The NoGo-Anteriorization and its relation to a central inhibitory mechanism

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Contents

Introduction

> Stopping is the first step in reorienting to new goals and new worlds, a general requirement in all kinds of cognitive control.
> Gordon D. Logan, 1994

As the statement of Logan implies interrupting a planned or initiated action is an everyday effort. Without this ability we would inevitably perform every action once we planned it. Surrounded by an environment that can immediately change, the inability to stop would result in many problems. For instance, we would cross the street although we see a car speeding towards us. A behavior one would have to explain to a psychiatrist if one is so lucky as to survive. Moreover, our social life would be very difficult without the ability to stop actions. For example, we could not stop making fun of our boss although we have seen him coming around the corner. This will not increase one's chances of being promoted. On the side of thought the inability to stop thinking of one thing in favor of another would cause perseverative thinking without creativity.

However, it is difficult to discover how we stop actions, since the underlying processes of inhibition are not directly observable. In an attempt to solve this problem, Logan and Cowan (1984) investigated how long people need to stop an action in the so called stop-signal paradigm. In this kind of tasks, the participants are instructed to press a key when a signal (Go signal) appears. Sometimes the Go signal is followed by another signal (the stop-signal), which requires the participants to withhold their response. Presumed that the Stop signal precedes the Go signal very soon participants successfully inhibit their ongoing processing. However, the later the Stop signal appears, the harder is the response inhibition. The proportion of successfully stopped responses provides a basis for estimating the duration of inhibition. Based on findings with the stop-signal paradigm Logan and Cowan developed their Horse Race Model. This model assumes a response process and an inhibitory process racing against each other. The process, which finishes first, determines performance. Logan and Cowan envisaged a single, central inhibitory mechanism, whereas De Jong, Coles, Logan, and Gratton (1990) assumed the existence of an additional peripheral inhibitory mechanism. However, the existence of a second peripheral inhibitory mechanism is not undisputed (Band & van Boxtel, 1999; van Boxtel, van der Molen, Jennings, & Brunia, 2001). Chapter 1 reports findings in the stop-signal paradigm, describes the Horse Race Model, discusses assumed inhibitory mechanisms, and gives an overview of brain regions, which are assumed to be the sources of inhibitory mechanisms.

The central inhibitory mechanism is associated with two event-related brain potentials (ERPs), the N200 and the NoGo-P300. The N200 is a negative deflection, which occurs 200-300 ms after the presentation of a stimulus (e.g., Kok, 1986). The amplitude of the N200 is decreased in trials in which a response is performed compared to that in trials in which a response is inhibited. The NoGo-P300 is a positive deflection, occurring 300-600 ms after the presentation of a stimulus, in trials in which a response is inhibited (e.g., Pfefferbaum, Ford, Weller & Koppell, 1985). The NoGo-P300's maximum of the brain electrical field is located at the Cz-electrode, whereas the maximum of the P300 in trials in which a response is performed (Go-P300) is located at the Pz-electrode. Fallgatter, Brandeis, and Strik (1997) labeled this difference in topography NoGo-Anteriorization (NGA) because the NoGo-P300's maximum is located more anterior than that of the Go-P300. Chapter 2 reviews literature about N200 and NGA. Furthermore, support and objections concerning their connection to inhibitory mechanisms are discussed.

While the N200 phenomenon has recently been explained by conflict monitoring and / or representing mental effort (e.g., Botvinick, Cohen, & Carter, 2004), there is still an unresolved discussion about the NGA's cause. The empirical part reports four Experiments that rule out objections against the view that NGA reflects an inhibitory mechanism.

Experiment 1 and Experiment 2 apply the method of subliminal priming to investigate whether negative motor potentials during Go trials cause the NGA by erasing the anterior part of the Go-P300. The results rule out this possibility and show that the NGA is related to a mechanism triggered by NoGo stimuli.

Experiment 3 compares P300s of Go and NoGo trials that contain the same kind of conflict. It can be shown that the NGA is not the result of conflict on a response level during NoGo trials.

Experiment 4 demonstrates that the NGA does not represent a greater influence of processes like stimulus identification on the NoGo-P300 topography than on the Go-P300 topography due to different cognitive requirements in terms of response selection in NoGo trials compared to Go trials. This is accomplished by comparing P300s of Go and NoGo trials that do not require the identification of a stimulus. In contrast, the frequencies' analyses of the Go- and NoGo-ERPs reveal that there is still higher activity over the Cz-electrode in the P300 frequency-range (0.5-3.5 Hz) in NoGo trials than in Go trials.

Therefore, neither motor potentials during Go trials, nor response conflict, nor decreased cognitive requirements in terms of response selection in NoGo trials are tenable alternatives to the assumption of an inhibitory mechanism being related to the NGA. The work closes with a general discussion that summarizes the results of all Experiments.

1 The investigation of inhibition

Since inhibition is not directly observable, researchers investigated tasks that were assumed to require inhibition to learn more about inhibitory control. One kind of these tasks is summarized with the term stop-signal paradigm. Logan and Cowan (1984) tried to explain the main findings of the stop-signal paradigm with their Horse Race Model. In the following, first the main findings of the stop signal paradigm and then the explanations of the findings by the Horse Race Model are described. Finally, the underlying assumptions of the model are discussed.

1.1 Findings of the stop-signal paradigm

In the stop signal paradigm, participants perform a response in accordance to a signal (Go signal). On some occasions, a stop signal (NoGo signal[1]) follows the Go signal after a variable interval. This interval between Go and NoGo signal is also termed stimulus onset asynchrony (SOA), due to the variable onset of the NoGo signal (Band & van Boxtel, 1999). The NoGo signal requires the participants not to respond in that trial. Within the stop signal paradigm, two kinds of reaction times (RTs) can be distinguished. RTs when there is no NoGo signal are called no-signal RTs and RTs that escaped inhibition are signal-respond RTs.

There are four main findings of the stop signal paradigm which should be explained by the Horse Race Model. First, the probability of responding increases with an increasing SOA. In other words, the longer the interval between Go and NoGo signal, the more responses will be performed. Secondly, participants with faster RTs in trials without NoGo signal (faster no-signal RTs) respond with a

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[1] Normally, the term NoGo signal is used in Go-NoGo tasks in which the occurrence of a Go stimulus instructs the participant to respond whereas the occurrence of a NoGo stimulus tells the participants not to respond (e.g., Fallgatter, 2001). Because van Boxtel, van der Molen, Jennings, and Brunia (2001) could show that the same kind of inhibition is active in stop signal trials as in NoGo trials, I use the terms stop signal and NoGo signal synonymously. Furthermore, according to Band and van Boxtel (1999), the stop signal paradigm is an advanced version of Go-NoGo tasks, since in the Go-NoGo tasks the SOA is always zero.

greater probability at a given SOA compared to those with slower no-signal RTs. Thirdly, the mean of RTs that escaped inhibition (signal-respond RTs) is faster than the mean of the no-signal RTs. Fourthly, signal-respond RTs increase with an increasing SOA.

1.2 The Horse Race Model

The Horse Race Model tries to explain these four major findings of the stop signal paradigm with the assumption of a “horse race” between a response process and an inhibitory process. The process that wins the race, i.e. is completed first, determines the performance. If the response process wins, the response is executed; if the inhibitory process wins, the response is inhibited. The ideas of the model are presented in Figure 1.

The model assumes that a Go signal triggers a probabilistic response process (see Figure 1, A). This response process includes the processing of the response stimulus, the response choice, motor preparation, and the execution of the response (e.g., van Boxtel, van der Molen, Jennings, & Brunia, 2001). Therefore, when the process is finished the response is executed. The possible responses after a Go signal result in the RTs-distribution.

In trials with a NoGo signal, the NoGo signal triggers an inhibitory process (see Figure 1, B). This inhibitory process encompasses the processing of the NoGo stimulus and the response inhibition (van Boxtel et al., 2001). The finishing time of this inhibitory process dissects the RTs-distribution. Responses with RTs that are faster than the finishing of the inhibitory process are executed. Responses that are slower than the finishing of the inhibitory process are inhibited.

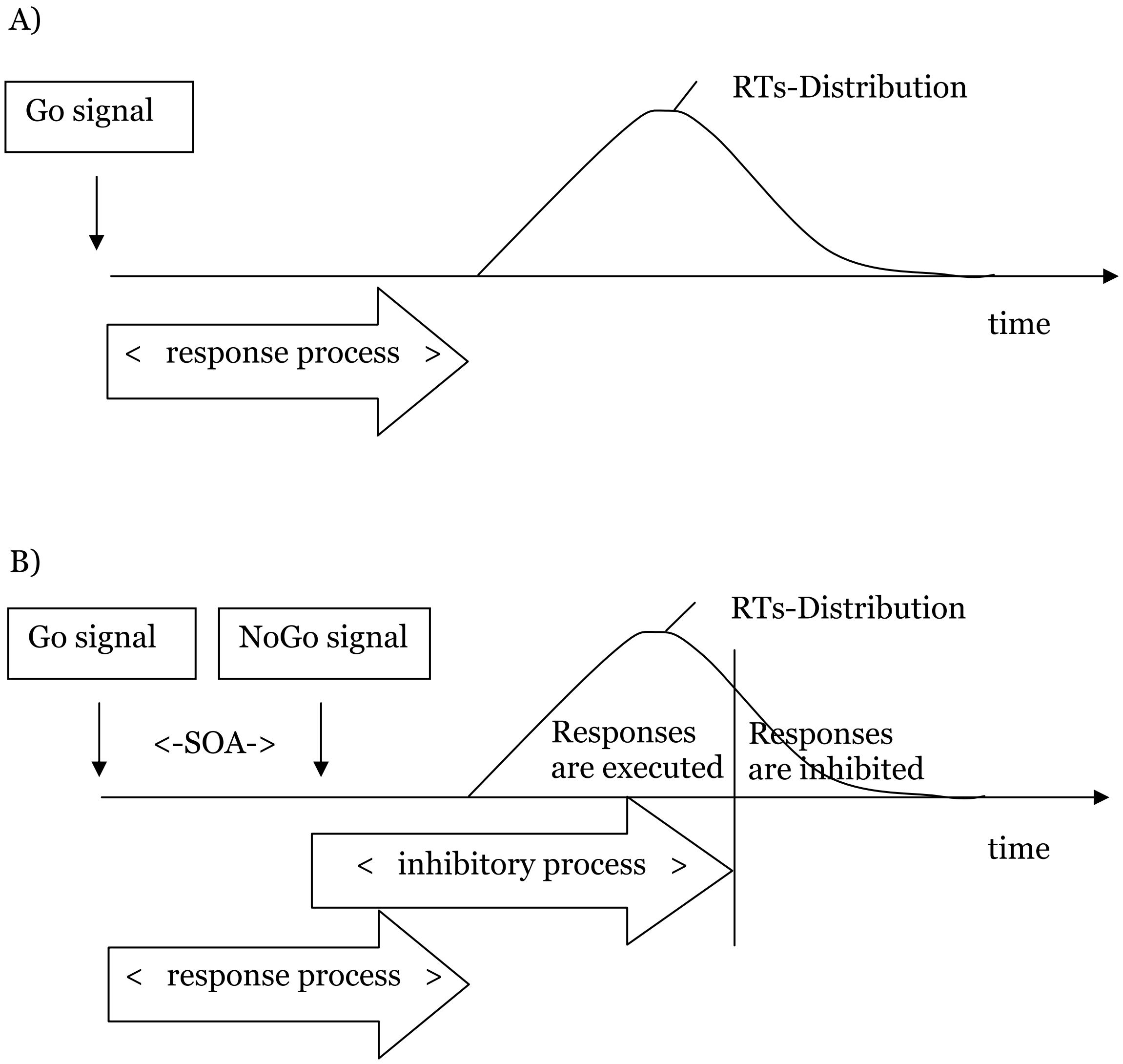

Figure 1. Illustration of the Horse-Race-Model. A: RTs-Distribution in trials without NoGo signal (no-signal trial). B: RTs-distribution in trials with NoGo signal.

The SOA in the stop signal paradigm influences the ratio of executed to inhibited responses. With longer SOAs, the probability of loosing the race increase for the inhibitory process. Therefore, the probability of responding in trials with a long SOA is greater than in those with a short SOA, which is the first finding of the stop signal paradigm. But the probability of responding is not solely dependent on the SOA. The duration of response and inhibitory process is also important for the observable performance. If a participant's duration of the response process is very short, the probability for his or her response process to win the race is increased, assuming that the participants' inhibitory process is not also very fast. In contrast, if the participant's response process is very slow, the probability of winning the race is increased for the inhibitory process. Therefore, the model can also explain the second finding of the stop signal paradigm, namely that the probability of responding in trials with a NoGo signal after a given SOA is increased for participants with faster no-signal RTs. Thirdly, the model can explain why the signal-respond RTs are faster than the no-signal RTs. The inhibitory process, which is triggered by the NoGo signal, wins in case of very slow response processes. This means that the inhibitory process truncates the upper tail of the RTs-distribution. Contrary, the distribution of the no-signal RTs represents the whole range of response processes. Thus, the mean reaction time of the signal-respond RTs-distribution is faster than that of the no-signal RTs-distribution. Finally, with an increasing SOA both distributions will become more and more similar, because longer SOAs permit slower response processes to win the race against the inhibitory process. Therefore, the mean signal-respond RTs will increase with an increasing SOA until there is no difference between the mean of the signal-respond RTs-distribution and the no-signal RTs-distribution.

1.3 Evaluation of the Horse Race Model

The main advantage of the Horse Race Model is that it allows estimating the duration of the inhibitory process by the so called stop signal reaction time (SSRT). Logan and Cowan (1984) proposed two methods of calculating the SSRT (for a description of both methods, see Logan, 1994; Band et al., 2003). The first method implies that the SSRT is constant. Logan and Cowan regarded this assumption as probably wrong. But they could show that the measurement errors according to this false assumption were very small. A more serious problem for the first method is its small reliability (see Logan, Schachar, & Tannock, 1997). Therefore, the second method is widely in use today.

One major assumption of the original model is that the response and inhibitory processes are independent in two respects. For one, the occurrence of the NoGo signal should not affect the distribution of RTs. That means that the RTs-distribution of trials without NoGo signal has the same shape as the distribution of the trials with a NoGo signal. De Jong, Coles, and Logan (1995) investigated this kind of signal independency in a task where participants should selectively inhibit responses of one hand when a NoGo signal occurred. When the task required the response with the other hand the NoGo signal was meaningless. In contrast to the assumptions of the Horse Race Model, the occurrence of a NoGo signal slowed down RTs even in trials where the NoGo signal had no meaning for the performance of the trial.

Secondly, the Horse Race Model assumes that response and inhibitory processes are stochastically independent variables. This means that the finishing times of the processes are uncorrelated. Logan and Cowan (1984) tried to rule out alternatives by contrasting them with data from stop signal paradigms. The main argument against the idea that response and inhibitory process could share limited resources grounds on the fact that no-signal RTs as well as stop-signal RTs are similar to RTs of simple Go tasks. Any resource sharing should lengthen the processes.

Logan and Cowan (1984) also rejected that participants would use strategies that determine when to respond and when not to respond, irrespective of which stimulus is presented. If that was the case, the probability of responding in a trial with a NoGo signal should not vary with the SOA.

However, the observed decrease in SSRTs with an increasing SOA (Logan & Cowan, 1984) seemed to compromise the assumption of independency between response and inhibitory processes. De Jong, Coles, Logan, and Gratton (1990) showed with Monte Carlo simulations that this result would be expected if response and inhibitory process were correlated.

To save the independency assumption, Logan and Burkell (1986) picked up an assumption of Logan and Cowan (1984). If there was variability in SSRTs, the decrease in SSRTs with an increasing SOA could be explained. In analogy to the RTs-distribution, the distribution of SSRTs would be cut off on the right side by the termination of the response process at longer SOAs. Therefore, only short SSRTs would be seen at long SOAs.

