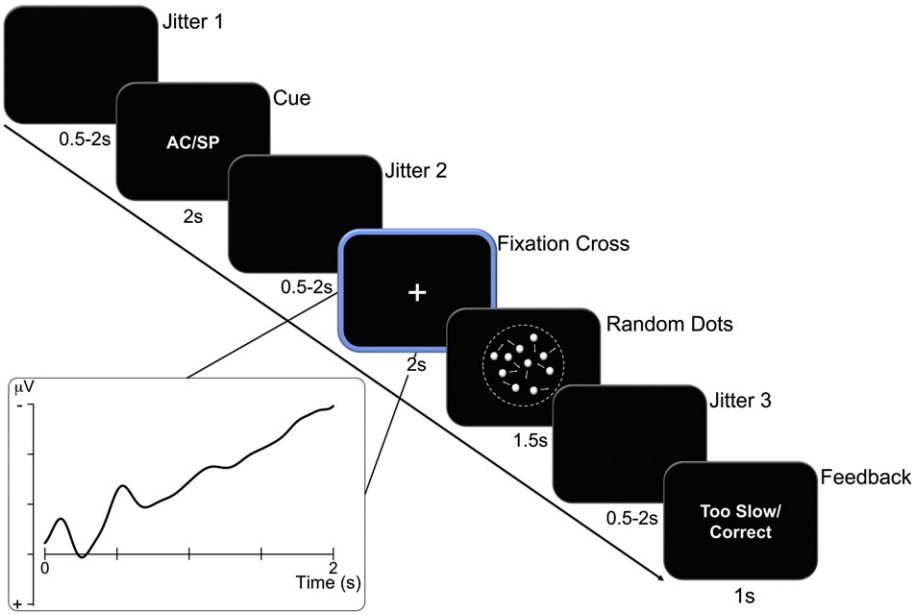

Fig. 1. Structure of the experimental task. Participants performed a random dot motion task where they had to decide whether a cloud of pseudo-randomly moving dots was drifting to the left or to the right. The blue frame indicates the period during which the CNV was measured.

experimental software system (Version 14.9, www.neurobs.com). Stimuli were presented on an Iiyama Vision Master Pro 513, 22in screen with a resolution of 1024 × 768 pixel and a refresh rate setting of 100 Hz. The cloud stimulus subtended a visual angle of 0.16°. Participants were instructed to press the response keys "V" and "M" if they thought the cloud was moving to the left or right, respectively. They were asked to use their left and right index fingers to press the buttons.

The experimental session started with a demographic questionnaire after which participants performed 5 blocks of 40 trials of a psychometric task to estimate the motion coherence at which they performed at about 80% correct. After that participants were prepared for the EEG recordings. Subsequently, they were given task instructions and performed 200 trials of the experimental task. Trials were presented in blocks of 40 trials between which a participant-controlled break was given. The order of SP and AC trials was completely randomized.

Psychometric task and coherence estimation

The psychometric task used the same random dot stimuli as the experimental task. For each trial the motion coherence was randomly selected from a set ranging from 5 to 80% (i.e., 5, 10, 15, 20, 25, 40, or 80%). Participants were instructed to respond as quickly and as accurately as possible. Trials started with a fixation cross that was presented for 0.5 s. After that a random dot stimulus was presented for 1.5 s, followed by a black screen for 0.5 s and a feedback screen presented for 0.5 s, informing participants whether their response was correct or incorrect.

We used the psychometric function obtained from a drift diffusion model to adjust the motion coherence for each participant. A psychometric function links the strength of a stimulus (here motion coherence) to an individual's probability of a correct response. To obtain an estimate of this function for each participant, we fitted the symmetric-bounds drift diffusion model described by Palmer et al. (2005) to the data from the psychometric task. We then set the coherence for the experimental task to the coherence level at which the psychometric function intersects the 80% accuracy criterion.

EEG data acquisition and preprocessing

Data were recorded with the TMS amplifier system and electrode cap (Electrodecap, Inc.) at 500 Hz using an average reference. Data were algebraically rereferenced to the mastoid electrodes after recording. Thirty scalp electrodes were placed according to the international 10–20 system at locations FP1, FP2, AFz, F7, F3, Fz, F4, F8, T7, C3, Cz, C4, T8, P7, P3, Pz, P4, P8, O1, O2, FC1, FC5, FCz, FC2, FC6, CP5, CP1, CPz, CP2, and CP6. Reference electrodes were placed at the mastoids. EOG recordings were obtained from two horizontal electrodes placed at the outer canthi of both eyes and two vertical electrodes placed above and below the left eye. Impedance values were kept below 10 kΩ.

The data were preprocessed using the Matlab-toolbox FieldTrip (Oostenveld et al., 2011). Trials were defined as segments from 5 s before the onset of the moving dots until 3 s after moving dots offset. Trials with an amplitude exceeding −300 μV or 300 μV or with clipping artifacts with a duration of more than 10 ms were excluded from further analysis. Jump and muscle artifacts were identified using FieldTrip's built-in artifact rejection function which is based on z-standardized amplitude values per channel. Jump artifacts were defined as trials with a z-value higher than 50; muscle artifacts were identified by band-pass filtering the data between 110 and 140 Hz and removing trials with a z-value higher than 25. This resulted in the removal of an average of 28 trials per participant (minimum = 14, maximum = 55).

After artifact rejection was completed, the data were low-pass filtered at 35 Hz. Independent component analysis was used to correct eye blink artifacts. This method has been shown to be efficient at detecting artifacts and to better recover the true brain activity underneath the artifact than other methods (Jung et al., 2000). The data were subsequently baseline-corrected to the mean amplitude during the baseline-window from 300 to 100 ms before the onset of the fixation cross. Unless indicated otherwise all further analyses reported here were carried out based on electrode 'FCz' as the CNV is usually measured on midline electrodes (e.g., Cui et al., 2000; Kononowicz and Van Rijn, 2011).

ERP analysis

For the ERP analysis the CNV amplitude was determined by computing the mean amplitude over a window from 200 ms to 100 ms before the onset of the moving dots on a single-trial basis. As pointed out by Luck (2005), the mean amplitude has a number of advantages over the peak amplitude, most importantly an expected value that is independent of noise. The measurement window was chosen close to the onset of the moving dots because the CNV amplitude was expected to

be more stable later during the preparation process and more representative of the state of excitation at the onset of the moving dots. Exploratory analyses with different measurement windows during the late CNV or different electrodes did not yield qualitatively different results and are therefore not reported in detail here.

Statistical analysis using linear mixed-effects and general additive models

Three trials with response times below 200 ms were excluded from the statistical analysis because they likely do not reflect the decision process of interest. We used linear mixed effects (LME) models for our statistical analysis of the single-trial relationship between response time and CNV amplitude and the relationship between response caution and CNV amplitude as these models account most appropriately for our experimental design (see Pinheiro and Bates, 2000, for an introduction). LME models are regression models that model the data on multiple levels. In our analysis, fixed effects estimated by the models describe the overall relationship between single-trial response time and CNV amplitude across participants. Random effects describe the deviation of the relationship between CNV amplitude and response time found within each individual participant from the overall relationship described by the corresponding fixed effect.

Additionally, we used general additive models (GAMs) to explore the specific shape of the relationship between CNV amplitude and response time as these models can account for non-linear relationships of any kind (see Wood, 2006, for an introduction). GAMs represent a dependent variable as the sum of a set of linear predictor variables and a set of smooth functions of covariates. This latter part of the model, the smooth functions, allows the model to account for any degree of non-linearity in the relationship between dependent variable and covariates. To do so, the relationship between dependent variable and covariates is represented in terms of a regression spline basis. The fitting is done under a penalized least squares criterion that penalizes the lack of smoothness of the resulting curve. That means smooth curves are preferred over wiggly curves as a description of the relationship between dependent variable and covariates. This property makes GAMs a suitable tool for exploring non-linear relationships while avoiding overfitting.