Another main assumption of Logan and Cowan's model (1984) is the idea that the inhibitory process is an executive-control process. This kind of process is believed to form intentions and transform these intentions into commands for a subordinate system. However, as Band and van Boxtel (1999) argue, such models are problematic, because they try to explain control by creating a structure that controls. This only shifts the problem that should be explained towards another level, which is also known as the homunculus problem (see for example Hommel, Daum, & Kluwe, 2004).

1.4 Mechanisms of inhibition

Originally, Logan and Cowan (1984) envisaged a single, central inhibitory mechanism being responsible for stopping actions. Later De Jong, Coles, Logan, and Gratton (1990) assumed the existence of an additional peripheral inhibitory mechanism. However, several researchers questioned the existence of this second inhibitory mechanism (Band & van Boxtel, 1999; Van Boxtel, van der Molen, Jennings, & Brunia, 2001). In the following, first the central inhibitory mechanism will be described. Then the findings that could indicate an additional peripheral inhibitory mechanism will be introduced. Finally, the objections to the existence of a peripheral inhibitory mechanism will be presented.

1.4.1 A central inhibitory mechanism

The small variability of stop-signal reaction-times (SSRTs) in different stopping tasks supported the assumption of a single inhibitory mechanism (for a review see Logan & Cowan, 1984). Although Logan and Cowan did not specify the neuroanatomical locus of this inhibitory mechanism, they assumed that the inhibitory mechanism controls the motor system. Therefore, the locus of inhibition has to be “upstream” from the primary motor cortex. In accordance with this assumed cortical locus for the inhibitory mechanism, De Jong, Coles, Logan, and Gratton (1990) called this mechanism a central inhibitory mechanism in contrast to a peripheral, non-cortical one.

An event-related brain potential, the so called NoGo-P300 was suggested to be related to the central inhibitory process (De Jong et al., 1990). The NoGo-P300 is a positive wave that occurs 300-600 ms after the presentation of a NoGo signal (e.g., Pfefferbaum, Ford, Weller, & Koppell, 1985). Compared to the Go-P300, which occurs after a signal that tells participants to respond, the NoGo-P300 has a maximum of the brain electrical field which is located more anteriorly. Fallgatter, Brandeis, and Strik (1997) called the difference in topography between Go-and NoGo-P300 NoGo-Anteriorization (NGA; see also section 2.2.1). Like De Jong et al., Fallgatter et al. interpreted the NGA as a sign for the occurrence of the central inhibitory process during stop conditions (see also Fallgatter, 2000; Fallgatter and Strik, 1999; Strik, Fallgatter, Brandeis, & Pascual-Marqui, 1998).

Inspired by the vector-integration-to-endpoint model of Bullock and Grossberg (1988, 1991), the central inhibitory mechanism was thought to modify movement commands to the muscles (De Jong, Coles, & Logan, 1995). Bullock and Grossberg's model is presented in Figure 2.

In their model, movement commands to the muscles (D) are generated by a present position command (C) which is updated by integrating the difference between an effector's actual (C) and its target position (A) at any time. Bullock and Grossberg describe the differences between an effector's actual and its target position as a difference vector (B). Before the difference vector (B) is integrated, it is energized by multiplication with a so called Go signal (E) resulting from a peripheral process. The updated present position command (C) in turn updates the difference vector (B) through negative feedback. A change of the effector's target position (A) that results in changing the difference vector (B) would easily allow the replacement of a once initiated response by another one (D) (De Jong et al., 1995). Therefore, the central inhibitory mechanism should affect the change of the effector's target position (A). Advantageously, with the central inhibitory mechanism there would be no need to restart the peripheral process (E) allowing a smooth replacement of one movement with another (De Jong et al.).

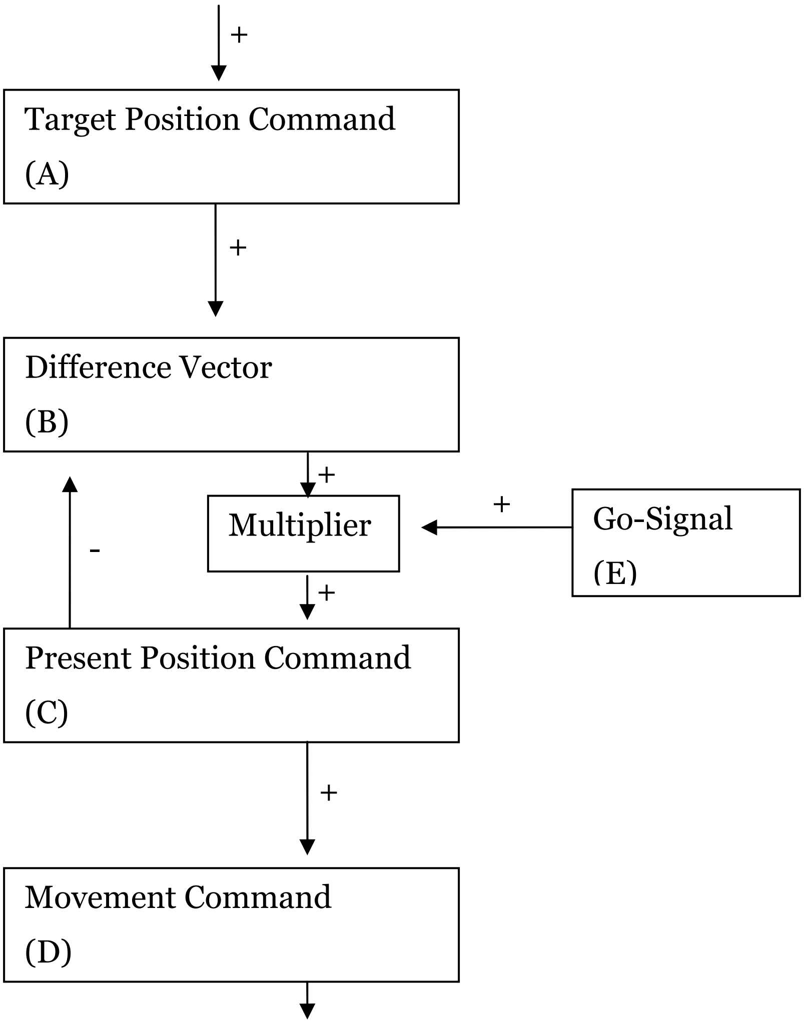

Figure 2. Illustration of the vector-integration-to-endpoint model (Bullock and Grossberg, 1988, 1991).

1.4.2 A peripheral inhibitory mechanism

Additionally to the central inhibitory mechanism, De Jong, Coles, Logan, and Gratton (1990) as well as De Jong, Coles, and Logan (1995) proposed a faster operating peripheral inhibitory mechanism. In accordance with Bullock and Grossberg's model (1988, 1991), which is depicted in Figure 2, the peripheral inhibitory mechanism should non-selectively stop the energizing of motor commands by the Go-signal (E) instead of changing the target position command (A). Because the Go-signal non-specifically energizes every difference vector of the system, inhibiting the Go-signal would result in inhibiting all movements at once. Therefore, the peripheral inhibitory mechanism should be involved in trials in which a once initiated response need to be stopped immediately. In contrast, the central inhibitory mechanism should inhibit responses in so called stop-change trials. In these trials a once initiated response has to be selectively inhibited and be replaced with an alternative response. For instance, in stop-change trials of De Jong, Coles, and Logan, a NoGo signal instructed participants to perform a response with their feet instead of a manual response. Therefore, NoGo signals in stop-change trials do not require withholding any response but they require performing another response instead.

The assumption of a peripheral inhibitory mechanism was based on three findings. First of all, the onset of the NoGo-P300 was too late to cause inhibition in stop-signal trials. De Jong et al. (1990) found the onset of the NoGo-P300 in stop-trials after the inhibitory process had already finished. In their study the duration of the inhibitory process was estimated by calculating the SSRT with respect to the onset of muscle activity measured by an electromyogram (EMG). If the NoGo-P300 represented the central inhibitory process, another, faster inhibitory mechanism must have been responsible for the inhibition in stop-signal trials.

Secondly, De Jong et al. (1990) found an interruption of a response within 60 ms after the onset of muscle activity. If the central inhibitory mechanism was responsible for the interruption, the mechanism would have to precede the inhibitory effects on the muscles by at least the time required for motor commands to become effective on muscle activity. However, De Jong et al. (1995) calculated that transmission of central motor commands to the peripheral motor system would take 80 ms.

Thirdly, under the fixed criterion hypothesis (Gratton, Coles, Sirevaag, Eriksen, & Donchin, 1988) a response is executed if a certain threshold of motor activity is reached. The threshold assumption is based on the observation that in no-signal trials the amplitude of the lateralized readiness potential (LRP)[2] is constant at the onset of EMG activity. Therefore, De Jong et al. (1990) used the LRP-amplitude at EMG-onset in trials without NoGo signal (no-signal trials) as threshold for executing a response. If the central inhibitory mechanism was the only inhibitory mechanism, the mean LRP-amplitudes of inhibited responses

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[2] The LRP reflects relative differences in activation levels of left and right motor cortices before the performance of a unimanual response. Therfore, it is thought to indicate response preparation processes (Coles, 1989).

should always be below the threshold. Empirically, the LRPs of successfully inhibited responses in stop signal trials with middle and long intervals between Go and NoGo signal (SOAs) reached the threshold, which normally leads to the execution of a response. De Jong et al. (1995) replicated this finding and could show that in stop-change trials, in which the central inhibitory mechanism should operate, the LRP was indeed smaller than that of no-signal trials.

The assumption of a peripheral inhibitory mechanism was further supported by observations concerning the heart rate. Jennings, van der Molen, Brock, and Somsen (1992) found that the heart rate decelerated in trials in which participants inhibited a response. This was true only if the inhibitory process operate before or just at the period of the cardiac pacemaker's maximal sensitivity for signals from the vagus. Jennings et al. therefore suggested a midbrain initiation of a peripheral inhibitory mechanism that was also responsible for the heart-rate deceleration during response inhibition.

1.4.3 Criticism of a peripheral inhibitory mechanism

However, the existence of a peripheral inhibitory process was also questioned (Band & van Boxtel, 1999; van Boxtel, van der Molen, Jennings, & Brunia, 2001). Band and van Boxtel interpreted the three findings that led De Jong, Coles, Logan, and Gratton (1990) to the assumption of a peripheral inhibitory mechanism in a way that redundantized the existence of a peripheral inhibitory mechanism. First, Band and van Boxtel replaced the NoGo-P300 as a sign for the central inhibitory mechanism with the N200. The N200 is a negative deflection in the EEG[3] that starts more than 100 ms earlier than the NoGo-P300. However, an earlier onset of the central inhibitory mechanism makes the assumption of a fast peripheral inhibitory mechanism superfluous. The connection between inhibition and the N200 was formulated because the amplitude of the N200 after a stop-signal is increased compared to that after a Go-signal (e.g., Kok, 1986). Furthermore, the N200's maximum of the electrical field is located over the frontal cortex which seems to play a central role for inhibition (see section 1.5). The connection between

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[3] The EEG (electroencephalogram) records voltage fluctuations at the surface of the head, which result from neuronal assemblies in the brain (Rösler, 1982).

inhibition and the N200 was formulated because the amplitude of the N200 after a stop-signal is increased compared to that after a Go-signal (e.g., Kok, 1986).

Secondly, the interruption of a response within 60 ms after the onset of muscle activity could also be explained by fading of activation from the motor cortex for muscle activity after some initial EMG-activity has already occurred. In that case, no additional inhibition would be necessary.

Thirdly, Band and van Boxtel (1999) criticized the De Jong et al.'s (1990) method of assessing the threshold for responding. De Jong et al. measured the LRP with regard to stimulus onset. Unfortunately, the resulting stimulus-locked LRP contains aside of response related activations also stimulus related contributions. Therefore, it is unclear to what extent this stimulus-locked LRP reflects a threshold for responding. Especially, since response related activation is not adequately represented by the mean stimulus-locked LRP. Averaging LRPs with different onsets of response related activation due to variability in the timing of responses results in an underestimate of the response related activation. Therefore, the more appropriate measure to determine the threshold for responding would be the amplitude of the mean response-locked LRP. This methodological objection from Band and van Boxtel was supported by Mordkoff and Gianaros (2000). The latter could show that an effect on the preparation of responses, for instance an effect by inhibitory mechanisms, would not affect the stimulus-locked LRP. However, De Jong et al. tried to prove the existence of a peripheral inhibitory mechanism with findings based on stimulus-locked LRPs.

Band and van Boxtel (1999) also explained the heart-rate deceleration cortically. According to them, there is no anatomical evidence for an initiation of inhibition by the midbrain. In contrast, Band and van Boxtel assumed input from the frontal cortex to subcortical cardiovascular centers. This assumption was based on findings from Neafsey (1990) who showed that stimulation of the anterior cingulate cortex leads to heart-rate-deceleration.

Beyond these more theoretical criticisms, van Boxtel, van der Molen, Jennings, and Brunia (2001) investigated the relationship between LRP, N200, and heart rate deceleration, empirically. In their opinion, the same LRP-duration in successful stop-trials and no-signal trials would indicate the existence of a peripheral inhibitory mechanism. According to them, exceeding the threshold amplitude only marks the time when central motor outflow to the periphery has started. In contrast, the duration of the LRP mirrors how long the muscles receive the motor outflow. In fact, van Boxtel et al. found a shorter LRP-duration in successful stop-trials than in no-signal trials. Furthermore, they showed that the LRP starts to decrease when the N200 starts to rise. There was also a connection between heart-rate deceleration and N200 amplitude. The N200 amplitude was highest in stop trials when the interval between NoGo stimulus and N200 onset was before or just at the period of the cardiac pacemaker's maximal sensitivity for signals from the vagus. In other words, N200 amplitude was maximal when inhibitory effects on the heart beat were also maximal. Therefore, van Boxtel et al. assumed that only one central inhibitory mechanism exists, which is represented by the N200.

1.5 The Locus of inhibition

Asking for the locus of inhibition is not as unambiguous as it may seem. For instance, Band and van Boxtel (1999, pp. 190-192) distinguished three types of loci: Brain regions, which are the source of the inhibitory mechanism (agent), processes, which are inhibited by the inhibitory mechanism (site), and the observable results of the inhibitory mechanism, namely the absence or attenuation of response processes (manifestation). However, when talking about the locus of inhibition mostly the agent is of special interest. Therefore, in the following only findings concerning the agent of inhibition will be reported.

Table 1 shows brain regions that were activated during tasks that required inhibition[4]. The brain regions (first column) are sorted according to the number of studies naming each region to be related to the inhibitory mechanism. The second column of the table contains the Brodmann areas (BAs) named in the studies. In the third column the letter X indicates whether studies found a particular region predominantly activated in the left hemisphere, bilateral, or in the right

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[4] All but one of the listed studies in Table 1 used functional magnetic resonance imaging to investigate brain activity during inhibition (for technical details concerning the method of fMRI see Parry & Matthews, 2002). Aron, Fletcher, Bullmore, Sahakian, and Robbins (2003) used magnetic resonance imaging to deteremine the locus of patients' lesions.

hemisphere. The last column lists the studies that investigated brain activation during tasks that required inhibition.

As can be seen in Table 1, most researchers found that the source of the central inhibitory mechanism is located in the inferior frontal cortex. For instance, Aron, Fletcher, Bullmore, Sahakian, and Robbins (2003) measured the stop signal reaction times (SSRTs) of patients with different lesions in the right frontal lobe. Afterwards, they correlated the SSRTs with the size of the lesions. This correlation was significantly larger in the inferior frontal cortex than in any other region. Furthermore, multiple regression showed that only the size of lesions in a sub-area of the inferior frontal cortex (pars triangularis, BA 45) explained variability in SSRTs.