One important strength of these modeling approaches is that they allow for formal model comparisons. That means the contribution of different model components to a model's account of the data can be evaluated in a principled and systematic manner. A model that does include a particular component can be compared to a model that does not include that particular component in order to assess whether the model's improved fit to the data merits the added complexity associated with the inclusion of that component.

Model-based estimation of response caution

The linear ballistic accumulation model (LBA; Fig. 2; Brown and Heathcote, 2008) was fit to participants' response time and accuracy data to obtain estimates of their response caution. The model conceptualizes decision-making as an evidence accumulation process over time. Separate accumulators for each decision alternative accumulate evidence at a fixed rate v until a decision boundary b is reached. The alternative for which the evidence reaches the boundary first is eventually selected. To account for variability in response times, the model assumes drift rates across trials to be normally distributed with variance s and mean v. Moreover, the starting point of the accumulation process is assumed to have a uniform distribution between 0 and an upper bound A. The model also estimates a component t_0 (the non-decision time) that accounts for all processes not related to the decision itself (e.g., execution of the motor response).

Response caution in the model is defined as the distance between the upper bound of the starting point distribution and the decision boundary, b–A. If the distance is high, response caution is high and the accumulation processes for all alternatives start well below the decision threshold. This results in high accuracies at the expense of long response times. If, on the other hand, the distance is low, response caution is low and the starting point for the incorrect alternative might be sampled very close to the decision boundary. In this case the evidence for the incorrect choice might reach the threshold first, resulting in lower accuracies and shorter response times.

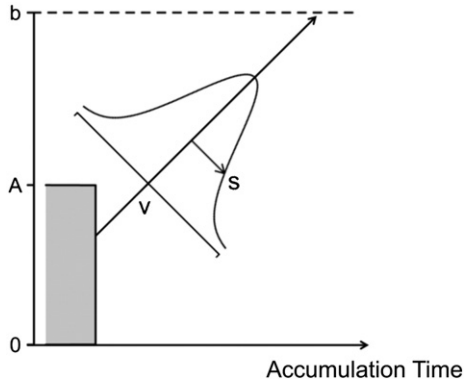

Fig. 2. Linear ballistic accumulation model (adapted from Brown and Heathcote, 2008). A random starting point for the evidence accumulation is drawn from a uniform distribution between 0 and A. Evidence is accumulated at a fixed rate drawn from a normal distribution with mean v and standard deviation s until the decision threshold b is reached.

A recent extension of the LBA model, the single-trial LBA model (STLBA; Ho et al., 2012; Van Maanen et al., 2011), was used to obtain estimates of response caution a_i and drift rate d_i for every trial for every participant. Following the procedure suggested by Van Maanen et al. (2011), we first fit the standard LBA model to the .1, .3, .5, .7, and .9 quantiles of the response time distributions for SP and AC for each participant (Donkin et al., 2011). Trials with response times shorter than 200 ms or longer than 1500 ms were removed as they are likely not due to the decision process of interest (4.5% of the data). To determine which of the five LBA model parameters (v, s, A, b, and t_0) should be free to vary between experimental conditions, all possible models that can be created by freeing any subset of the five parameters were fit to the data. All models were then compared using Schwarz weights (Raftery, 1996; Schwarz, 1978). These weights are computed based on the Bayesian information criterion (BIC) and higher weights imply stronger evidence in favor of a model. We computed the average of the Schwarz weights for each model across participants and selected the model with the highest average weight. The LBA parameter estimates of the best-fitting model were then used to obtain maximum likelihood estimates of single-trial response caution and drift rate for every participant. As with the response time data, we used LME models to investigate the relationship between these single-trial estimates and the CNV amplitude.

Results

Behavior

Our behavioral data exhibited the typical patterns usually observed in the random dot motion task (Fig. 3). Participants' accuracies were lower on SP compared to AC trials (we applied an arcsine transformation to account for the non-normality of accuracy scores; $t\ (19) = 8.07$, $p < .01$, $M_{diff} = 0.15$, $SE_{diff} = 0.01$). We analyzed the response time data with a 2×2 repeated measures ANOVA with a factor for accuracy (incorrect, correct) and a factor for experimental condition (SP, AC). The average response times in SP were shorter than the average response times in AC, which was indicated by a significant main effect of experimental condition ($F\ (1,19) = 172.14$, $p < .01$, $\eta^2_{partial} = .90$). Moreover, a significant disordinal interaction between accuracy and experimental condition ($F\ (1,19) = 25.28$, $p < .01$, $\eta^2_{partial} = .57$) suggests

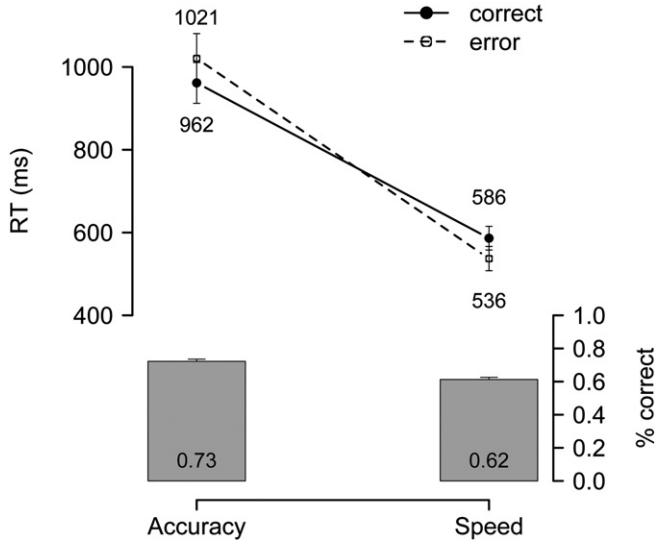

Fig. 3. Mean accuracies and response times for the speed (SP) and accuracy (AC) condition. Numbers show the mean response times and accuracies. Error bars show 1 SE within-subjects (Loftus and Masson, 1994). Standard errors for response times were computed separately for each cell mean according to formula (4) in Loftus & Masson because ANOVA error terms varied between factors.

that errors tended to be faster than correct responses in SP while correct responses were faster than errors in AC. This was confirmed by post-hoc t-tests that showed that responses were significantly faster for errors than correct responses in SP (t (19) = −5.57, p < .01, M_{diff} = −49.33, SE_{diff} = 8.86) while correct responses were faster than errors in AC (t (19) = −3.14, p < .01, M_{diff} = 59.06, SE_{diff} = 18.83). A nonsignificant main effect of accuracy did not provide any evidence for a difference in average response times between correct and error responses (F (1,19) < 1, p = .63, $\eta^2_{partial}$ = .01).

Further, we tested whether participants performed the experimental task in SP over the whole range of response times or whether fast response times were due to participants ignoring the random dot motion task completely and responding based on guesses. That is, we tested whether participants' accuracy was above chance for all response times in SP. Therefore, we split each participant's SP response times into five quantiles (.2, .4, .6, .8, 1.0) and tested whether their arcsine-transformed accuracy was higher than 50%. Although not significant for the first two quantiles, the accuracies tended to be slightly above chance and were significantly so for the last three quantiles ($q_{.2}$: t (19) = 1.30, p = .21, M = .53, SE = 0.02; $q_{.4}$: t (19) = 1.72, p = .10, M = .54, SE = 0.03; $q_{.6}$: t (19) = 5.32, p < .01, M = .62, SE = 0.02; $q_{.8}$: t (19) = 4.24, p = <.01, M = .66, SE = 0.03; $q_{1.0}$: t (19) = 5.98, p = <.01, M = .71, SE = 0.03).