However, there are fewer consensuses concerning the involvement of other brain regions in the inhibitory mechanism. For instance, different studies named the following brain regions as being linked to inhibition: The middle frontal cortex, the anterior cingulate cortex, the inferior parietal cortex, the superior frontal cortex, the insula, the precentral cortex, the Pre-SMA, the middle temporal cortex, and the thalamus (see also Table 1). Some of the observed differences may result from the tasks which were used in the different studies. For instance, participants in the study of Konishi, Nakajima, Uchida, Sekihara, and Miyashita (1998) performed a Go-NoGo task with equiprobable Go and NoGo trials. The participant’s task was to press a key when a green square was presented and to withhold a key press when a red square appeared. The intertrial interval was 32-40 s (!). The requirements concerning working memory were at best small compared to the requirements to stay awake. Contrary, the participants in the Go-NoGo task of Garavan, Ross, and Stein (1999) saw a stream of letters, consisting of Xs and Ys, which were presented serially every 500 ms. Participants had to press a key each time the presented letter changed from X to Y or vice versa resulting in a higher working memory load than in the Go-NoGo task of Konishi et al. Accordingly, Konishi et al. found only the pars triangularis (BA 45) to be more activated during NoGo trials, whereas Garavan et al. found regions within the inferior frontal cortex (BA 10), the middle frontal cortex (BA 9), the anterior cingulate cortex (BA 32), the inferior parietal cortex (BA 40), the insula (BA 13), the middle occipital gyrus (BA 19), the superior occipital gyrus (BA 19), the angular gyrus (BA 39), and the fusiform gyrus (BA 37) to be more activated during NoGo trials compared to Go trials[5].

Additionally, the studies vary concerning the applied methods. For instance, in the study of Watanabe et al. (2001) the brain activity after the presentation of single NoGo trials was compared to that after single Go trials. In the study of Menon, Adleman, White, Glover, and Reiss (2001) the brain activity in blocks of Go and NoGo trials were compared to that in pure blocks of Go trials. However, the brain activity in the Go trials during the Go/NoGo blocks could be different from those in Go trials in pure Go blocks due to switching between Go and NoGo trials within the Go/NoGo blocks. These differences might be misinterpreted as inhibitory activity. This could be the reason for Watanabe et al. reporting more brain activity during NoGo trials in the middle frontal cortex (BA 10), in the posterior intraparietal cortex (BA 7), and in the occitotemporal cortex (BA 19), whereas Menon et al. reported more brain activity during NoGo trials in the dorsolateral prefrontal cortex (BA 9/46), in the inferior frontal cortex (BA 45/47), in the Pre-SMA (BA 6), in the anterior cingulate cortex (BA 24), in the lingal gyrus (BA 37), in the inferior parietal lobe (BA 39), and in the caudate.

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[5] Note, only regions that were reported by more than one study were listed in Table 1.

Table 1. Regions found in fMRI-studies during tasks that require inhibition of an action.

Regions associated with inhibition	Brodman Area	Hemispheric dominance			Studies
		left	bilateral	right	
1) Inferior frontal cortex	9			X	Bellgrove, Hester, and Garavan (2004)
	10			X	Garavan, Ross, and Stein (1999)
	9/44			X	Durston, Thomas, Worden, Yang, and Casey (2002)
Pars opercularis	44			X	Aron, Fletcher, Bullmore, Sahakian, and Robbins (2003); Garavan, Ross, Murphy, Roche, and Stein (2002)
	44/45		X		Rubia et al. (2001)
	44/46	X			Durston, Thomas, Worden, Yang, and Casey (2002)
Pars triangularis	45			X	Aron, Fletcher, Bullmore, Sahakian, and Robbins (2003); Konishi, Nakajima, Uchida, Kikyo, Kameyama, and Miyashita (1999); Konishi, Nakajima, Uchida, Sekihara, and Miyashita (1998)
	45/47		X		Menon, Adleman, White, Glover, and Reiss (2001)
2) Middle frontal cortex	6	X			Bellgrove, Hester, and Garavan (2004)
	6/8	X			Durston, Thomas, Worden, Yang, and Casey (2002)
	9/6			X	Rubia et al. (2001)
	9		X		Bellgrove, Hester, and Garavan (2004)
	9			X	Garavan, Ross, Murphy, Roche, and Stein (2002); Garavan, Ross, and Stein (1999)
	9/46		X		Menon, Adleman, White, Glover, and Reiss (2001)

Table 1 continued.

Regions associated with inhibition	Brodman Area	Hemispheric dominance			Studies
		left	bilateral	right	
2) Middle frontal cortex (continued)	10		X		Watanabe et al. (2002)
	46			X	Bellgrove, Hester, and Garavan (2004); Mostofsky et al. (2003)
3) Anterior cingulate cortex	24			X	Menon, Adleman, White, Glover, and Reiss (2001)
	24/6			X	Garavan, Ross, Murphy, Roche, and Stein (2002)
	32/24	X			Bellgrove, Hester, and Garavan (2004)
	8/32			X	Rubia et al. (2001)
	32			X	Durston, Thomas, Worden, Yang, and Casey (2002)
	32		X		Garavan, Ross, Murphy, Roche, and Stein (2002); Garavan, Ross, and Stein (1999)
4) Inferior parietal cortex	39/7			X	Garavan, Ross, Murphy, Roche, and Stein (2002)
	39		X		Menon, Adleman, White, Glover, and Reiss (2001)
	40			X	Garavan, Ross, Murphy, Roche, and Stein (2002); Rubia et al. (2001)
	40		X		Bellgrove, Hester, and Garavan (2004); Durston, Thomas, Worden, Yang, and Casey (2002); Garavan, Ross, and Stein (1999)

Table 1 continued.

Regions associated with inhibition	Brodman Area	Hemispheric dominance			Studies
		left	bilateral	right	
5) Insula	13			X	Durston, Thomas, Worden, Yang, and Casey (2002); Garavan, Ross, and Stein (1999)
	13		X		Bellgrove, Hester, and Garavan (2004); Garavan, Ross, Murphy, Roche, and Stein (2002)
6) Pre-SMA	6			X	Rubia et al. (2001)
	6	X			Bellgrove, Hester, and Garavan (2004)
	6		X		Menon, Adleman, White, Glover, and Reiss (2001)
7) Precentral cortex	6			X	Garavan, Ross, Murphy, Roche, and Stein (2002)
	6	X			Bellgrove, Hester, and Garavan (2004)
	44/6	X			Bellgrove, Hester, and Garavan (2004)
8) Superior frontal cortex	6		X		Mostofsky et al. (2003)
	6			X	Durston, Thomas, Worden, Yang, and Casey (2002)
	8	X			Durston, Thomas, Worden, Yang, and Casey (2002)
9) Middle temporal cortex	21		X		Rubia et al. (2001)
	21			X	Garavan, Ross, Murphy, Roche, and Stein (2002)
10) Thalamus			X		Garavan, Ross, Murphy, Roche, and Stein (2002)
				X	Bellgrove, Hester, and Garavan (2004)

Note. Regions are listed only when they were found in more than one study.

2 Event-related potentials as correlates of inhibition

2.1 N200 and inhibition

Several researchers found a more pronounced N200-amplitude at the Fz- and Cz-electrodes after a signal requiring the inhibition of a response than after a stimulus requiring the execution of a response (e.g., Kok, 1986). It seemed most intuitive to assume a connection between the N200-amplitude and the central inhibitory mechanism (for a review, see Band & van Boxtel, 1999). The first section reviews findings, which support this point of view. Recently, researchers linked the N200-amplitude after a NoGo-signal to the monitoring of conflict and / or representation of mental effort. Therefore, the second section will report findings that question a relationship between N200 and a central inhibitory mechanism.

2.1.1 N200 as representation of a central inhibitory mechanism

Eimer (1993) investigated the influence of response priming on the N200-amplitude. In his study participants had to perform a Go-NoGo task. In a Go-NoGo task, one stimulus instructs the participants to perform a response (Go target) whereas another stimulus instructs the participants to withhold a response (NoGo target). In Eimer’s study Go- and NoGo targets were uppercase letters (either M or W) occurring equiprobable on the left or on the right side of the monitor. Before Go and NoGo targets an arrow was presented, which pointed in 75% of the trials to the location where the target symbol would subsequently appear (valid cuing). In the remaining trials the arrow pointed to the opposite side (invalid cuing). When the Go target appeared on the left side participants had to respond with their left hand; when the Go target occurred on the right side they had to respond with their right hand. Therefore, in Go trials the valid cue primed the correct response side.

Eimer assumed that the valid cuing in NoGo trials would require more inhibition than the invalid cuing. If the N200-amplitude represented the central inhibitory mechanism, the N200 amplitude after validly cued NoGo trials should be enhanced compared to that after invalidly cued NoGo trials. In fact, this was the case.

Moreover, Eimer (1993) investigated, whether the difference in N200-amplitude between Go and NoGo trials was based on the different stimulus probability of Go and NoGo trials. Therefore, he compared N200-amplitudes of Go and NoGo trials for two kinds of Go probabilities (0.75 and 0.50). Even though the difference in the N200-amplitude between Go and NoGo trials decreased at the Cz-electrode, when Go and NoGo trials were equiprobable, there was still a greater N200-amplitude in NoGo trials than in Go trials at the Fz-electrode. Therefore, Eimer concluded that mostly the anteriorly located part of the N200 represents the inhibitory mechanism.

Kopp, Mattler, Goertz, and Rist (1996) strengthened Eimer's position (1993) that the N200 is linked to the inhibitory mechanism by investigating the N200 in a flanker task (Eriksen and Eriksen, 1974). In their paradigm Go targets were arrowheads pointing to the left or right side whereas NoGo targets were octagons. The Go targets were surrounded by stimuli (flankers) that either pointed in the same direction as the Go target (congruent flankers), pointed in no direction (neutral flankers = squares), or pointed in the opposite direction as the target (incongruent flankers). The Go probability was 0.67. NoGo targets were surrounded by right-pointing flankers, left-pointing flankers, or neutral flankers. The idea was that left- and right-pointing flankers would be associated with a specific response with one hand whereas the neutral flankers would be unspecifically associated with responses with both hands. Therefore, Kopp et al. assumed that response priming would occur only with left- and right-pointing flankers. In fact, the N200-amplitude of NoGo targets was more enhanced when they were flankered by right- or left-pointing flankers, than when they were flankered neutrally. More interestingly, the N200-amplitude after incongruently flankered Go trials was more increased than that after congruently or neutrally flankered Go targets (for similar results see Kopp, Rist, & Mattler, 1996).

Therefore, Kopp et al. argued that the N200-amplitude represents either the detection or the inhibition of an inappropriate response tendency.

Additional support for the assumption that the N200-amplitude represents a central inhibitory mechanism came from Falkenstein, Hoormann, and Hohnsbein (1999). They assumed that participants who frequently perform an erroneous key press in NoGo trials (poor performers) would have a weakened and / or delayed inhibitory mechanism. Interestingly, the N200-amplitude of poor performers was smaller than that of participants who perform less erroneous key presses in NoGo trials (good performers). Furthermore, the onset of the N200 was delayed for poor performers.

2.1.2 Arguments against the N200 representing a central inhibitory mechanism

Contrary to findings suggesting that the N200 represents an inhibitory mechanism there are also arguments against such a conclusion. One argument is that the onset of the N200 occurs too late for the N200 representing the inhibitory mechanism. According to Filipović, Jahanshahi, and Rothwell (2000), in most studies the onset of muscle activity measured by EMG was earlier or at least concurrently with the onset of the N200 (e.g., Naito & Matsumara, 1996). Filipović et al. investigated inhibition with a S1-S2-paradigm. The participants saw two stimuli (S1 and S2) that were separated by a 2-s interval. The first stimulus was a Go cue (short green light) or a NoGo cue (short red light). The red light was always followed by the NoGo target (long red light) whereas the green light was followed with equal probability by a Go or a NoGo target. Filipović et al. compared the event-related potentials after S1 (decision to prepare a response or not) with those after S2 (decision to respond or not). In addition to a greater N200-amplitude in event-related potentials after a NoGo target (when S2 required no response) they found a more negative deflection in the time range 90-175 (N1) ms after NoGo associated stimuli (NoGo cue and NoGo target) than after Go associated stimuli (Go cue and Go target). Interestingly, the N200-amplitude after a Go cue (when S1 required preparing a response) was more negative than that after NoGo cue (when S1 meant not to prepare a response). In contrast to the N200, the N1 component arose before the onset of muscle activity. Therefore, according to the model of Bullock and Grossberg (1988; 1991) the N1 could represent inhibition of the Go-signal.

Furthermore, the more enhanced N200-amplitude after NoGo trials compared to that after Go trials is often not found for auditory stimuli (for a review see Falkenstein, Hoormann, & Hohnsbein, 1999). Therefore, Falkenstein et al. assumed that the N200-amplitude represents a modality specific inhibitory mechanism.

Another problem for accounts which assume that the N200 represents a central inhibitory mechanism arises from findings of a mainly bilateral locus of the N200 (e.g., van Boxtel, van der Molen, Jenning, & Brunia, 2001). As can be seen in Table 1, most researchers report a right hemispheric dominance concerning inhibition. Therefore, the N200 should also be more pronounced on the right side if it represented inhibitory brain activity. However, right dominance for the N200 was reported only occasionally (Filipović et al. 2000). In conclusion, it is very doubtful that the N200-amplitude represents the central inhibitory mechanism.

Moreover, the findings of Bruin, Wijers, and van Staveren (2001) are even in direct conflict with the assumption that the increased N200-amplitude after NoGo stimuli would be specific to inhibition. Bruin et al. failed to replicate Eimer's finding (1993) concerning the effect of response priming on the N200-amplitude. They had four different primes: A NoGo prime, which consisted of two brackets ([]), a specific Go prime pointing to the right response side ([>), a specific Go prime pointing to the left response side (<]), and a non-specific Go prime consisting of a double arrow (<>). The NoGo prime always preceded a NoGo target, which was symbolized by a circle. In one half of the trials with specific Go priming the specific Go primes preceded a Go target, which consisted of an arrow pointing to the same direction as the prime. In the other half, the specific Go primes were followed by a NoGo target. In one half of the non-specific Go priming the non-specific Go primes preceded both Go targets with equal probability, while in the other half, the non-specific Go primes preceded a NoGo target. Like in Eimer's experiments, the idea was that a NoGo target following a specific Go prime required more response inhibition than a NoGo target following a non-specific Go prime. If the N200 represents the central inhibitory mechanism, there should be an enhanced N200-amplitude in NoGo-trials with specific Go primes. Empirically, the N200-amplitude was not influenced by the different sorts of response priming. Therefore, Bruin et al. concluded that the increased N200-amplitude after NoGo stimuli is not specific to inhibition.

2.1.3 The N200 as representation of conflict monitoring

Inspired by the findings of Kopp, Rist, and Mattler (1996), Nieuwenhuis, Yeung, van den Wildenberg, and Ridderinkhof (2003) proposed that the N200 represents conflict monitoring. To test this assumption, Nieuwenhuis et al. varied response probability in a Go-NoGo paradigm. The Go probability was either 0.20, or 0.50, or 0.80. Nieuwenhuis et al. assumed that an infrequent stimulus should cause conflict because the frequency of a stimulus biases the subject's response tendency. If the rare stimulus occurs, the tendency to perform the response associated with the frequent stimulus must be overcome. Therefore, there should be more conflict following a rare stimulus than following a frequent stimulus. If the N200-amplitude represented response conflict, the probability of a stimulus, regardless of whether it is a Go or a NoGo stimulus should influence the N200-amplitude. Indeed, Nieuwenhuis et al. found an increased N200-amplitude after rare Go stimuli as well as after rare NoGo stimuli. While the idea that the N200-amplitude represents an inhibitory process can account for the finding that the N200-amplitude was enhanced for rare NoGo stimuli compared to that for frequent NoGo stimuli, it can not explain the modulation of N200-amplitude with Go stimuli.

However, there are also findings contradicting a connection between the N200 and conflict monitoring. Similarly to Nieuwenhuis et al. (2003), Bruin and Wijers (2002) compared the N200-amplitude after Go and NoGo trials with different Go probabilities (0.25, 0.50, and 0.75). But in contrast to the findings of Nieuwenhuis et al. the N200-amplitude in the study of Bruin and Wijers was increased after frequent Go trials. This finding contradicts the assumption that the N200-amplitude represents response conflict because in the frequent Go trials no prepotent response bias has to be overcome and therefore no conflict between two response tendencies should occur.