Electroencephalographic recordings

The averaged EEG data showed a higher negativity in SP than in AC during the preparation period while there was no difference during the presentation of the random dot kinematogram (Fig. 4). The typical early deflections (i.e., N1, e.g., Haider et al., 1964; P2, e.g., Hillyard and Münte, 1984) were clearly visible shortly after the onset of the preparation period preceding the onset of the random dot kinematogram. The CNV slowly built up during the preparation period in both experimental conditions. It can be seen that the CNV amplitude during the measurement window, marked in red, was higher for SP than for AC (t (19) = 3.78, p = .01, M_{diff} = 3.16 μV, SE_{diff} = 0.86). After the early sensory potentials in response to the onset of the random dot kinematogram, the EEG activity reached similar levels in both conditions. An analysis of the average EEG activity between 200 and 100 ms before participants' response showed no evidence for a difference in amplitude (t (19) = 1.23, p = .24, M_{diff} = 0.81 μV, SE_{diff} = 0.66). We excluded all trials with response times smaller than 500 ms from the computation of the mean amplitude to avoid confounds caused by the early visual potentials overlapping with later EEG activity. This resulted on average in the removal of 36 trials in SP and 2 trials in AC, leaving on average 48 trials per participant in SP and 82 trials per participant in AC for the computation of the mean amplitude. Comparisons of mean amplitude measures that are based on different numbers of trials are unproblematic as the expected value of the mean amplitude is independent of noise introduced by variation in the number of trials (Luck, 2005). Despite the relatively large difference in the number of remaining trials between SP and AC the data basis was sufficiently large to compute reliable estimates of the mean amplitude.

The negative voltage associated with the CNV (Fig. 5) focused on central midline electrodes but also spread laterally to more temporal and parietal areas. Although a slight lateralization was visible, it was not statistically significant in a repeated measures ANOVA with factors response hand (left, right), experimental condition (SP, AC), electrode (FC5, FC6), and the mean CNV amplitude as dependent variable. Although there was a tendency for the CNV amplitude to be more negative at FC5, the main effect of electrode (F (1,19) = 3.91, p = 0.06, $\eta^2_{partial}$ = .17) was not significant, nor were there any significant interactions with electrode (F (1,19) ≤ 1, p ≥ .40, $\eta^2_{partial}$ ≤ .04). An analysis based on C3 and C4 instead of FC5 and FC6 showed similar

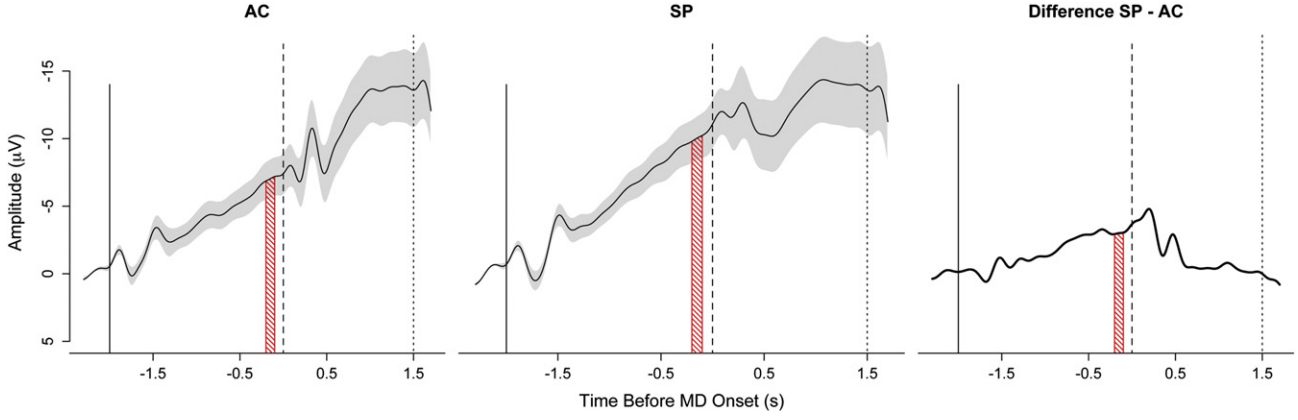

Fig. 4. EEG data aligned to the onset of the moving dots, averaged across participants for SP, AC and the difference waveform SP–AC, low-pass filtered at 5 Hz. The red striped area indicates the measurement window for the mean amplitude. The gray shaded area indicates 1 SE. The lines indicate fixation onset (solid), moving dots onset (dashed), and moving dots offset (dotted).

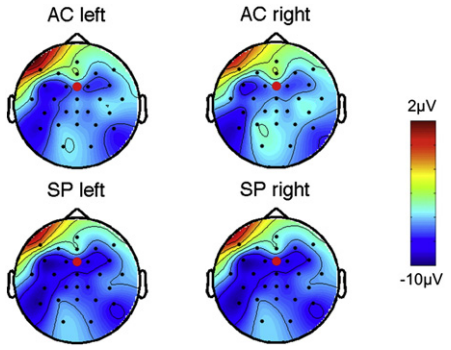

Fig. 5. Topographic maps for the EEG data for SP and AC trials and left and right-hand responses. Data are averaged from 200 ms to 100 ms before moving dots onset. Dots indicate the positions of the electrodes, the red dot indicates FCz.

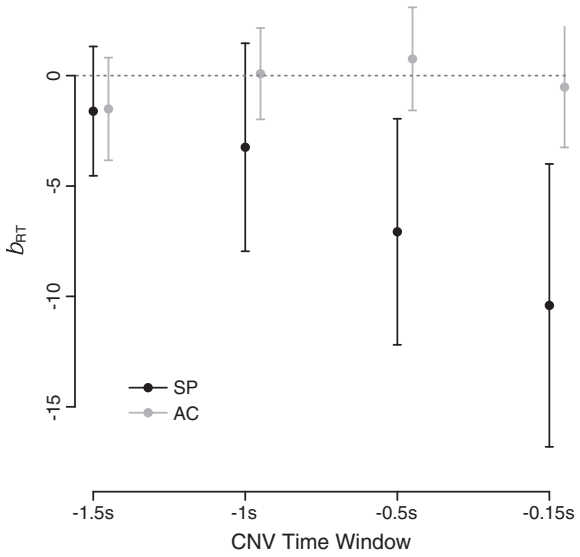

Fig. 6. Development of the beta regression weights for RT in SP and AC as the CNV built up. Beta weights are based on the RT × Cond model. Regression weights for AC were obtained by recoding AC to 0 and SP to 1 in the LME model. Error bars show approximate 95% confidence intervals based on the t-statistics computed by the lme4 R-package (Bates et al., 2013).

results (main effect of electrode: $F(1,19) \leq 1, p = .49, \eta^2_{partial} = .03$; interaction effects with electrode: $F(1,19) \leq 3.12, p \geq .09, \eta^2_{partial} \leq .14$). Similar to the ERP plots shown in Fig. 4, a stronger negativity was visible in SP than in AC.

Single-trial CNV amplitude and response speed

Our statistical analysis of the relationship between CNV and response time showed that, on a single trial level, shorter response times were associated with more negative CNV amplitudes in SP but not in AC. We used a hierarchical modeling approach with LME models that used negative CNV amplitude as dependent variable. Our first model, the baseline model, included a fixed effect for experimental condition as well as a random effect for experimental condition (coded as 0 for SP and 1 for AC) per participant. Our second model, the RT model, additionally included a fixed and a random effect for response time but no interaction between response time and experimental condition. A formal comparison of these two models (Table 1) showed that including response time as a predictor increased the fit of the model to the data, which means that response time correlated with CNV amplitude on a single-trial basis. AIC as well as a chi-squared test preferred the second model over the first model ($\Delta AIC = 16$; $\chi^2(4) = 24.47, p < .01$).

We also considered a model that only included a fixed and a random effect for response time but not for experimental condition. A formal comparison with the RT model showed that the RT model was superior to the model without any effects for experimental condition ($\Delta AIC = 5$; $\chi^2(4) = 24.47, p = .01$). This means that response time alone, just as experimental condition alone, did not provide a sufficient account for trial-by-trial fluctuations in CNV amplitude.

We tested further whether response time was a better predictor of CNV amplitude in SP than in AC. Therefore, we created a third model, the RT × Cond model, that included a fixed and a random effect for the interaction between response time and experimental condition in addition to the predictors included in the RT model. AIC as well as a chi-squared test preferred the RT × Cond model over the RT model ($\Delta AIC = 21$; $\chi^2(5) = 31.16, p < .01$). This result implies that the strength of the relationship between response time and CNV amplitude differed between SP and AC. Inspection of the estimated model parameters shows that the relationship between response time and CNV amplitude was stronger in SP than in AC. The regression weight for SP (i.e., $\hat{\beta}_{RT}$) means that a decrease in response time of 100 ms was associated with an increase in CNV amplitude (i.e., a more negative CNV) of about 1.0 μV. The regression weight for AC (i.e., $\hat{\beta}_{RT} + \hat{\beta}_{RT \times Condition}$), on the other hand, means that a decrease in response time of 100 ms was associated with an increase in CNV amplitude of less than 0.1 μV.

An exploratory analysis using different time windows showed that these results held for all but the earliest parts of the CNV. Moreover, using a different electrode, Cz, did not yield qualitatively different results. We computed the mean CNV amplitude over three additional time windows, namely from 0.55 s to 0.45 s, from 1.05 s to 0.95 s, and from 1.55 s to 1.45 s before the onset of the moving dots. The results showed that in all cases except the earliest time window the RT × Cond model was superior to the RT model and the RT model was superior to the baseline model (see Table S2 for the details). Furthermore, the RT × Cond model's estimated beta regression weight for RT in SP (Fig. 6) increased in size over consecutive time windows, meaning that response time became a better predictor of the CNV amplitude as the CNV built up. In AC, on the other hand, the estimated regression weight stayed closed to zero and did not become a good predictor of CNV amplitude even during the late CNV. We further repeated the above analyses for electrode Cz. The results showed the same qualitative patterns, namely that the RT × Cond model was superior to the RT model and the RT model was superior to the baseline model for the two late time windows. During the two earliest time windows the RT model was preferred over the baseline model as well as the RT × Cond model (see Table S3 for the details).

Table 1
Model parameters for linear mixed effects of the relationship between response time and CNV amplitude. Experimental condition is dummy-coded as SP = 0 and AC = 1.

	Baseline model		RT model		RT × Cond model	
AIC	28,566		28,550		28,529	
	$\hat{\beta}$ (SE)	t-Value	$\hat{\beta}$ (SE)	t-Value	$\hat{\beta}$ (SE)	t-Value
Intercept	10.021 (1.405)	7.134	11.504 (2.036)	5.650	15.288 (2.864)	5.338
Condition	−3.157 (0.836)	−3.774	−1.929 (0.839)	−2.300	−8.404 (2.965)	−2.835
RT			−0.003 (0.004)	−2.054	−0.010 (0.003)	−3.186
RT × Condition					0.001 (0.003)	3.114

An additional analysis that took into account individual differences in the location of the electrodes showing the experimental effect most clearly confirmed the qualitative results of the previous analyses. Due to individual differences in anatomy, the electrodes showing the effect of response caution on CNV amplitude might differ between participants. To account for this fact, we determined the electrode per participant from a band around the midline (i.e., Cz, Pz, FC1, FCz, FC2, CP1, CPz, and CP2) that showed the largest mean AC–SP difference. We entered the single-trial CNV amplitude from the corresponding electrode per participant for each of the four time windows described above as the dependent variable into an LME analysis. The results showed that for the latest time window the RT × Cond model was superior to the RT model and the RT model was superior to the baseline model (see Table S4 for details). For the earlier time windows no clear pattern emerged which is presumably due to the additional noise introduced by including different electrodes per participant. Because some electrodes were only included for one or two participants in the analysis, the additional noise could not be accounted for by a random effect for electrode location.

Nonlinear relationship between CNV amplitude and response time

The single-trial relationship between CNV amplitude and response time (Fig. 7) was stronger for short than for long response times in SP while there was no relationship in AC. This raises the question whether the relationship between response time and CNV amplitude in SP found in the LME analysis might merely be driven by the strong relationship for short response times. We explored this relationship using GAMs. Firstly, we tested whether the difference between experimental conditions we found in the LME-based analysis was statistically reliable once any nonlinearities in the data were taken into account in the model. Therefore, we created a first model that included a smooth (i.e., nonlinear) overall term for response time (analogous to a fixed effect in LME models, fit using cubic regression splines, cr) and a smooth term for response time per participant (analogous to a random effect in LME models, fit using smooth factors with penalties on each null space component, fs; Wood, 2013). All parameters were estimated using restricted maximum likelihood (REML) estimation. Our second model included a smooth term for response time that was fit separately for each experimental condition (equivalent to a RT × experimental condition interaction and main effects for RT and experimental condition in LME models, cr) and a smooth term for response time per participant (fs). A formal model comparison showed that the second model did not provide a better fit for our data than the first model (Model 1 effective degrees of freedom $EDF = 37.53$, deviance explained $r^2_{dev} = .15$, AIC = 28,442; Model 2 $EDF = 36.685$, $r^2_{dev} = .15$, $AIC = 28{,}450$; a chi-squared test was not be computed as the explained deviance for the more complex model was lower than that for the simpler model). This means that there was no evidence for different nonlinear relationships in the two experimental conditions.

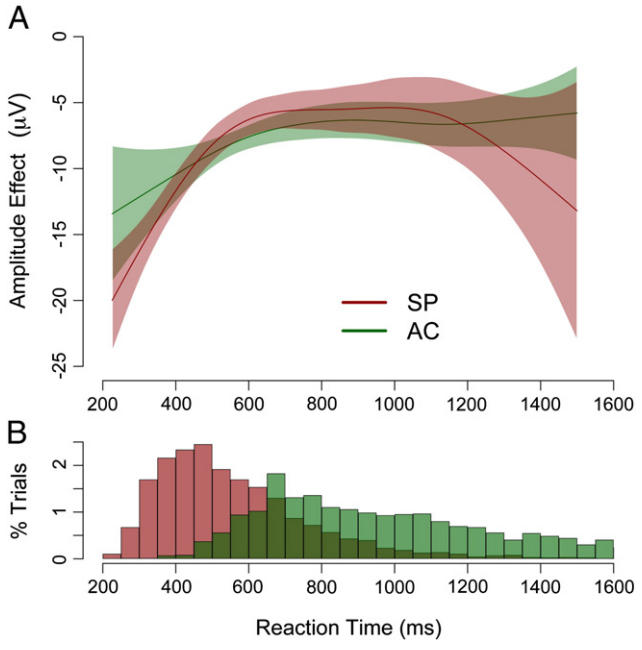

Fig. 7. A) General additive model (GAM) fit for the relationship between response time and CNV amplitude. GAMs model the dependent variable as the sum of a set linear terms and a set of smooth spline functions of the predictors. Model fitting is based on likelihood maximization that penalizes models with a lack of smoothness more heavily than models with a high degree of smoothness. The model included a smooth term for response time for each experimental condition as well as a smooth term for response time per participant as predictors. Lines show the model fit (SP red, AC green). Shaded areas show 1 SE of the model's predictions. B) Histograms of the response time distribution across participants for SP (red) and AC (green).