2.1.4 The N200 as representation of mental effort

If the N200 does not represent an inhibitory mechanism and it does not represent the monitoring of conflict, what is the cause of the N200 phenomenon? Perhaps we find an answer if we recapitulate the study of Bruin and Wijers (2002). They varied response probability in a Go/NoGo paradigm with two response modes. In one response mode participants had to perform an overt key press (manual condition), in the other response mode participants had to count the Go stimuli silently (count condition). Participants performed both response modes in separate blocks which were randomly mixed. Maybe, this intermixed structure of the paradigm induced participants to count Go stimuli in the manual condition as well. One could argue that this strategy would increase mental effort in a high Go probability condition. Contrariwise, in the study of Nieuwenhuis, Yeung, van den Wildenberg, and Ridderinkhof (2003) the most mental effort relative to the other Go conditions is needed in the rare Go stimuli condition because the prepotent NoGo response has to be overcome.

Furthermore, Nieuwenhuis et al. (2003) computed dipole source models of the observed scalp voltage distributions for the N200. The results of the modeling were consistent with a source for the N200 in the anterior cingulate cortex (ACC: Brodman's areas 24 and 32; for similar results see van Veen & Carter, 2002[b]). The role of the ACC is debated lively. As Table 1 shows, the ACC was often found to be more activated during tasks requiring inhibition than during tasks requiring the execution of a response. However, the ACC has long been interpreted as monitoring the occurrence of conflict on the response level (Botvinick, Nystrom, Fissell, Carter, & Cohen, 1999; Carter et al., 2000; Kerns et al., 2004; Macdonald, Cohen, Stenger, & Carter, 2000; Milham et al., 2001; van Veen, Cohen, Botvinick, Stenger, & Carter, 2001; van Veen & Carter, 2002[a]). Other studies found greater activation of the ACC in situations in which action-outcome was evaluated (see for a review Rushworth, Walton, Kennerley, & Bannerman, 2004). Recently, Botvinick, Cohen, and Carter (2004) tried to integrate the ACC-findings. They proposed that the ACC monitors task difficulty or mental effort.

In summary, there is some evidence that the N200-amplitude represents mental effort instead of a central inhibitory mechanism. Therefore, the relationship between the NoGo-Anteriorization and the central inhibitory mechanism is reconsidered in the next section.

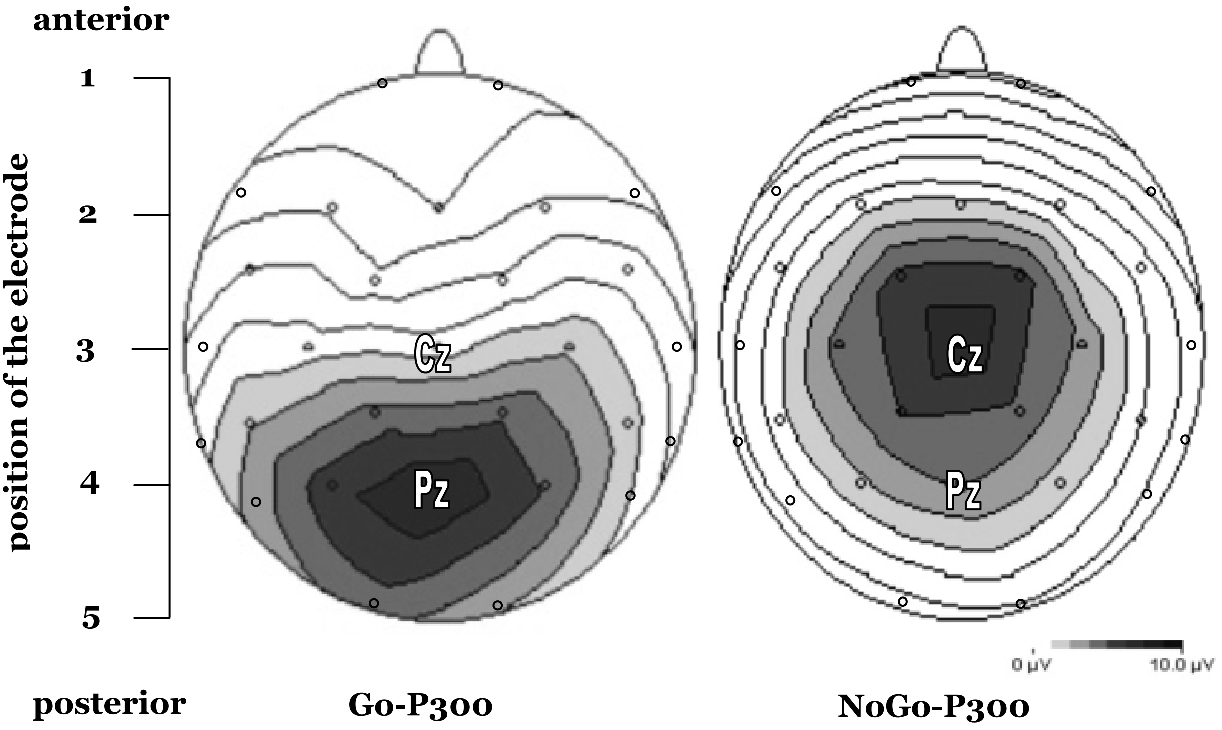
2.2 The NoGo-Anteriorization and inhibition

Despite the late onset of the NoGo-P300 (see section 1.4.2) the NoGo-Anteriorization could still be linked to inhibition. For example, the NoGo-Anteriorization could reflect a late general, modality–unspecific inhibitory effect on central response activation processes (e.g., De Jong, Coles, Logan, & Gratton, 1990; Falkenstein, Hoormann, & Hohnsbein, 2002). Therefore, it could be specific to inhibition. In fact, there is some evidence for this claim. However, there are also contrary findings. In the next sections first the NoGo-Anteriorization will be explained in more detail. Secondly, findings in support of a link between the NoGo-Anteriorization and the inhibitory mechanism will be listed. Finally, the arguments against a relationship between NoGo-Anteriorization and the inhibitory mechanism will be discussed.

2.2.1 The NoGo-Anteriorization

The NoGo-Anteriorization describes the topographic difference between the Go- and the NoGo-P300 at the time of the maximal root mean square of the brain electrical field (Peak of the Global Field Power; e.g., Lehmann, 1987) in the P300 time frame. The topography of the Go- and the NoGo-P300 is determined by calculating the amplitude-weighted center of gravity of the positive area of the average referenced electrical field (e.g., Fallgatter, Brandeis, & Strik, 1997; Fallgatter, 2000; Fallgatter & Strik, 1999; for a general description of the method to calculate centroids, see Lehmann, 1987, and Lehmann & Skrandies, 1984). The great advantage of this method is that the calculation of the topography depends on measured voltages of all electrodes. To visualize the centroid’s location in the anterior-posterior direction the electrodes as well as the location of the centroids are projected onto a line in the anterior-posterior direction (Figure 3). For this purpose, each centroid is quantified by a value ranging from 1 to 5 that indicates the anterior-posterior position of each electrode.

Figure 3 shows an illustration of the NoGo-Anteriorization. The scale on the left side shows the electrode position in anterior-posterior direction. The electrode positions are projected in a plane on the surface of a headshape. The voltage (µV) at each electrode is represented by different shades of gray, with darker grays indicating higher voltages. The Figure shows that the Go-P300 centroid (left panel) is located more posteriorly (close to Pz: Electrode position 4) than that of the NoGo-P300 (right panel; close to Cz: Electrode position 3). Therefore, in the following, the term NoGo-Anteriorization is used to describe the topographic difference between Go-and NoGo-P300[6].



Go-P300

NoGo-P300

Figure 3. Scalp topographic voltage maps of Go- and NoGo-P300 at the peak of the global field power in the P300 time frame.

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[6] The critical reader might note that Fallgatter and Strik (1999) narrowed the term NoGo-Anteriorization down to a parameter, which reflects the numerical topography-difference between Go- and NoGo-P300. However, in the following the term NoGo-Anteriorization is used to describe the topography-difference in a broader sense.

2.2.2 Arguments for a relationship between the NoGo-Anteriorization and an inhibitory mechanism

First of all, the NoGo-Anteriorization (NGA) is quite a robust phenomenon in the context of tasks that require inhibition of an action. It is elicited in Go-NoGo tasks with visual stimuli as well as in Go-NoGo tasks with auditory stimuli (e.g., Tekok-Kilic, Shucard, & Shucard, 2001). Nakata et al. (2004) found the NGA even in a Go-NoGo task with somatosensory stimuli (electrical stimulation of the hand).

Falkenstein, Hoormann, and Hohnsbein (2002) showed that the NoGo-P300 is also largely uninfluenced by mental fatigue. In their study participants had to perform four blocks of a speeded Go-NoGo task with visual and auditory stimuli. The first two blocks were divided from the second two blocks by four hours in which the participants had to perform other tasks with either high visual or auditory workload. Falkenstein et al. did not find any influence of time-on-task on the NoGo-P300.

Additionally, Fallgatter, Aranda, Bartsch, and Herrmann (2002) investigated the stability of the NGA (location of the Go and NoGo centroid, difference between Go and NoGo centroid) for different participants. They found that the NGA differed between participants. However, the individual characteristics of the NGA were very stable over time. More than two years after the first performance in a Go-NoGo task the test-retest reliability was more than 0.85 for the characteristics of the NGA.

Furthermore, Eimer (1993) as well as Bruin, Wijers, and van Staveren (2001) showed that the NoGo-P300 was affected by response priming. A prime that preceded NoGo stimuli and instructed the participants to prepare a response with a specific hand elicited a greater NoGo-P300 than a prime that only unspecificly instructed the participants to prepare a response. This is well in line with the assumption that the NoGo-P300 is linked to the inhibitory mechanism because inhibition should be greater with specific Go primes. Interestingly, in the study of Bruin et al. a prime that instructed participants not to prepare a response and always preceded NoGo stimuli almost erased the NoGo-P300. Moreover, Fallgatter et al. (2001) also reported that the NGA vanished in predictable Go and NoGo sequences. This is well in line with the assumption of a relationship between

NGA and an inhibitory mechanism. When the participant knows that no response will be required, the participant does not prepare a response. Consequently, no inhibition occurs in expected NoGo trials.

Additional support for a relationship between NGA and the inhibitory mechanism came from Strik, Fallgatter, Brandeis, and Pascual-Marqui (1998). They computed a right frontal source for the NoGo-P300[7]. As Table 1 shows, right-hemispheric sources were commonly implicated in studies investigating the locus of the inhibitory mechanism.

2.2.3 Objections against a relationship between NoGo-Anteriorization and an inhibitory mechanism

There are three alternatives to the assumption that the NGA is specific to an inhibitory mechanism. In the following these alternatives are introduced.

(1) NoGo-Anteriorization as an artifact due to motor activation

A very basic criticism against a relationship between NoGo-Anteriorization (NGA) and the inhibitory mechanism is the assumption that the NGA is merely an artifact of EEG-deflections that accompany motor responses. For instance Kok (1988) proposed that motor related potentials and potentials that represent response expectation or preparation (contingent negative variation, CNV) distort the topographies of Go- and NoGo-P300. According to Kok, the Go-P300 on its own does not differ from the NoGo-P300. However, the topographic difference between Go- and NoGo-P300 emerges, because negative motor related potentials with maxima at midline electrodes erase the more anteriorly located, positive parts of the Go-P300. Additionally, the CNV (negative deflection before responses, with a negative maximum at the Cz-electrode) decrease earlier in NoGo conditions than in Go conditions. Accordingly, the absence of the negative potential at the Cz-electrode in the NoGo-P300 timeframe increases the positivity at midline electrodes in NoGo trials. In line with Kok's assumptions, van Boxtel, van der Molen, Jennings, and Brunia (2001) reported an interaction between response

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[7] For a different result, namely a left-frontal source for the NoGo-P300, see Bokura, Yamaguchi, and Kobayashi (2001).

side and topography of the Go-P300, which they assumed as typical for contralaterally organized motor related potentials (p. 249). Therefore, they concluded that the NGA was the result of motor related negativity in Go trials instead of the result of an inhibition-related positivity.

(2) NoGo-Anteriorization as representation of response conflict

A second objection against a link between NGA and the inhibitory mechanism concerns the source of the NGA. Fallgatter, Bartsch, and Herrmann (2002) computed the anterior cingulate cortex (ACC) as the source for the NGA, because the ACC was more activated in NoGo conditions than in Go conditions. This finding is problematic for the assumption of the NGA being specific to the occurrence of inhibition, because, as we have seen in section 2.1.4, recent studies suggest the ACC to be the location of conflict on a response level (Milham et al., 2001; VanVeen, Cohen, Botvinick, Stenger, & Carter, 2001) or to be active during mental effort (Bottvinick, Cohen, & Carter, 2004). Contrary, both lesion studies and neuroimaging studies favor the inferior frontal cortex as being relevant for response inhibition (see Table 1). Thus, if the ACC is responsible for the anterior shift of the NoGo-P300-topography, the NGA could be the result of greater conflict and or higher mental effort in the NoGo condition compared to the Go condition. According to the Horse Race Model, this sounds reasonable because in NoGo trials two competing processes (response process and inhibitory process) are active at the same time. Therefore, a conflict between these processes could be the reason for the NGA.

(3) NoGo-Anteriorization as representation of less cognitive response selection in NoGo trials

A third objection against a connection between NGA and the inhibitory mechanism came from Falkenstein, Koshlykova, Kiroj, Hoormann, and Hohnsbein (1995). They proposed that the P300 is the result of two event-related potentials (ERPs) in the P300 time frame. One ERP, which they named P-SR, is assumed to have its maximum over the Cz-electrode and to represent stimulus identification (Hohnsbein, Falkenstein, & Hoormann, 1998). The other ERP (P-CR) is assumed to be maximal over the Pz-electrode and to represent cognitive response selection

(Falkenstein et al., 1995). While P-SR is assumed to have the same amplitude in Go as in NoGo trials, the amplitude of P-CR is assumed to be greater in Go trials than in NoGo trials. In fact, Falkenstein et al. observed two positive ERPs in the P300 time frame after auditory Go and NoGo stimuli. They proposed that P-SR and P-CR always occur after Go stimuli as well as after NoGo stimuli. However, in Go-NoGo tasks with visual stimuli the P-CR and the P-SR overlap[8] . Therefore, only one ERP is seen in visual Go-NoGo tasks. Furthermore, Falkenstein et al. proposed that the topographic difference between Go- and NoGo-P300 could be the result of a differential overlap of P-SR and P-CR in Go and NoGo trials.

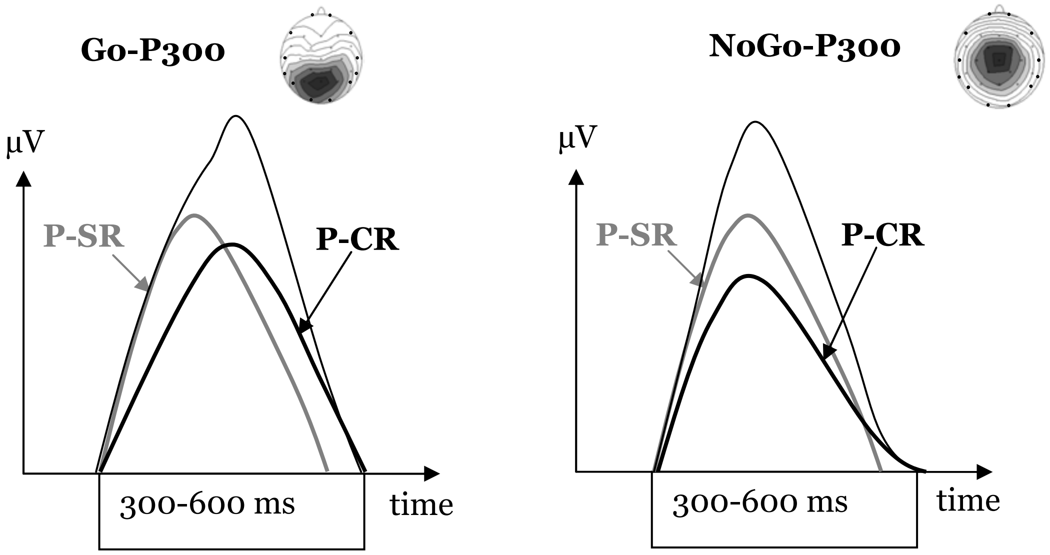

Figure 4. Illustration of the differential overlap of P-SR and P-CR in Go-(left panel) and NoGo-P300 (right panel).

Figure 4 illustrates the assumptions for the Go- (left panel) and the NoGo-P300 (right panel). In Go trials the P-CR should peak later and it would be enhanced compared to the P-CR in NoGo trials. Therefore, the overlap would start later and the peak of the observed Go-P300 would be shifted towards the maximum of the P-CR potential. Furthermore, the greater influence of the P-CR in Go trials than in NoGo trials would lead to a posteriorly located maximum of the Go-P300. In contrast, the smaller and earlier peaking P-CR in NoGo trials than in Go trials

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[8] Falkenstein et al. (1995) do not explain why they suppose a greater overlap of P-SR and P-CR with visual stimuli compared to auditory stimuli. However, the graphical presentation of their model suggests slower stimulus identification for visual than for auditory stimuli.

would increase the amplitude of the NoGo-P300 over the maximum of the P-SR. Thus, the topography of the NoGo-P300 would be especially influenced by the P-SR.

These assumptions are problematic because of the fact that the NoGo-P300 usually peaks later than the Go-P300 (e.g., Fallgatter, Brandeis, & Strik, 1997; Pfefferbaum, Ford, Weller, & Kopell, 1985). However, an earlier peaking P-CR in NoGo trials is not necessary in order to save Falkenstein et al.'s ideas. A smaller P-CR amplitude in NoGo trials than in Go trials could explain the more anterior topography of the NoGo-P300 on its own. The smaller influence of the P-CR amplitude on the NoGo-P300 would result in a shift towards the topography of the P-SR. In contrast, the greater influence of the P-CR on the Go-P300 would shift the topography towards that of the P-CR. Therefore, the NGA could be the result of a smaller P-CR representing less cognitive response selection in NoGo trials than in Go trials.

2.3 Open questions

The former section demonstrates that the real meaning of the NGA is lively debated. The aim of the empirical part is to exclude false assumptions about the NGA's nature. However, the inhibitory mechanism is not directly observable. Therefore, a relationship between the inhibitory mechanism and the NGA can not be investigated directly. Thus, hypotheses are developed which question each counter-argument to a connection between NGA and inhibitory mechanism.

(1) Is the NoGo-Anteriorization an artifact due to motor activation?

To test this thesis, Go and NoGo primes are subliminally presented before Go and NoGo targets in Experiment 1 and in Experiment 2 (for details see section 3 and section 4). If the NGA represents motor potentials during Go trials, prepotent motor activation, which is induced by Go primes, should also erase the anterior part of the P300 after a NoGo target. Furthermore, NoGo primes should have no influence on the P300-topography. On the contrary, if the NGA is related to the inhibitory mechanism, prepotent motor activation should even increase the necessity for inhibition in a NoGo trial. Therefore, Go primes preceding NoGo targets should cause a P300 with a more anteriorly located centroid compared to that of a normal Go-P300. Additionally, a NoGo prime preceding a Go target should influence the topography of the Go-P300.

(2) Does NoGo-Anteriorization represent response conflict?

In Experiment 3, a Go and a NoGo condition with a similar intense of conflict are compared. The topographic difference between Go- and NoGo-P300 (NGA) under these conditions should be small, if the NGA is the result of higher response conflict and or higher mental effort in NoGo trials. However, if the NGA is connected to the inhibitory mechanism, the NGA should be unaffected by the amount of conflict in conditions.

(3) Does the NoGo-Anteriorization represent less cognitive response selection in NoGo trials?

According to Falkenstein, Koshlykova, Kiroj, Hoormann, and Hohnsbein (1995) the P300 consists of two overlapping event-related potentials (ERPs). These ERPs are: An ERP with a maximum over the Cz-electrode that is assumed to represent stimulus identification (P-SR) and another ERP with a maximum over the Pz-electrode that is assumed to represent cognitive response selection (P-CR). The topographic difference could be the result of a larger P-CR in Go trials than in NoGo trials.

To test these assumptions, in Experiment 4 Go and NoGo trials are compared without an exogenous signal, that has to be evaluated. If the NGA is the result of a larger P-CR in Go than in NoGo trials, a smaller P300 with a parietal maximum should occur in NoGo trials than in Go trials. Contrary, there should be still more activity over the Cz-electrode in NoGo trials, if the NGA is linked to the inhibitory mechanism.

3 Is the NoGo-Anteriorization an artifact due to motor activation?

3.1 Experiment 1: Does motor activation erase more anteriorly located P300 activity?

The thesis that motor activation erases more anteriorly located P300 activity (Kok, 1988) is at odds with the finding, that the NoGo-Anteriorization (NGA) was still present in experiments in which participants counted the Go stimuli silently (e.g., Pfefferbaum, Ford, Weller, & Kopell, 1985, Bruin & Wijers, 2002). Therefore, the NGA can not simply be the result of motor potentials erasing the anterior part of the P300 in Go trials, unless silently counting Go stimuli evoke motor potentials.

In fact, Bruin and Wijers considered the possibility that inner speech in Go trials might activate vocal system muscles. This activation might in turn cause the erasement of the anterior part of the Go-P300. This idea was supported by findings of Shergill et al. (2002) who demonstrated that a faster rate of inner speech correlated with higher activation in the frontal cortex. Accordingly, experiments using silent counting of Go trials are not suitable for answering the question if motor activation erases more anteriorly located P300 activity.

Therefore, instead of trying to create a Go condition without motor activation, this experiment used a NoGo condition that was contaminated by motor activation. This was achieved by means of priming. A NoGo condition in which a Go prime preceded a NoGo stimulus should result in motor activation during a NoGo condition. To exclude strategic influences on the part of the participants, primes were presented subliminally. That means, stimuli that were used as primes were presented very briefly (35 ms) and they were followed immediately by a mask to erase the retinal afterimage. Recent studies show that subliminally presented primes elicit LRPs, indicating that the primes do indeed cause motor activation in the absence of conscious stimulus processing (e.g., Eimer & Schlaghecken, 1998). The visibility of the primes is usually tested in an additional discrimination task at the end of the experiment.

Table 2 shows the expectations regarding topography if the NGA was an artifact of motor activation (left column). If the NGA is the result of the negative, central potentials that accompany motor activation, then these potentials should erase the anterior part of the P300 in Go conditions (Congruent_Go, Neutral_Go, and NoGo_Go; first three rows in the left column) as well as in the NoGo condition with Go priming (Go_NoGo; last row in the left column). This means that the P300 of the Go_NoGo condition (last row in the left column) should resemble a Go-P300 instead of a NoGo-P300. Consequently, the maximum of the Go_NoGo condition should be located more posteriorly than the maximum of a NoGo condition with NoGo priming (Congruent_NoGo; fourth row in the left column) or than that of a NoGo condition that was preceded by a symbol that was never presented supraliminally (Neutral_NoGo; fifth row in the left column)[9].

In contrast, if the NGA was caused by the central inhibitory mechanism elicited by the NoGo symbol (right column of Table 2), the maximum of a NoGo primed Go condition (NoGo_Go; third row in the right column) should be located more anteriorly than that of a Go condition preceded by a Go prime (Congruent_Go; first row in the right column) or than that of a Go condition preceded by a symbol that participants never saw supraliminally (Neutral_Go; second row in the right column)[10]. However, the maximum of NoGo_Go (third row in the right column) should be located more posteriorly than that of NoGo conditions (Congruent_NoGo, Neutral_NoGo, and Go_NoGo; lower part of the right column), since in the NoGo conditions more inhibition has to take place. Furthermore, the P300-centroid of the Go_NoGo condition (last row in the right

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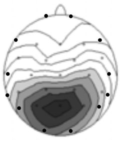
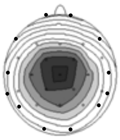
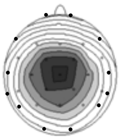
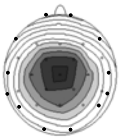
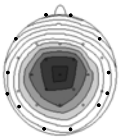
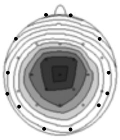
[9] The exclusively subliminal presentation of the neutral prime assured that the prime was neither linked to a response process nor linked to an inhibitory process in the course of the experiment.

[10] Of course, this can only be expected under the assumption that a subliminally presented prime is able to elicit the central inhibitory mechanism.

column) should not be located more posteriorly than that of the other NoGo conditions.

Table 2. Summary of the expected topographies under the hypothesis that the NGA is an artifact of motor potentials or that it is caused by a central inhibitory mechanism.

Conditions (prime_target)	NGA = artifact of motor potentials	NGA=central inhibitory mechanism
Congruent_Go		
Neutral_Go		
NoGo_Go		
Congruent_NoGo		
Neutral_NoGo		
Go_NoGo		

3.1.1Method

(1) Participants

A total of 15 women and 9 men aged between 19 and 48 years (mean age 24.4 years) took part in the experiment. None of them suffered from neurological or psychiatric diseases (self-reported). All had normal or corrected-to-normal vision. Two persons were left-handed; the others were right-handed (self-reported). They received partial course credit or were paid for participation. Participants were neither informed about the presentation of subliminal primes nor about the study’s specific hypotheses until the end of the experiment. After the experiment all participants gave their informed consent prior to their inclusion in the study.

One participant had to be excluded from further analysis because he had a very high discrimination rate (d’ = 1.34) in the prime discrimination task. Furthermore, 4 participants had to be excluded from the EEG data set due to having less than 20 artifact-free segments per condition after the artifact rejection procedure. The final EEG-data set consisted of 12 women and 7 men aged between 19 and 48 years (mean age 23.9 years). All but one of those remaining participants were right-handed.

(2) Apparatus and stimulus materials

Participants performed the experiment in a dimly illuminated and sound attenuated room. An IBM-compatible computer (Pentium IV with 1.8 GHz) with a 17 inch VGA-Display (85 Hz) and the software E-Prime (Schneider, Eschman, & Zuccolotto, 2002) served for stimulus presentation and data acquisition. The viewing distance was 60 cm. Responses were executed on the space-bar of a standard keyboard.

Stimuli were presented in white on a black background. The letters X, T, and O served as primes, the letters X and O served as targets. Primes and targets were presented in the font Times New Roman (36 points). Pre- and postmask consisted of five Greek letters displayed in the font Symbol (36 points). The five letters were randomly drawn from a set of 10 letters (ΩΠΣΔΦΓϑΛΨΞ).

(3) Procedure

Participants were instructed to perform a simple Go-NoGo-task. Thus, they had to press a key when the X was presented and to withhold responding after the presentation of an O or vice versa. They were not informed that there was any prime before target onset.

Table 2 shows the types of priming. When prime and target were the Go symbol, the trial was a congruent Go trial (Congruent_Go). When prime and target were a NoGo symbol, the trial was a congruent NoGo trial (Congruent_NoGo). The letter T served as neutral prime prior to the Go (Neutral_Go) or the NoGo stimulus (Neutral_NoGo). When a NoGo prime preceded a Go target, the trial was called NoGo_Go, whereas a Go prime followed by a NoGo target was called Go_NoGo trial.

Each trial started with the presentation of the premask for 70-ms which was followed by the presentation of the prime for 35-ms. Afterwards, a 70-ms postmask was presented which was followed by the 200-ms target. The duration of prime, pre-, and postmask resulted as an adaptation to the retrace rate of the monitor (11.7 ms). The participants had to respond within the subsequent 800 ms with their right index finger; otherwise the German word for “faster” appeared for 1500 ms on the monitor. Response times were recorded from target-onset to response onset. The next trial was presented 2000 ms after target offset.

Participants performed four blocks with 78 trials each, in a random order. Each of the six types of trials was presented 52 times.

After completing the Go-NoGo task, participants performed a prime-discrimination task, which consisted of 96 trials. Half of the trials contained the neutral prime T, the other half consisted of the other types of primes, with each type of prime appearing an equal number of times. Participants were informed about the presence and possible types of primes. They were instructed to indicate which of the three letters they had seen by pressing the corresponding key of the computer keyboard.

(4) EEG-Recordings and data analysis

Electrophysiological recordings were obtained by using two electrode caps in the sizes 54 and 58 cm (BrainCap, BrainProducts GmbH). The electrodes of these caps (FP1, FP2, F7, F3, Fz, F4, F8, FC5, FC1, FC2, FC6, T7, C3, Cz, C4, T8, CP5, CP1, CP2, CP6, P7, P3, Pz, P4, P8, O1, O2) were placed according to the enhanced International 10-20-system (Jasper, 1958) and referenced to the FCz-electrode. The ground electrode was placed between the electrode Fz and the position of the electrode FPz. Eye movement was monitored by the use of two electrodes attached to the outer canthus of each eye (horizontal electrooculogram, EOG) and one electrode attached under the left eye (vertical EOG). Electrode resistance was kept below 5 kΩ. The sampling rate of the electroencephalogram (EEG) was 1000 Hz. A bandpass filter of 0.1-70 Hz was used as well as an online notchfilter.

The software Brain Vision Analyzer 1.05 was used for analysis of the EEG-data. After data acquisition, the data were rereferenced to average reference (i.e., the average voltage of all electrodes, except the ocular electrodes, served as new reference). To reduce high-frequency activity, the data were filtered digitally with a 30 Hz low pass filter. Event related potentials (ERPs) containing eye movements were corrected using the regression procedure of Gratton, Coles, and Donchin (1983) without raw average subtraction. An ERP was defined as starting with prime onset and lasting 700 ms. ERPs with amplitudes exceeding ±50µV in any of the electrodes were discarded.

Only artifact-free or artifact corrected ERPs, recorded during correctly answered trials, were averaged per condition and person. Four participants, who had less than 20 artifact-free ERPs per condition, were excluded. Mean amplitude values were computed within each 50 ms interval of the 700-ms averages, resulting in 14 segments per participant, condition, and electrode.

Additionally, the peaks of the global field power (GFP, Lehmann & Skrandies, 1980) within the P300-time window were used to determine the area centroids of the six conditions (Lehmann, 1987; see also section 2.2.1). The peaks were searched by the semi-automated peak-detection tool of the Vision Analyzer software[11]. To quantify the difference in topography between Go and NoGo conditions in the anterior-posterior direction, the centroids were calculated. The quantification of the centroids' anterior-posterior location is described in section 2.2.1.

All statistical analyses were performed with SPSS, version 11.5.1. As recommended by Quintana and Maxwell (1994), the Huynh-Feldt correction (Huynh & Feldt, 1976) was applied with a Huynh-Feldt epsilon ≥ 0.75, whereas the Greenhouse Geisser correction (Greenhouse & Geisser, 1954) was used with a Huynh-Feldt epsilon < 0.75.

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[11] If in the EEG-data of a participant the P300 peak was not within the P300-time frame of 300-600 ms the time frame was enhanced to catch the peak.

3.1.2 Results

(1) Prime visibility

For each participant the discrimination performance was calculated as d' (i.e., the ratio of detecting the neutral prime when it was presented [hits] and indicating to have seen the neutral prime when the two other primes were presented [false alarms]). One participant had a d'-value greater than 1 (1.34) and was excluded from further analysis. For the remaining participants the mean d' was .10 (mean hit rate 39, 7%, false alarm rate 36.0%), which did not differ significantly from zero, $t(22) = 1.53$, $p = .14$.

(2) Behavioral data

Mean reaction times (RTs) for the Go conditions and error rates for the Go and the NoGo conditions are presented in Table 3. Participants responded fastest in the congruent Go condition (407 ms), slightly slower in the neutral Go condition (408 ms), and slowest in the NoGo_Go condition (412 ms). There were almost no errors in the Go conditions (< 1%). Of these few errors, the most were committed in the NoGo_Go condition (0.8%), somewhat less in the congruent Go condition (0.7%) and the least in the neutral Go condition (0.6%). For the NoGo condition participants had the most errors in the Go_NoGo condition (3.2%), less in the neutral NoGo condition (1.7%) and least in the congruent NoGo condition (1.5%).