Secondly, we tested whether a simpler model that assumes no nonlinearities in AC could account for our data. Therefore, we created a third model that included a linear overall effect of response time, a smooth term for response time for SP (cr) but not for AC and a smooth term for overall response time for each participant (fs). A comparison of the first and the third model showed that the third model provided a better account of the data ($\chi^2(0.55) = 2.52$, $p = .05$, Model 1 $EDF = 37.53$, $r^2_{dev} = .15$, $AIC = 28{,}442$; Model 3 $EDF = 38.08$, $r^2_{dev} = .15$, $AIC = 28{,}440$). This means that there was evidence against a nonlinear effect in the AC condition.

Thirdly, we tested whether there was any relationship between CNV amplitude and response time in AC at all. To address this question, we created a fourth model that included the same smooth terms for response time in SP (cr) and per participant (fs) as the third model but did not include a linear effect of response time. That is, the model did not include any effect of response time for AC. A model comparison showed that there was no difference between the third and the fourth model ($\chi^2(0.864) = 0.83$, $p = .31$, Model 3 $EDF = 38.08$, $r^2_{dev} = .15$, $AIC = 28{,}440$; Model 4 $EDF = 37.22$, $r^2_{dev} = .15$, $AIC = 28{,}440$), which means that there was no evidence for any relationship between CNV amplitude and response time in AC. This can also be seen from Fig. 6 where the standard errors for AC nearly completely overlap a straight line with a zero-slope that could be drawn at about −7 μV.

LBA model fit

The best account for the data was provided by a model with response caution and starting point free to vary between conditions (average Schwarz weight 0.12, average BIC 944). Compared to the full model with all parameters free to vary (average Schwarz weight .02, average BIC 950), the selected model had only a small loss of information while being more parsimonious. At the same time, the model was clearly superior to the simplest model with all parameters fixed (average Schwarz weight < .01, average BIC 1042).

The model predictions for the defective cumulative response time distribution (see e.g., Brown and Heathcote, 2008) averaged across participants aligned well with the averaged empirical distribution (Fig. 8). Comparing the estimated free model parameters across participants showed that response caution was generally higher in AC than in SP ($t\ (19) = 8.75$, $p < .01$, $M_{diff} = 174.51$, $SE_{diff} = 20.47$), whereas the starting point did not vary systematically between conditions ($t\ (19) = 1.67$, $p = .11$, $M_{diff} = 72.96$, $SE_{diff} = 44.71$).

Single-trial response caution and CNV amplitude

The LME and GAM-based analyses showed that longer response times are associated with lower CNV amplitudes in SP while there was no evidence for such a relationship in AC. Moreover, the relationship in SP was stronger for shorter response times. While those analyses

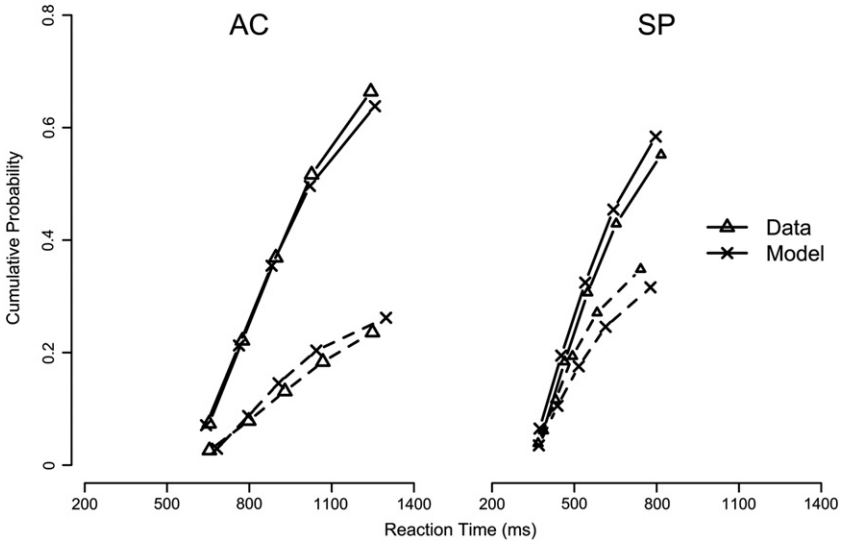

Fig. 8. Defective cumulative response time distribution (see e.g. Brown and Heathcote, 2008) and model fit averaged across participants. Dashed lines show error trials, solid lines show correct trials. Points show the .1, .3, .5, .7 and .9 quantiles. Cumulative probability is the cumulative number of correct or error trials with response times lower than or equal to a certain response time relative to the total number of trials.

only took into consideration the raw response times, the single-trial linear ballistic accumulation model combines response time and accuracy data, both of which should be related to response caution, to obtain estimates of single-trial response caution in a principled manner. In the following analysis we test whether these estimates also show a systematic relationship with CNV amplitude.

On a single trial level, lower response caution was associated with more negative CNV amplitudes in SP but not in AC. We assessed the relationship between CNV and response caution statistically using LME models. All models used negative CNV amplitude as dependent variable. Trials with negative drift rate estimates were excluded from the analysis (1.2% of the trials). The baseline model included a fixed and a random effect for experimental condition (coded as 0 for SP and 1 for AC). The second model, the RC (response caution) model, additionally included a fixed and a random effect for response caution but no interaction between response caution and experimental condition. There was no statistical evidence for a difference between the two models ($\Delta AIC = 0$; $\chi^2(4) = 8.69$, $p = .07$; see also Table 2), meaning that response caution in general was not a good predictor of single-trial CNV amplitude.

We also considered a model that only included a fixed and a random effect for response caution but not for experimental condition. A formal comparison with the RC model showed that the RC model was superior to the model without any effects for experimental condition ($\Delta AIC = 37$; $\chi^2(4) = 45.49$, $p < .01$). This means that response time alone, just like experimental condition alone, did not provide a good account for trial-by-trial fluctuations in CNV amplitude.

A further model comparison showed that response caution was a predictor of CNV amplitude in SP but not in AC. We created a third model, the RC × Cond model, that included a fixed and a random effect for the interaction between response caution and experimental condition in addition to the predictors included in the RC model. A formal model comparison preferred the RC × Cond over the RC model ($\Delta AIC = 24$; $\chi^2(5) = 33.30$, $p < .01$) as well as the baseline model ($\Delta AIC = 24$; $\chi^2(5) = 41.99$, $p < .01$). This result implies that response caution was related to CNV amplitude in SP ($\hat{\beta}_{RC} = 0.024$) but not in AC ($\hat{\beta}_{RC} + \hat{\beta}_{RC\times Condition} = -0.002$). A comparable analysis with a model that used single-trial drift rate and its interaction with experimental condition as predictors showed that drift rate by itself was not a good predictor of CNV amplitude but contained additional information about CNV amplitude beyond that conveyed by single-trial response caution (see Supplementary material).

Similar to the results of the response time-based analysis, an exploratory analysis using different time windows showed that these results held for different parts of the late CNV but not for the early CNV. Moreover, using a different electrode confirmed these qualitative patterns. We computed the mean CNV amplitude over three additional time windows, namely from 0.55 s to 0.45 s, from 1.05 s to 0.95 s, and from 1.55 s to 1.45 s before the onset of the moving dots. The RC × Cond model was preferred over the RC as well as the baseline model during the 0.55 s to 0.45 s window while the baseline model was the preferred model during the two earliest time windows (see Table S5 for further details). We further repeated the above analyses for electrode Cz which led to comparable results. The RC × Cond model was superior to the other two models for the two late time windows while the baseline model was the preferred model during the two early time windows (see Table S6 for the details).