Table 3. Mean RTs of the three Go conditions and error rates for Go and NoGo conditions

Condition	Mean RT (SD)	Error rates (SD)
Congruent_Go	407 ms (32 ms)	0.7 % (1.1%)
Neutral_Go	408 ms (34 ms)	0.6% (1.1%)
NoGo_Go	412 ms (31 ms)	0.8% (1.7%)
Congruent_NoGo		1.5% (1.8 %)
Neutral_NoGo		1.7% (1.7%)
Go_NoGo		3.2% (4.6%)

Note. RT = reaction time, SD = standard deviation

An analysis of variance (ANOVA) on mean RTs of correct Go trials with repeated measures on the factor type of priming (congruent, neutral, and NoGo) just missed significance, $F(2, 44) = 2.40$, $p < .11$, $\eta^2 = .10$.

While the factor type of priming was not significant in an ANOVA with the within-subject-factor type of priming (congruent, neutral, and NoGo) for the error rates in the Go conditions, $F(2, 44) = 0.77$, $p = .47$,. $\eta^2 = .03$, it was marginally significant in the ANOVA with the within-subject-factor type of priming (congruent, neutral, and Go) for the NoGo conditions, $F(2, 44) = 3.10$, $p = .07$,. $\eta^2 = .12$.

(3) ERP-data

Figure 5 shows the ERPs at the Cz- and the Pz-electrode for the three Go- and NoGo-conditions. Since the ERPs start with prime onset, the onset of the target is indicated by an arrow. The Cz- and the Pz-electrode were chosen, because the centroid of the NoGo-P300 is close to Cz whereas the centroid of the Go-P300 is typical at Pz. Graphical inspection revealed that the ERPs differed between Go- and NoGo-conditions. Differences associated with the type of priming were small at best.

Since the main interest of this study lies in the P300-amplitude, the statistical analyses were limited to the segments in the P300 time frame of the target (400 -700 ms after prime onset).

For the Cz-electrode data, an ANOVA was conducted with repeated measures on the factors task (Go vs. NoGo), type of priming (congruent, neutral, NoGo_Go/Go_NoGo), and time (50 ms-segments spanning the period from 400-700 ms after prime onset).

There was a significant main effect of time, $F(5, 90) = 5.42$, $p < .05$, $\eta^2 = .23$, and a significant interaction between the factors task and time, $F(5, 90) = 9.63$, $p < .001$, $\eta^2 = .35$. T-tests revealed a significantly smaller Cz-amplitude for the Go conditions compared to the NoGo conditions in the segments 500-600 ms after prime onset, all *p*s < 05.

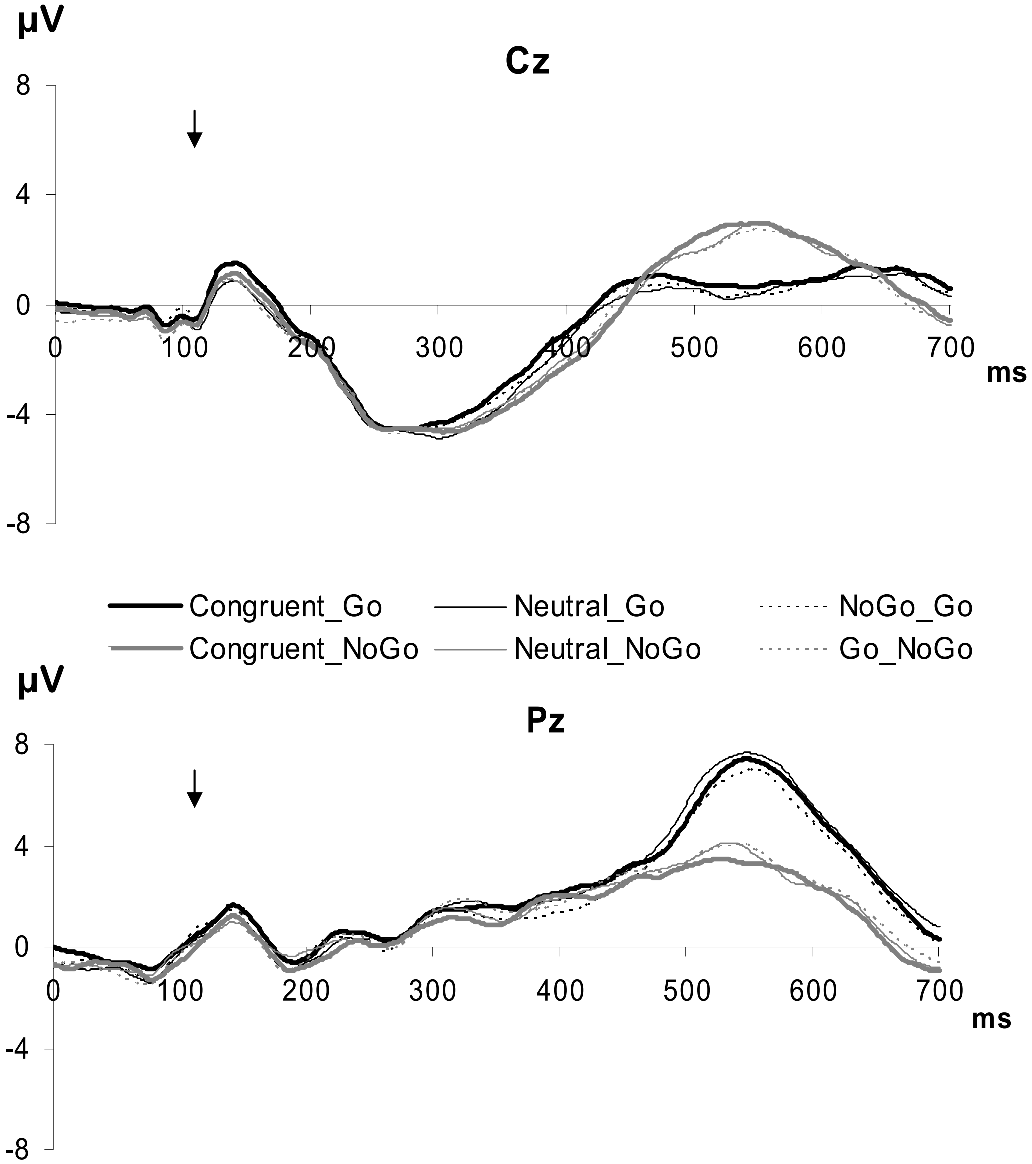

Figure 5. Grand-averaged event-related potentials over 19 participants. Comparisons between the three Go conditions (congruent, neutral, and NoGo_Go) and the three NoGo conditions (congruent, neutral, and Go_NoGo). ERPs are shown for the Cz- (above) and the Pz-electrode (below). The arrow indicates target onset.

For the Pz-electrode data, an ANOVA was conducted with repeated measures on the factors task (Go vs. NoGo), type of priming (congruent, neutral, NoGo_Go/Go_NoGo), and time (50 ms-segments spanning the period from 400-700 ms after prime onset).

The factors task, $F(1, 18) = 77.36$, $p < .001$, $\eta^2 = .81$, type of priming, $F(2, 36) = 4.82$, $p < .05$, $\eta^2 = .21$, and time, $F(5, 90) = 28.44$, $p < .001$, $\eta^2 = .61$, had a significant influence on the amplitude. Like for the Cz-electrode data, there was a significant interaction between the factors task and time, $F(5, 90) = 21.49$, $p < .001$, $\eta^2 = .54$. No other effects reached the level of significance, *p*s > .30.

T-tests showed a significantly enhanced Pz-amplitude for the Go conditions compared to the NoGo conditions in the segments 500-700 ms after prime onset, all *p*s < .05. The main effect for type of priming resulted from a significantly smaller Pz-amplitude for Go_NoGo and NoGo_Go compared to congruent and neutral priming conditions, all *p*s < .01.

(4) Topographical mappings

The upper half of Figure 6 shows the scalp topographic voltage maps for Go and NoGo conditions at the global field power peak in the P300 time frame. Graphical inspection showed that while all three Go centroids resembled each other highly, they differed from the NoGo centroids. Even though all three NoGo centroids are located more anteriorly than the Go centroids, the centroid of the congruent NoGo condition appeared to be located less anteriorly than the centroids of the other two NoGo conditions.

In an ANOVA with repeated measures on the factors task (Go and NoGo) and type of priming (congruent, neutral, and NoGo_Go/Go_NoGo), main effects were observed for the factors task, $F(1, 18) = 11.55$, $p < .01$, $\eta^2 = .39$, and type of priming, $F(2, 36) = 3.70$, $p < .05$, $\eta^2 = .19$. There was also a significant interaction between task and type of priming, $F(2, 36) = 4.94$, $p < .05$, $\eta^2 = .30$.

Additional t-tests showed, that the Go conditions did not differ significantly ($p > .30$), whereas there was a significant difference between the NoGo conditions. The centroid of the congruent NoGo condition was located less anteriorly than the centroid of the neutral or the Go_NoGo condition (*p*s < .05). In fact, the centroid of the congruent NoGo condition did not differ from any of the centroids of the three Go conditions in the anterior-posterior direction (*p*s > .12), whereas the centroids of the other two NoGo conditions were located more anteriorly than the centroids of the Go conditions (*p*s < .01). Furthermore, the centroids of the Neutral_NoGo condition and the Go_NoGo condition did not differ from each other concerning their location, $t(18) = 1.06$, $p = .31$.

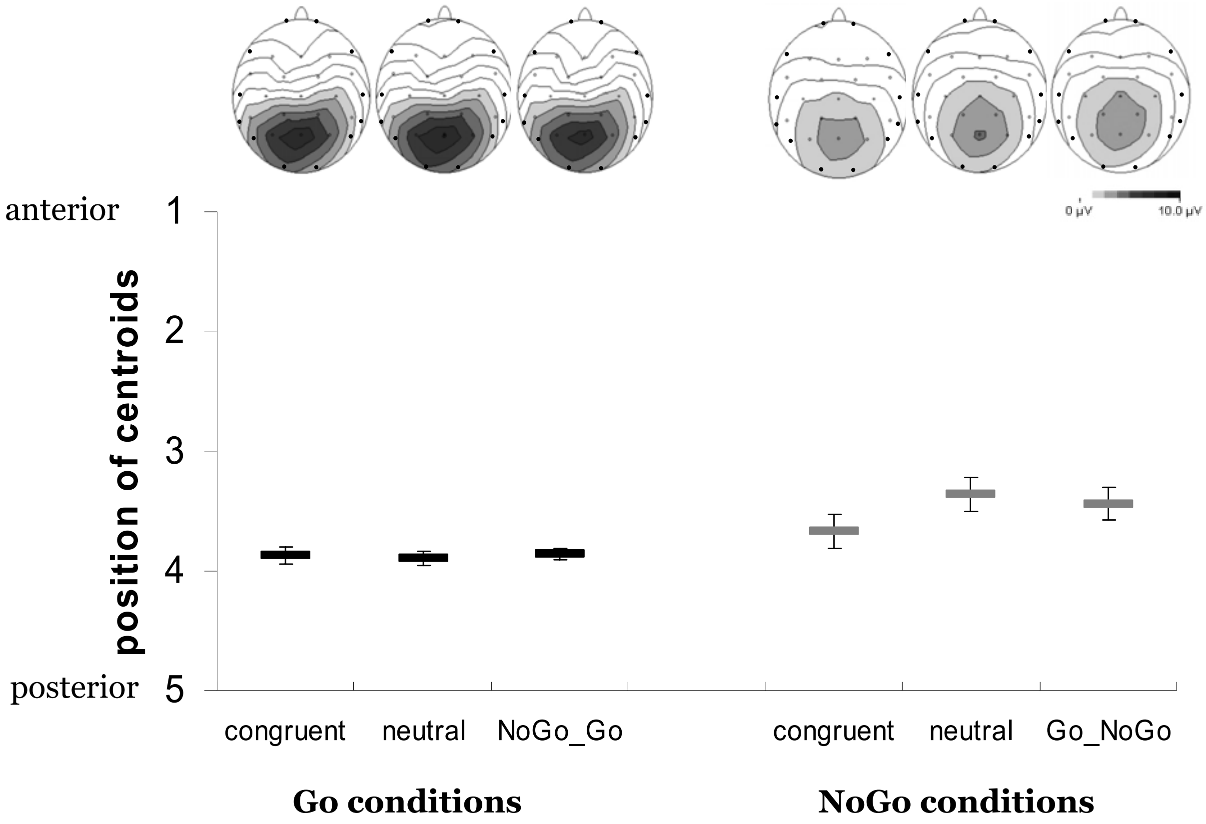

Figure 6. Positions of the centroids of the Go and NoGo conditions at the peak of the global field power in the P300 time frame and corresponding scalp topographic voltage maps (upper panel).

3.1.3 Discussion

Experiment 1 investigated, whether motor activation erases more anteriorly located P300 activity in Go trials or whether the topographic difference between Go- and NoGo-P300 is the result of a central process elicited by NoGo signals. For this purpose, Go, neutral, or NoGo primes were subliminally presented prior to Go or NoGo targets. The subliminal presentation of the primes was assured, since the mean discrimination rate for the primes did not deviate from zero.

However, the priming effect in behavioral data was small at best. There was a slight trend in the Go RTs indicating that priming had an influence on response speed such that Go primes in the Congruent_Go condition speeded up responses whereas NoGo primes in the NoGo_Go condition slowed the response time down. But these effects did not reach significance. Similarly, there was a non-significant trend in the error rates of the NoGo conditions. The least errors were performed in the Congruent_NoGo condition whereas the most errors were performed in the Go_NoGo condition.

Similar to the small behavioral effects of the primes, the ERPs showed only few prime-induced effects. The Cz-amplitudes of all NoGo conditions in the P300 time frame of the target were enhanced compared to those of the Go conditions, independent of priming. Also the Pz-amplitudes of the three Go conditions in the P300 time frame of the target were increased compared to those of the NoGo conditions. Therefore, the typical Go/NoGo differences were present on an ERP level. Interestingly, the subliminally presented Go and NoGo primes in the Go_NoGo and NoGo_Go condition decreased the Pz-amplitude in these conditions compared to that in the congruent and neutral conditions. The reason for this finding is difficult to interpret.

The centroid of the Go_NoGo condition was not located more posterior than that of the other NoGo conditions. The enhanced motor activity triggered by the Go prime did not result in a more posterior topography. Interestingly, the centroid of the congruent NoGo condition in Experiment 1 was located more posterior than those of the other NoGo conditions. It was not even more anteriorly located than the centroids of the Go conditions. It seems as if the NoGo prime has already inhibited any preparation and therefore no inhibition is needed at the time the NoGo stimuli occur. This is well in line with the results of Fallgatter et al. (2001) who showed that the NGA vanished in predictable NoGo trials.

Taken together, Experiment 1 does neither support the hypothesis that motor activation erases more anteriorly located P300 activity in Go trials nor does it support the idea that the topographic difference between Go- and NoGo-P300 is the result of a central process during NoGo trials. However, the small priming effects do not allow any firm conclusions. Therefore, a second experiment was conducted not just to replicate, but to increase the priming effects of experiment 1. The idea of experiment 2 was to bring in a second response alternative to complicate the task and enhance the benefits and cost of primes. Additionally, the somewhat arbitrarily selected letters were replaced by arrow stimuli, for which a natural relation exists between the symbol and the response.

3.2 Experiment 2: Does the NoGo-Anteriorization represent a central process triggered by the NoGo stimuli?

The results of Experiment 1 did not support the hypothesis that the topographic difference between Go- and NoGo-P300 is due to an artifact. The negative motor activation induced by Go primes did not erase the anterior part of the NoGo-P300. Conversely, the results do not support the assumption of a central process triggered by NoGo signals either. However, priming effects were at best small. Therefore, Experiment 2 was designed to increase the priming effects.

Primes and targets were borrowed from a study by Kunde (2003; see also Vorberg, Mattler, Heinecke, Schmidt, & Schwarbach, 2003). The critical characteristics of these stimuli is that the primes exactly fit within a cutting in the middle of the targets, which results in efficient masking of the primes. The participants' task was to respond to the direction of two Go targets (arrows pointing to the left or to the right side; see Figure 7). They had to withhold their response for a NoGo target, which consisted of the overlapping right-and left-pointing Go targets. The targets were preceded by one of four types of primes (left-pointing-arrow, right-pointing arrow, neutral prime = square, and NoGo prime = double-arrow; see Figure 7).

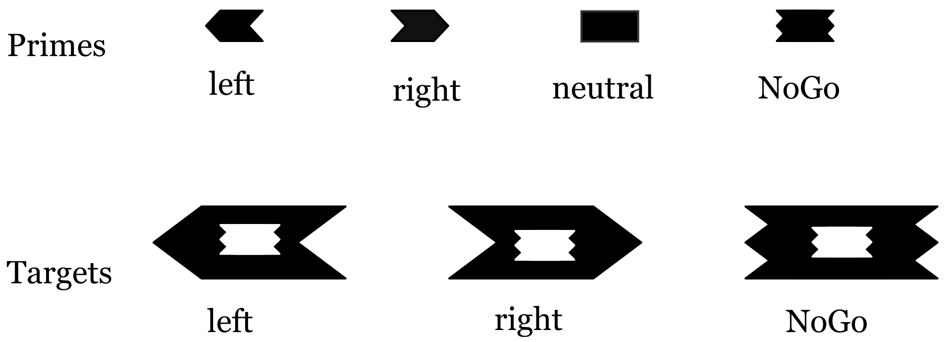

Figure 7. Stimuli applied in Experiment 2.

So in the congruent Go and NoGo condition a smaller version of the target preceded the target, whereas in the neutral Go and NoGo condition the neutral prime preceded Go and NoGo targets. The Go_NoGo condition consisted of the left-or the right-pointing arrow preceding a NoGo target, and the NoGo_Go condition consisted of one of the two Go targets following a NoGo prime. Finally, in the incongruent Go condition the prime pointed to the opposite direction as the Go target.

The more natural relationship between a left-pointing arrow and a left response and between a right-pointing arrow and a right response, respectively, is expected to result in strong motor activation. Furthermore, the use of two response alternatives allows the comparison of a NoGo primed Go condition with a Go condition in which the opposite response side has been primed. According to De Jong, Coles, and Logan (1995) inhibition has to take place in this incongruent Go condition. The activation of the triggered response side resulting from the prime has to be overcome and a response with the opposite response side has to be executed. Therefore, the following results were expected in Experiment 2.

First of all, as the results of Experiment 1 indicated, the NoGo-P300 is no result of missing motor potentials in NoGo trials. Therefore, the same anteriorly located P300 centroid is assumed for the Go primed NoGo condition as for the other NoGo conditions. Secondly, if a NoGo-signal triggers a central process, the P300 centroid of a NoGo primed Go condition should be located more anteriorly than that of a Go or neutrally primed Go condition. Thirdly, if a central process is necessary in the incongruent Go condition to avoid the execution of a wrong response and the NoGo-P300 is linked to this central process, the P300 centroid of the incongruent Go condition should be located more anteriorly than those of the Go or neutrally primed Go condition.

3.2.1 Method

(1) Participants

A total of 12 women and 4 men aged between 19 and 29 (mean age 22.9 years) took part in the experiment. None of them suffered from neurological or psychiatric diseases (self-reported). All had normal or corrected-to-normal vision. One person was left-handed; the others were right-handed (self-reported). They received partial course credit or were paid for participation. Participants were neither informed about the presentation of subliminal primes nor about the study’s specific hypotheses until the end of the Experiment. After the Experiment all participants gave their informed consent prior to their inclusion in the study.

All participants had at least 20 artifact-free segments per condition after the artifact rejection procedure. Therefore, all participants were included in the EEG-data analyses.

(2) Apparatus and stimulus materials

Participants were seated in a dimly illuminated and sound attenuated room. An IBM-compatible computer (Pentium IV with 2.4 GHz) with a 17 inch VGA-Display (75 Hz) and the software E-Prime (Schneider, Eschman, & Zuccolotto, 2002) served for stimulus presentation and data acquisition. The viewing distance was 60 cm. Participants executed their responses on the keys “1” and “3” of the numeric pad of a standard keyboard.

Stimuli were presented in blue on a white background. Figure 7 shows an illustration of the stimulus set. The prime was 18 mm x 40 mm in size and could be left-pointing, right-pointing, neutral or a NoGo-symbol. While the neutral prime was a rectangle, the NoGo-symbol resulted from an overlap of a left-pointing arrow and a right-pointing arrow. The targets were enlarged left-pointing, right-pointing, or NoGo primes with a size of 41 mm x 91 mm (target to prime ratio of 2.28). The primes fitted exactly into the cutting in the middle of the targets.

(3) Procedure

Participants were instructed to respond to the direction of the Go target with their right and left index finger, respectively. For a NoGo target, they had to withhold their response. They were not told that there was any prime before target onset.

Figure 7 shows the four types of priming. The possible combinations of the four primes (left-pointing, right-pointing, neutral or a NoGo-symbol) and the three targets (left-pointing, right-pointing, or a NoGo-symbol) resulted in four types of Go trials [Go prime and Go target pointing in the same direction (Congruent_Go), Go targets with the neutral prime (Neutral_Go), Go targets with the NoGo-prime (NoGo_Go), and Go prime and Go target pointing in the opposite direction

(Incongruent_Go)] and in three types of NoGo trials[12] [NoGo targets with NoGo-prime (Congruent_NoGo), NoGo targets with the neutral prime (Neutral_NoGo), and NoGo targets with left-or right pointing Go prime (Go_NoGo)].

Each trial started with the presentation of a prime stimulus for 13 ms which was followed by a 40-ms blank interval. The duration of prime and blank interval was chosen according to the duration of the retrace rate of the monitor (13.3 ms). Then the target was presented for 200 ms. The participants had to respond with their left or right index finger within the subsequent 800 ms; otherwise the German word for "faster" appeared for 1500 ms on the monitor. Response times were recorded from target onset to response onset. The next trial began 2000 ms after target offset.

Participants performed six blocks of 120 trials each, in a random order. Each of the 12 types of trials was presented 10 times in each block in the course of the entire experiment.

Following these experimental trials, the participants performed a prime-discrimination task which consisted of 108 trials. Half of the trials contained the neutral prime, the other half included the other types of primes, with each type of prime appearing equally often. Participants were informed about the presence and possible types of primes. They were instructed to indicate which of the four primes they had seen by pressing one of four keys on the numeric pad (they had to press "1" for the left pointing arrow, "2" for the NoGo symbol, "0" for the neutral sign, and "3" for the right-pointing arrow).

(4) EEG-Recordings and data analysis

Electrophysiological recordings and data analyses were identical to Experiment 1.

3.2.2 Results

(1) Visibility

Participant's mean discrimination performance (d') was .20 (mean hit rate was 51, 5%, false alarms rate 44.1%) and deviated significantly from zero, $t(15) = 3.44$, $p <$

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[12] There were only three types of NoGo trials, because the NoGo trials with left-and right-pointing Go primes were combined in a single category Go_NoGo.

.01. However, d’ did not correlate with the amount of the greatest priming effect (difference in RT between congruent Go condition and incongruent Go condition), $r = .15$, $p = .58$.

(2) *Behavioral data*

Mean RTs for the Go conditions and error rates for the Go and the NoGo conditions are listed in Table 4. For the RTs of correct Go trials, an ANOVA with repeated measures on the factor type of priming (congruent, neutral, NoGo, and incongruent) revealed a significant main effect for the factor type of priming, $F(3, 45) = 146.82$, $p < .001$, $\eta^2 = .91$.

Table 4. Mean RTs of the four Go conditions and error rates for Go and NoGo condition.

Condition	Mean RT (SD)	Error rates (SD)
Congruent_Go	366 ms (39 ms)	0.7 % (0.6%)
Neutral_Go	393 ms (39 ms)	0.9% (1.0%)
NoGo_Go	405 ms (39 ms)	0.7% (1.3%)
Incongruent_Go	422 ms (34 ms)	6.0% (5.9%)
Congruent_NoGo		0.4% (1.0 %)
Neutral_NoGo		4.4% (6.0%)
Go_NoGo		5.8% (6.8%)

Note. RT = reaction time, SD = standard deviation

RTs in the congruent Go condition were faster than those in the neutral Go condition, $t(15) = 16.93$, $p < .001$, which in turn were shorter than those in the NoGo_Go condition, $t(15) = 6.04$, $p < .001$. RTs were slowest in the incongruent Go condition compared to all other Go conditions, all ps < .001.

For the error rates in the Go conditions, an ANOVA with repeated measures on the factor type of priming (congruent, neutral, NoGo, and incongruent) was conducted. The factor had a significant effect, $F(3, 45) = 12.76$, $p < .01$,. $\eta^2 = .46$.

While the error rates in the congruent, neutral, and NoGo_Go condition did not differ significantly, all *p*s > .36, participants committed the most errors in the incongruent Go condition, all *p*s < .01.

Also, the errors in the NoGo conditions were entered into an ANOVA with repeated measures on the factor type of priming (congruent, neutral, and Go), which revealed a significant effect, *F*(2, 30) = 9.46, *p* < .01,. η^2 = .39.

The error rate in the congruent NoGo condition was smaller than that in the neutral NoGo condition, *t*(15) = 2.76, *p* < .05, which in turn was smaller than that in the Go_NoGo condition, *t*(15) = 2.55, *p* < .05.

(3) ERP-data

Since there were four Go conditions but only three NoGo conditions, the analysis of Experiment 2 concentrated on the priming effects in the four Go and the three NoGo conditions separately. However, the topography differences between Go and NoGo conditions are well known and were already replicated in Experiment 1.

The main interest of this study lies in the P300-amplitude. Therefore, the statistical analyses were limited to the segments in the P300 time frame of the target (350 - 650 after prime onset).

Figure 8 shows the ERPs at the Cz- and the Pz-electrode for the four Go-conditions. Since the ERPs start with prime onset, the onset of the target is marked by an arrow.

For the Cz-electrode data, an ANOVA was conducted with repeated measures on the factors type of priming (congruent, neutral, NoGo, and incongruent) and time (50 ms-segments spanning the period from 350-650 ms after prime onset).

The ANOVA revealed that the significant main factors type of priming, *F*(3, 45) = 16.37, *p* < .001, η^2 = .52, and time, *F*(5, 75) = 24.87, *p* < .001, η^2 = .62, interacted significantly, *F*(15, 225) = 9.87, *p* < .001, η^2 = .40.

The ANOVAs for the single segments with repeated measures on the factor type of priming (congruent, neutral, NoGo, and incongruent) showed a significant main effect of the factor prime in the segments from 350-550 ms after prime onset, all *p*s < 05, η^2s ≥ .20.

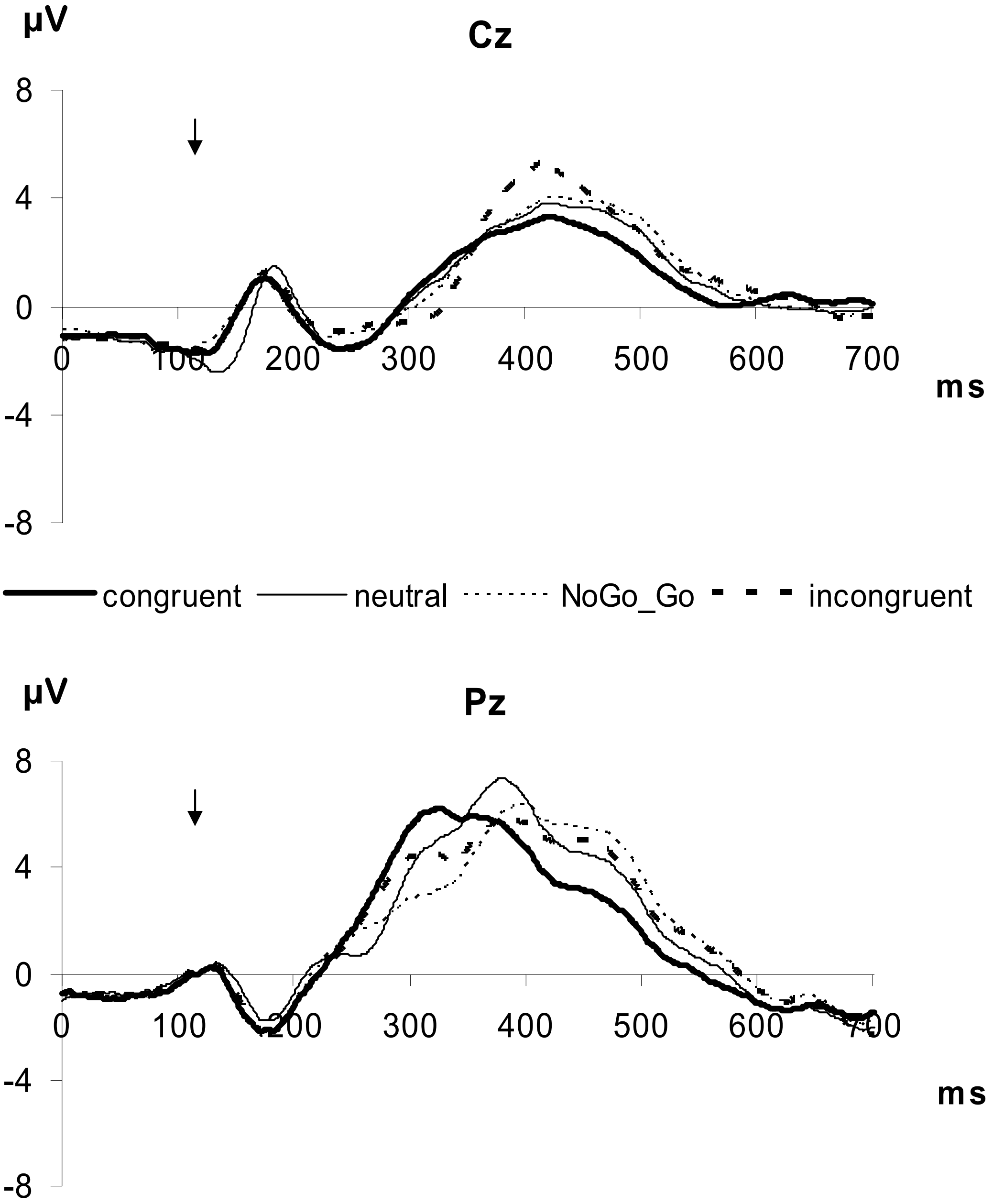

Figure 8. Grand-averaged event-related potentials over 16 participants. Comparisons between the four Go conditions (congruent, neutral, NoGo_Go, and incongruent). ERPs are shown for the Cz- (above) and the Pz-electrode (below). The arrow indicates target onset.

Further analyses revealed that the amplitude in the congruent Go condition was the smallest compared to that in all other Go conditions in the segment 400-450 ms, all *p*s < .05, and in the segments 500-600 ms, all *p*s < .05. Only in the segment 450-500 ms, the amplitude in the congruent Go condition was significantly enhanced compared to that in the neutral Go condition, $t(15) = 3.95$, $p < .01$. In the segments 450-500 ms, $t(15) = 7.79$, $p < .001$, and 550-600 ms, $t(15) = 2.52$, $p < .05$, the amplitude in the neutral Go condition was smaller than the amplitude in NoGo_Go, which in turn was smaller compared to that in the incongruent Go condition in the segments 350-450 ms, all *p*s < .01. The amplitude in the incongruent Go condition was also significantly enhanced compared to that in the neutral Go condition in the segments 350-550 ms, all *p*s < .05.

For the Pz-electrode data, an ANOVA was conducted with repeated measures on the factors type of priming (congruent, neutral, NoGo, and incongruent) and time (50 ms-segments spanning the period from 350-650 ms after prime onset).