Finally, we examined whether response caution explains additional variance in CNV amplitude beyond that explained by the raw response times. A formal comparison showed the RC × Cond model to be superior to the RT × Cond model ($\Delta AIC = 2$; a chi-squared test could not be computed because the RT × Cond and the RC × Cond model have the same number of degrees of freedom). This means that response caution was a better predictor of CNV amplitude than raw response time.

LRP onset and response speed

We examined the LRP-R based on the grand-average LRPs on SP trials with more positive and more negative CNV amplitudes in the response time range from 200 to 600 ms where we observed the strongest relationship between CNV amplitude and response time (see Fig. 7). Following the standard procedure for the computation of LRPs (Müller-Gethmann et al., 2003; Leuthold, et al., 1996; Osman, et al., 1995) we obtained single-trial LRPs by subtracting the waveform at C3 from the waveform at C4 for left-hand responses and vice versa for right hand response. The data for each participant were median-split into a group with more positive and a group with more negative-CNV amplitudes and the grand average LRP was computed for each half of the data. The response times for more negative-CNV amplitude trials were on average 24 ms faster than the response times for the more positive-CNV trials ($t(19) = -4.00$, $p < .01$, $SE_{diff} = 6.06$). We defined LRP-R onset as the point in time when the amplitude of the LRP crossed the −0.5 μV criterion (e.g., Miller et al., 1998; Ulrich, and Miller, 2001). The LRP-Rs did not differ significantly between the two groups of trials

Table 2
Model parameters for linear mixed effects models of the relationship between response caution and CNV amplitude. Experimental condition is dummy-coded as SP = 0 and AC = 1.

	Baseline model		RT model		RT × Cond model	
AIC	26,862		26,862		26,838	
	$\hat{\beta}$ (SE)	t-Value	$\hat{\beta}$ (SE)	t-Value	$\hat{\beta}$ (SE)	t-Value
Intercept	9.942 (1.400)	7.100	8.740 (1.294)	6.753	5.160 (1.167)	4.422
Condition	−3.079 (0.853)	−3.609	−3.615 (0.965)	−3.746	1.858 (1.576)	1.179
RC			0.006 (0.003)	1.851	0.024 (0.007)	3.469
RC × Condition					−0.025 (0.008)	−3.037

and the average difference in LRP-Rs of 4 ms (Fig. 9) could not account for the much larger differences in average response times (t (19) = −0.28, p = .39, SE_{diff} = 14.45; the standard error was computed using the jackknife-based method suggested by Miller et al., 1998; Ulrich, and Miller, 2001; JZS Bayes Factor (H_0/H_A) = 5.65).

We did not examine the S-LRPs for differences between more positive and more negative-CNV amplitude trials because differences in S-LRPs could not be determined reliably. The fact that the variation in response times increases with increasing response times (e.g., Wagenmakers et al., 2005) together with the observation that the mean response times were significantly longer for more positive-CNV amplitude trials implies that the individual S-LRP onsets will also vary more for those trials. This, in turn, means that the grand-average S-LRP waveform will be wider for more positive-CNV amplitude trials and thus will cross the −0.5 μV criterion later due to the higher degree of temporal jittering alone (Luck, 2005).

Discussion

In this study we investigated whether the CNV reflects response caution adjustments in perceptual decision-making. We found that more negative CNV amplitudes were associated with shorter response times at the cost of lower accuracy. Our analysis showed that the average CNV amplitude was more negative in the speed than in the accuracy condition while average response times were shorter and average accuracies were lower in the speed than in the accuracy condition. Moreover, we found that on a single-trial level, more negative CNV amplitudes were associated with faster responses in the speed but not in the accuracy condition. These results held for different central electrodes and different time windows during the late CNV. In our model-based analysis we used the LBA model to obtain estimates of participants' response caution from their behavioral data (see also Forstmann et al., 2011). This analysis yielded results complementary to the analysis of the behavioral data. Across participants response caution was lower in the speed than in the accuracy condition, consistent with previous results (e.g., Forstmann et al., 2008, 2010; Mulder et al., 2013; Winkel et al., 2012). Moreover, estimates of single-trial response caution correlated with fluctuations in CNV amplitude in the speed but not in the accuracy condition. Again, these findings held across different time windows during the late CNV and different central electrodes. The findings from both, the analysis of the behavioral data and the model-based analysis, support the hypothesis that the CNV reflects the adjustment of the brain-circuitry underlying perceptual decision-making. This circuitry implements human decision makers' ability to change their decision policy in favor of quick but possibly faulty decisions in the face of environmental restrictions on processing time.

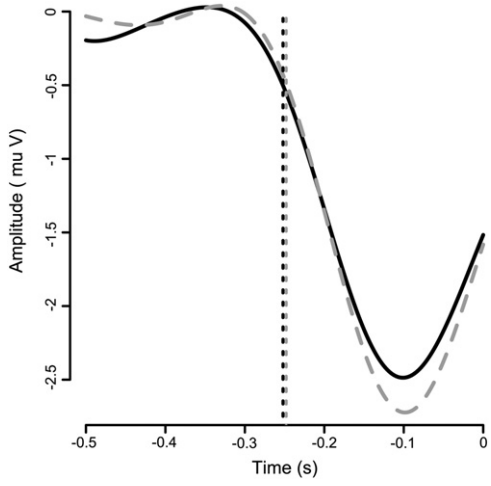

Fig. 9. Response-locked lateralized readiness potential (LRP-R) for SP trials with response times between 200 and 600 ms and CNV amplitudes below (solid black line) and above the median (dashed gray line). The vertical lines indicate the time point when the LRP exceeds the −0.5 μV criterion.

One alternative explanation for our findings might be that the CNV simply reflects motor preparation. On SP trials, fast responses might simply be due to more advanced motor preparation rather than adjustments of response caution. To test this possibility, we examined the time between the onset of the response-locked LRPs and response execution, which is assumed to reflect motor processes (Leuthold et al., 2004). According to the motor account of the CNV, more negative CNV amplitudes should be associated with a higher degree of motor preparation and hence shorter LRP-Rs. Examining the LRP-Rs of fast SP trials with more and less negative CNV amplitudes showed no difference between the two groups of trials. Therefore, it seems unlikely that advanced motor preparation can fully account for the difference in response times.

Additional evidence against a pure motor account of the CNV effects found here comes from the fact that the cues used in our experiment did not support any response-specific motor preparation. Most experiments relating the CNV to motor preparation employ warning stimuli that convey advance information about the parameters of the required motor response, such as the response hand, response finger (Leuthold et al., 1996), or required force (Ulrich et al., 1998). In the present experiment we used an uninformative cue that did not convey any information about motor parameters. It therefore seems unlikely that participants could have engaged in any specific motor preparation. Moreover, the fact that the response hand varied randomly from trial to trial makes it unlikely that the specific motor program used on the previous trial remained pre-activated, leading to a higher CNV and faster responses, as Bender et al. (2004) suggest. We are therefore convinced that, even though a contribution of very high-level motor preparation to the CNV effects reported here cannot fully be ruled out, these effects are not due to any effector specific motor preparation.

Our findings align well with the results of previous fMRI studies. For example, Forstmann et al. (2008) found that under speed instructions, a stronger BOLD response in the right anterior striatum and the right pre-SMA was associated with lower response caution. This result is similar to our finding that the speed condition is associated with a higher average CNV amplitude and lower response caution compared to the accuracy condition. Van Maanen et al. (2011) showed that, on a single-trial basis, the BOLD response from the pre-SMA, among other areas, correlates with participants' response caution under speed but not under

accuracy instructions. This result is analogous to our finding that the single-trial CNV amplitude correlates with response caution in the speed but not in the accuracy condition.

Speed-accuracy tradeoff is set before the decision process

Many studies investigating the physiological basis of the speed-accuracy tradeoff in decision-making rely on fMRI data (Forstmann et al., 2008, 2010; Ivanoff et al., 2008; Van Maanen et al., 2011; Van Veen et al., 2008; Winkel et al., 2012). Although these data provide insight into the brain structures involved in controlling the speed of information processing, they give little information about the time course of the underlying neural processes due to the sluggish nature of the BOLD response (Buckner, 1998). The present study provides exactly that temporal information. Our results show that the CNV, occurring before the onset of the decision process, predicts the degree to which participants give priority to quick deciding.

A recent fMRI-EEG co-registration study by Plichta et al. (2013) reported interesting complementary findings. This study used a reward anticipation paradigm in which participants were informed by a cue about whether a sufficiently fast response would earn them a reward. Following the cue, participants performed a simple signal detection task in which they had to detect a light flash as quickly as possible and respond with a button press. In the reward condition, a fast response earned them a monetary reward while in the control condition only verbal feedback was provided. Using dynamic causal modeling, Plitcha et al. found that the amplitude of the early CNV was best predicted by the activity of a reward anticipation network comprising of ventral Striatum and the Thalamus in connection with the supplementary motor area (SMA). In the reward condition, they found an excitatory information flow from thalamus through the ventral striatum to the SMA, which then gave rise to the early CNV. These findings are well in line with the neurophysiological model of decision-making proposed here. The early CNV might reflect the activity of a reward anticipation network which then informs the subsequent adjustment of response caution to ensure the potential reward is obtained. This latter process might then be reflected by the late CNV, as suggested by the present findings.

Conclusions

To conclude, our study shows more negative CNV amplitudes to reflect an increased speed of decision-making at the cost of lower accuracy. We were able to confirm this relationship not only on the basis of averaged data but we also found such a relationship on a single-trial level. These findings parallel the relationship between BOLD response from a number of areas close to the basal ganglia and the pre-SMA and response caution found in fMRI studies. Our EEG experiment extends these previous studies by providing the high temporal resolution that allows us to conclude that adjustments of response caution take place before decision makers engage in the decision process. Although the pre-SMA seems a likely source of the CNV, further studies will be needed to determine the exact neural source.

Our results also align well with the emerging view that the CNV reflects a general cognitive preparedness to process information (Leuthold et al., 2004; Van Boxtel and Böcker, 2004; Van Rijn et al., 2011). The neurobiological framework from the area of perceptual decision-making we adopted here promises to account for a wide variety of findings related to the CNV and to provide testable, quantitative predictions that will guide future research.

Acknowledgments

UB and HvR were supported by the European project COST ISCH Action TD0904 Time In MEntaL activitY: theoretical, behavioral, bioimaging, and clinical perspectives (TIMELY; www.timely-cost.eu). UB was supported by Netherlands Organisation for Scientific Research grant 406-12-125. BUF was supported by a Vidi grant from the Netherlands Organisation for Scientific Research and a European Research Council grant.

Appendix A. Supplementary data

Supplementary data to this article can be found online at http://dx.doi.org/10.1016/j.neuroimage.2014.03.063.

References

Bates, D., Maechler, M., Bolker, B., 2013. Linear mixed-effects models using S4 classes. R package Version 0.999999-2.
Bender, S., Resch, F., Weisbrod, M., Oelkers-Ax, R., 2004. Specific task anticipation versus unspecific orienting reaction during early contingent negative variation. Clin. Neurophysiol. 115 (8), 1836–1845.
Birbaumer, N., Elbert, T., Canavan, A.G.M., Rockstroh, B., 1990. Slow potentials of the cerebral cortex and behavior. Physiol. Rev. 70 (1), 1–41.
Bogacz, R., Hu, P.T., Holmes, P.J., Cohen, J.D., 2010. Do humans produce the speed-accuracy trade-off that maximizes reward rate? Q. J. Exp. Psychol. 63 (5), 863–891.
Britten, K.H., Shadlen, M.N., Newsome, W.T., Movshon, A.J., 1992. The analysis of visual motion: a comparison of neuronal and psychophysical performance. J. Neurosci. 12 (12), 4745–4765.
Brown, S.D., Heathcote, A., 2008. The simplest complete model of choice response time: linear ballistic accumulation. Cogn. Psychol. 57 (3), 153–178.
Buckner, R.L., 1998. Event-related fMRI and the hemodynamic response. Hum. Brain Mapp. 6 (5–6), 373–377.
Coles, M.G.H., 1989. Modern mind-reading: psychophysiology, physiology, and cognition. Psychophysiology 26 (3), 251–269.
Cui, R.Q., Egkher, A., Huter, D., Lang, W., Lindinger, G., Deecke, L., 2000. High resolution spatiotemporal analysis of the contingent negative variation in simple or complex motor tasks and a non-motor task. Clin. Neurophysiol. 111 (10), 1847–1859.
De Jong, R., Wierda, M., Mulder, G., Mulder, L.J., 1988. Use of partial stimulus information in response processing. J. Exp. Psychol. Hum. Percept. Perform. 14 (4), 682–692.
Donkin, C., Brown, S., Heathcote, A., 2011. Drawing conclusions from choice response time models: a tutorial using the linear ballistic accumulator. J. Math. Psychol. 55 (2), 140–151.
Elbert, T., 1990. Slow cortical potentials reflect the regulation of cortical excitability. In: McCallum, W.C. (Ed.), Proceedings of a NATO Advanced Research Workshop on Slow Potential Changes in the Human Brain, vol. 65390, pp. 235–251. New York, Plenum Press.
Forstmann, B.U., Dutilh, G., Brown, S., Neumann, J., von Cramon, D.Y., Ridderinkhof, K.R., Wagenmakers, E.-J., 2008. Striatum and pre-SMA facilitate decision-making under time pressure. Proc. Natl. Acad. Sci. 105 (45), 17538–17542.
Forstmann, B.U., Anwander, A., Schäfer, A., Neumann, J., Brown, S., Wagenmakers, E.-J., Turner, R., 2010. Cortico-striatal connections predict control over speed and accuracy in perceptual decision making. Proc. Natl. Acad. Sci. 107 (36), 15916–15920.
Forstmann, B.U., Wagenmakers, E.-J., Eichele, T., Brown, S., Serences, J.T., 2011. Reciprocal relations between cognitive neuroscience and formal cognitive models: opposites attract? Trends Cogn. Sci. 15, 272–279.
Gaillard, A.W., Näätänen, R., 1973. Slow potential changes and choice reaction time as a function of interstimulus interval. Acta Psychol. 37, 173–186.
Grünewald, G., Grünewald-Zuberbier, E., Netz, J., Homberg, V., Sander, G., 1979. Relationships between the late component of the contingent negative variation and the Bereitschaftspotential. Electroencephalogr. Clin. Neurophysiol. 46 (5), 538–545.
Haider, M., Spong, P., Lindsley, D.B., 1964. Attention, vigilance, and cortical evoked-potentials in humans. Science 145 (3628), 180–182.
Hillyard, Steven A., 1969. Relationships between the contingent negative variation (CNV) and reaction time. Physiol. Behav. 4, 351–357.
Hillyard, S.A., Münte, T.F., 1984. Selective attention to color and location: an analysis with event-related brain potentials. Percept. Psychophys. 36 (2), 185–198.
Ho, T., Brown, S., Van Maanen, L., Forstmann, B.U., Wagenmakers, E.-J., Serences, J.T., 2012. The optimality of sensory processing during the speed-accuracy tradeoff. J. Neurosci. 32 (23), 7992–8003.
Ivanoff, J., Branning, P., Marois, R., 2008. fMRI Evidence for a dual process account of the speed-accuracy tradeoff in decision-making. PLoS ONE 3 (7), e2635.
Jung, T.-P., Makeig, S., Humphries, C., Lee, T., 2000. Removing electroencephalographic artifacts by blind source separation. Psychophysiology 37, 163–178.
Kononowicz, T.W., Van Rijn, H., 2011. Slow potentials in time estimation: the role of temporal accumulation and habituation. Front. Integr. Neurosci. 5 (48). http://dx.doi.org/10.3389/fnint.2011.00048.
Kononowicz, T.W., Van Rijn, H., 2014. Decoupling interval timing and climbing neural activity: a dissociation between CNV and N1P2 amplitudes. J. Neurosci. 34 (8), 2931–2939.
Kutas, M., Donchin, E., 1980. Preparation to respond as manifested by motor-related brain potentials. Brain Res. 202, 95–115.
Leuthold, H., Sommer, W., Ulrich, R., 1996. Partial advance information and response preparation: inferences from the lateralized readiness potential. J. Exp. Psychol. Gen. 125 (3), 307–323.
Leuthold, Hartmut, Sommer, W., Ulrich, R., 2004. Preparing for action: inferences from CNV and LRP. J. Psychophysiol. 18, 77–88.
Loftus, G.R., Masson, M.E.J., 1994. Using confidence intervals in within-subject designs. Psychon. Bull. Rev. 1 (4), 476–490.
Loveless, N.E., Sanford, A.J., 1973. The contingent negative variation related to preparatory set in a reaction time situation with variable foreperiod. Electroencephalogr. Clin. Neurophysiol. 35 (4), 369–374.

Loveless, N.E., Sanford, A.J., 1974. Slow potential correlates of preparatory set. Biol. Psychol. 1 (4), 303–314.
Luck, S.J., 2005. An introduction to the event-related potential technique. MIT Press, Cambridge.
MacKay, W.A., Bonnet, M., 1990. CNV, stretch reflex and reaction time correlates of preparation for movement direction and force. Electroencephalogr. Clin. Neurophysiol. 76 (1), 47–62.
McAdam, D.W., Knott, J.R., Rebert, C.S., 1969. Cortical slow potential changes in man related to interstimulus interval and to pre-trial prediction of interstimulus interval. Psychophysiology 5, 349–358.
Miller, J., Patterson, T., Ulrich, R., 1998. Jackknife-based method for measuring LRP onset latency differences. Psychophysiology 35, 99–115.
Mulckhuyse, M., Theeuwes, J., 2010. Unconscious attentional orienting to exogenous cues: a review of the literature. Acta Psychol. 134 (3), 299–309.
Mulder, M.J., Keuken, M.C., Van Maanen, L., Boekel, W., Forstmann, B.U., Wagenmakers, E.-J., 2013. The speed and accuracy of perceptual decisions in a random-tone pitch task. Atten. Percept. Psychophys. 75, 1048–1058.
Müller-Gethmann, H., Ulrich, R., Rinkenauer, G., 2003. Locus of the effect of temporal preparation: evidence from the lateralized readiness potential. Psychophysiology 40, 597–611.
Näätänen, R., 1970. Evoked potential, EEG, and slow potential correlates of selective attention. Acta Psychol. 33, 178–192.
Oostenveld, R., Fries, P., Maris, E., Schoffelen, J., 2011. FieldTrip: open source software for advanced analysis of MEG, EEG, and invasive electrophysiological data. Comput. Intell. Neurosci. 201. http://dx.doi.org/10.1155/2011/156869.
Osman, A., Moore, C.M., Ulrich, R., 1995. Bisecting RT with lateralized readiness potentials: precue effects after LRP onset. Acta Psychol. 90, 111–127.
Palmer, J., Huk, A.C., Shadlen, M.N., 2005. The effect of stimulus strength on the speed and accuracy of a perceptual decision. J. Vis. 5, 376–404.
Pinheiro, C.J., Bates, D.M., 2000. Mixed-Effects Models in S and S-Plus. Springer, New York.
Plichta, M.M., Zangl, M., Meyer-Lindenberg, A., Wolf, I., Hohmann, S., Baumeister, S., Yordanova, J., 2013. Simultaneous EEG and fMRI Reveals a Causally Connected Subcortical-Cortical Network during Reward Anticipation. J. Neurosci. 33 (36), 14526–14533.
Praamstra, P., 2006. Prior information of stimulus location: effects on ERP measures of visual selection and response selection. Brain Res. 1072 (1), 153–160.
Raftery, A., 1996. Approximate Bayes factors and accounting for model uncertainty in generalised linear models. Biometrika 83 (2), 251–266.
Rohrbaugh, J.W., Gaillard, A.W.K., 1983. Sensory and motor aspects of the contingent negative variation. In: Gaillard, A.W.K., Ritter, W. (Eds.), Tutorials in Event-related Potential Research: Endogenous Components. Elsevier, Amsterdam, pp. 269–310.
Schwarz, G., 1978. Estimating the dimension of a model. Ann. Stat. 6 (2), 461–464.
Ulrich, Rolf, Miller, J., 2001. Using the jackknife-based scoring method for measuring LRP onset effects in factorial designs. Psychophysiology 38, 816–827.
Ulrich, R., Leuthold, H., Sommer, W., 1998. Motor programming of response force and movement direction. Psychophysiology 35 (6), 721–728.
Van Boxtel, G.J.M., Böcker, K.B.E., 2004. Cortical measures of anticipation. J. Psychophysiol. 18 (2–3), 61–76.
Van Maanen, L., Brown, S.D., Eichele, T., Wagenmakers, E.-J., Ho, T., Serences, J., Forstmann, B.U., 2011. Neural correlates of trial-to-trial fluctuations in response caution. J. Neurosci. 31 (48), 17488–17495.
Van Rijn, H., Kononowicz, T.W., Meck, W.H., Ng, K.K., Penney, T.B., 2011. Contingent negative variation and its relation to time estimation: a theoretical evaluation. Front. Integr. Neurosci. 5 (91). http://dx.doi.org/10.3389/fnint.2011.00091.
Van Veen, V., Krug, M.K., Carter, C.S., 2008. The neural and computational basis of controlled speed-accuracy tradeoff during task performance. J. Cogn. Neurosci. 20 (11), 1952–1965.
Wagenmakers, E.-J., Grasman, R.P.P.P., Molenaar, P.C.M., 2005. On the relation between the mean and the variance of a diffusion model response time distribution. J. Math. Psychol. 49 (3), 195–204.
Walter, W.G., Cooper, R., Aldridge, V.J., McCallum, W.C., Winter, A.L., 1964. Contingent negative variation: an electric sign of sensorimotor association and expectancy in the human brain. Nature 203 (4943), 380–384.
Wickelgren, W.A., 1977. Speed-accuracy tradeoff and information processing dynamics. Acta Psychol. 41, 67–85.
Winkel, J., Van Maanen, L., Ratcliff, R., Van der Schaaf, M.E., Van Schouwenburg, M.R., Cools, R., Forstmann, B.U., 2012. Bromocriptine does not alter speed-accuracy tradeoff. Front. Neurosci. 6 (126). http://dx.doi.org/10.3389/fnins.2012.00126.
Wood, S.N., 2006. Generalized Additive Models: An Introduction with R. Chapman & Hall, London.
Wood, S., 2013. Mixed GAM computation vehicle with GCV/AIC/REML smoothness estimation. R package Version 1.7-26.
Wühr, P., Kunde, W., 2008. Precueing spatial S–R correspondence: is there regulation of expected response conflict? J. Exp. Psychol. Hum. Percept. Perform. 34 (4), 872–883.