The ANOVA revealed a significant main effect for the factors type of priming, $F(3, 45) = 16.58$, $p < .001$, $\eta^2 = .53$, and time, $F(5, 75) = 81.13$, $p < .001$, $\eta^2 = .84$, and a significant interaction between the two factors, $F(15, 225) = 13.60$, $p < .001$, $\eta^2 = .48$.

The ANOVAs for the single segments with repeated measures on the factor type of priming (congruent, neutral, NoGo, and incongruent) showed a significant main effect of type of priming in the segments from 350-550 ms (*p*s < 001, η^2s > .30) after prime onset.

Further analyses revealed that the amplitude in the congruent Go condition was the smallest compared to that in all other Go conditions in the segments 400-600 ms, all *p*s < .05. In the segment 350-400 ms, the amplitude in the neutral Go condition was significantly enhanced compared to that in all other Go conditions, all *p*s < .001. The amplitude in the NoGo_Go condition was enhanced compared to that in the neutral Go condition in the segments 450-600 ms, all *p*s < .05, and it was enhanced compared to that in the incongruent Go condition in the segments 450-500 ms, all *p*s < .05.

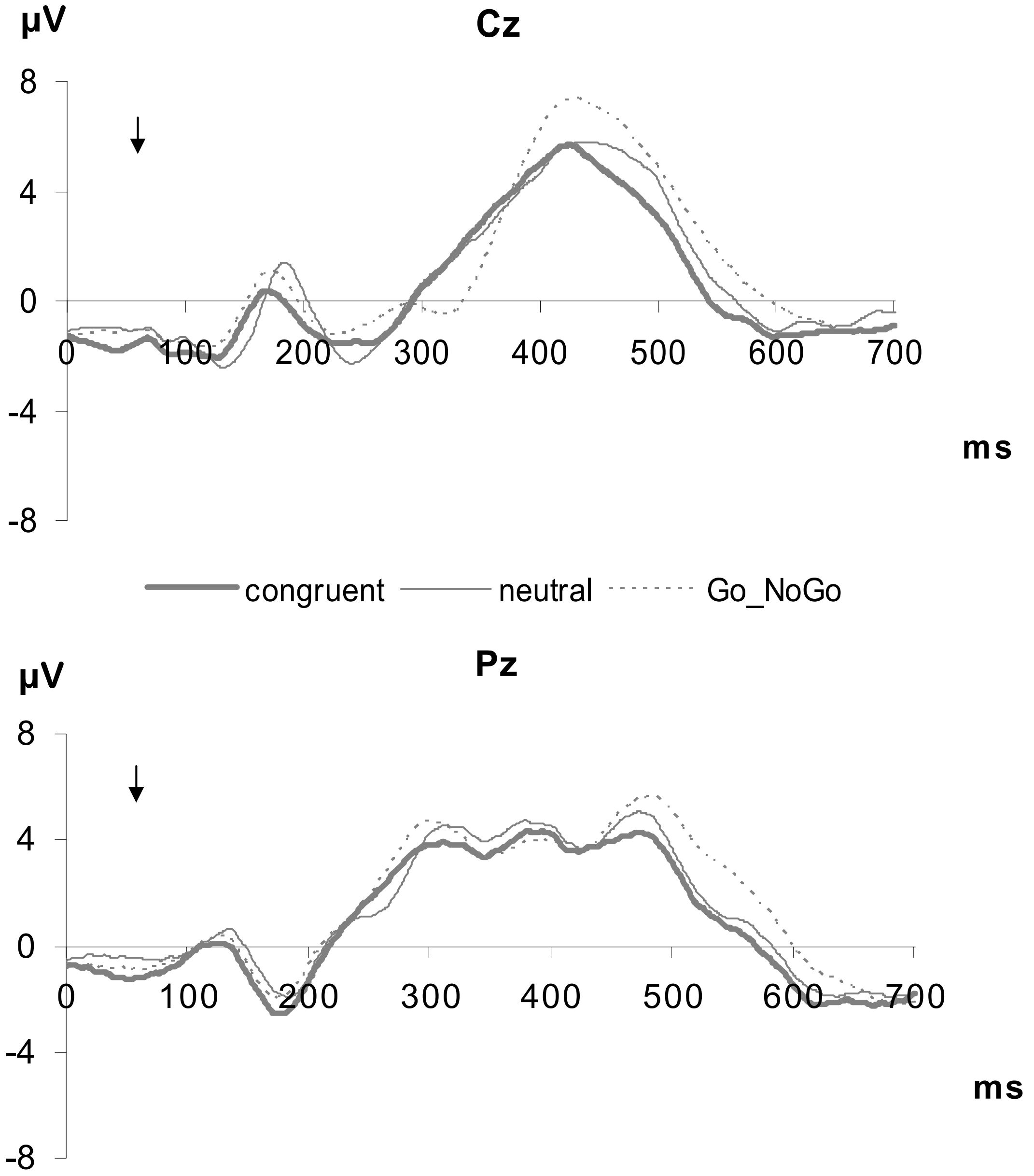

Figure 9. Grand-averaged event-related potentials (ERPs) over 16 participants. Comparisons between the three NoGo conditions (congruent, neutral, and Go_NoGo). ERPs are shown for the Cz- (above) and the Pz-electrode (below). The arrow indicates target onset.

Figure 9 shows the ERPs at the Cz- and the Pz-electrode for the three NoGo conditions.

For the Cz-electrode data, an ANOVA was conducted with repeated measures on the factors type of priming (congruent, neutral, and Go) and time (50 ms-segments spanning the period from 350-650 ms after prime onset).

The ANOVA showed that the significant main effects of type of priming, $F(2, 30) = 39.48$, $p < .001$, $\eta^2 = .73$, and time, $F(5, 75) = 48.77$, $p < .001$, $\eta^2 = .77$, interacted significantly, $F(10, 150) = 6.409$, $p < .001$, $\eta^2 = .29$.

The ANOVAs for the single segments with repeated measures on the factor type of priming (congruent, neutral, and Go) showed a significant main effect of type of priming in the segments from 400–650 ms after prime onset, ps < 05, η^2s > .20.

Further analyses revealed that the amplitude in the Go_NoGo condition was significantly enhanced compared to that in all other NoGo conditions in the segments 400-600 ms, all ps < .05. In the segment 600-650 ms, it was only significantly enhanced compared to that in the congruent NoGo condition, $t(15) = 2.67$, $p < .05$. The amplitude in the neutral NoGo condition was enhanced compared to the congruent NoGo condition in the segments 450-600 ms, all ps < .05.

For the Pz-electrode data, the ANOVA with repeated measures on the factors type of priming (congruent, neutral, and Go) and time (50 ms-segments spanning the period from 350-650 ms after prime onset) revealed that the significant main effects of type of priming, $F(2, 30) = 9.25$, $p <.01$, $\eta^2 = .38$, and time, $F(5, 75) = 64.67$, $p < .001$, $\eta^2 = .81$, interacted significantly, $F(10, 150) = 9.98$, $p < .001$, $\eta^2 = .40$.

The ANOVAs for the single segments with repeated measures on the factor type of priming (congruent, neutral, and Go) showed a significant main effect of type of priming in the segment from 350-400 ms, $F(2, 30) = 3.65$, $p < .05$, $\eta^2 = .20$, and in the segments from 450-650 ms (all ps < 01, η^2s > .25) after prime onset.

Further analyses revealed that the amplitude in the Go_NoGo condition was significantly enhanced compared to that in the congruent NoGo condition in the segment 450-500, $t(15) = 3.35$, $p < .01$, and it was enhanced compared to that in the congruent and the neutral NoGo condition in the segments 500-650 ms, all ps < .05. The amplitude in the neutral NoGo condition was enhanced compared to that in the Go_NoGo condition in the segment 350-400, $t(15) = 3.37$, $p < .01$, and it was enhanced compared to that in the congruent NoGo condition in the segment 450-500 ms, $t(15) = 2.18, p < .05$.

(4) Topographical mappings

Figure 10 shows the scalp topographic voltage maps of the four Go conditions at the global field power peak in the P300 time frame. Graphical inspection suggests that the centroids are located more anteriorly in the NoGo_Go and the incongruent Go condition than in the other conditions.

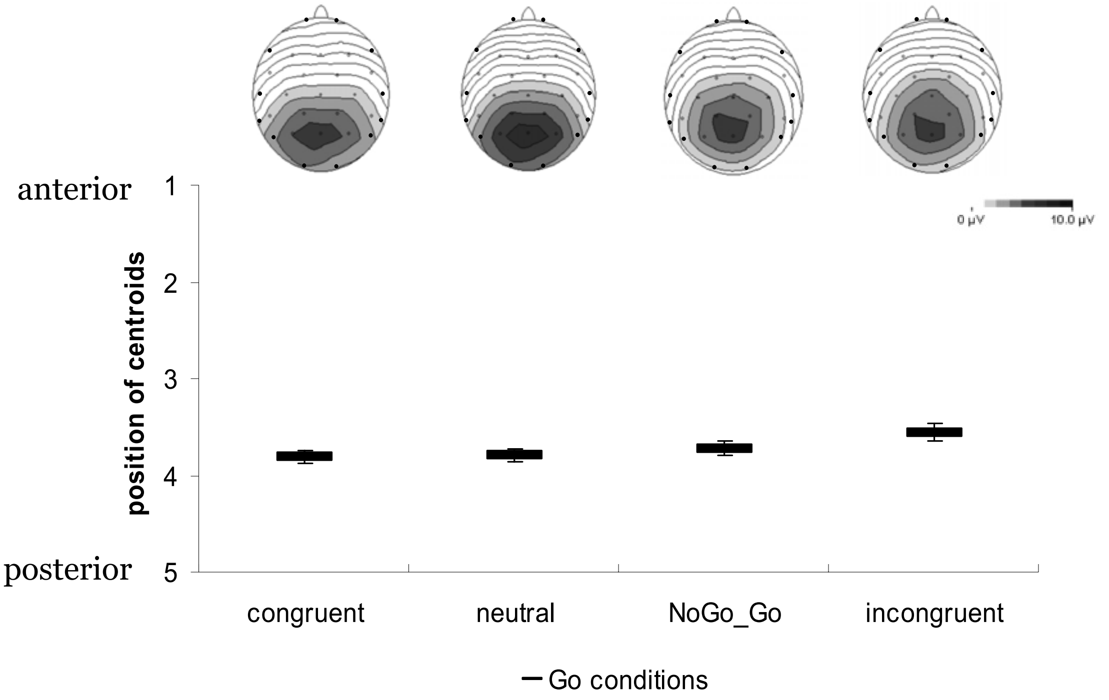

Figure 10. Positions of the centroids of the Go and NoGo conditions at the peak of the global field power in the P300 time frame and corresponding scalp topographic voltage maps (upper panel).

An ANOVA with repeated measures on the factor type of priming (congruent, neutral, NoGo, and incongruent) revealed a significant main effect of type of priming, $F(3, 45) = 11.52, p < .001, \eta^2 = .43$.

Planned t-tests showed, that the centroid of the NoGo_Go condition was indeed located more anteriorly than that of the congruent and the neutral Go conditions, ps =.05 (one-tailed). Also the centroid of the incongruent Go condition was located significantly more anteriorly than the centroids of the other Go conditions, ps < .001 (one-tailed).

The scalp topographic voltage maps of the three NoGo conditions at the global field power peak in the P300 time frame are presented in Figure 11. Graphical inspection suggests that the centroids of the Go_NoGo condition are located more anteriorly than the centroid of the neutral NoGo condition, which in turn seems to be more anterior than that of the congruent NoGo condition.

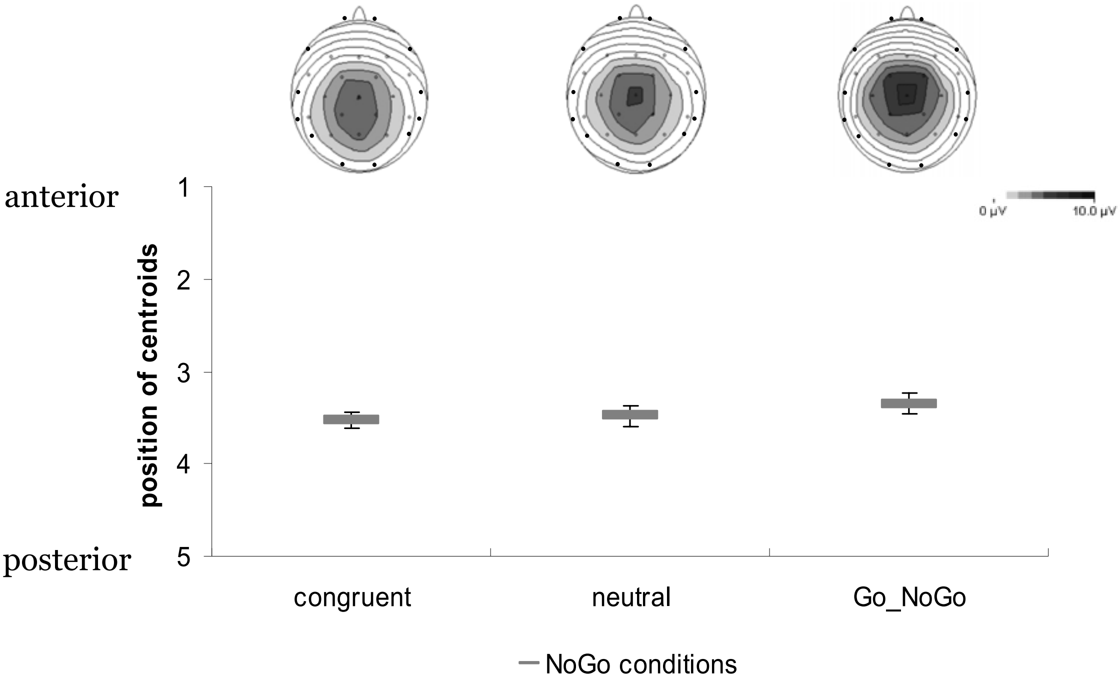

Figure 11. Positions of the centroids of the Go and NoGo conditions at the peak of the global field power in the P300 time frame and corresponding scalp topographic voltage maps (upper panel).

The within-subject-factor type of priming (congruent, neutral, and Go) just missed the conventional level of significance, $F(2, 30) = 3.20$, $p = .06$, $\eta^2 = .18$.

Planned contrasts showed that the centroid of the Go_NoGo condition was located more anteriorly than those of the congruent and the neutral NoGo condition, all ps < .05 (one-tailed). Congruent_NoGo and Neutral_NoGo did not differ significantly in topography, $t(15) = 0.57$, $p = .29$.

3.2.3 Discussion

In Experiment 2, the introduction of a second response alternative and the application of a somehow more natural set of stimuli (arrows) served to increase the small priming effects of Experiment 1. Unlike Experiment 1, the presentation of the primes preceding Go and NoGo targets was not subliminal in Experiment 2, since the mean discrimination performance (d’) for the primes did deviate significantly from zero. However, the visibility of the primes was not related to the size of the priming effects, which was proved by a non-significant correlation[13].

The trend apparent in the Go RTs in Experiment 1 is replicated in Experiment 2. Go priming in the congruent Go condition speeded up response times, while NoGo priming in the NoGo_Go condition slowed down the response time down compared to that of the neutral Go condition. To the best of my knowledge, this is the first time that NoGo priming has been demonstrated. This finding is well in line with the findings of Kramer, Humphrey, Larish, Logan, and Strayer (cited in Logan, 1994). They found that the RTs in trials after successfully inhibited trials were slower than that after Go trials. It seems that the process triggered by the NoGo signal has some aftereffects. Moreover, De Jong, Coles, and Logan (1995) also found that presenting a NoGo signal had an effect on reaction time. They investigated trials in which a NoGo signal was only relevant for responses with one hand. For responses with the other hand (Go trials), the NoGo signal had no meaning. The presentation of the NoGo signal slowed down RTs in Go trials although the NoGo signal had no meaning for those trials. De Jong et al. assumed that the processing of the NoGo signal interfered with the Go task.

However, the incongruent Go condition had the slowest Go RTs in Experiment 2[14]. The slow RTs in the incongruent Go condition could indicate that

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[13] The size of the priming effect was calculated as the difference between the congruent and the incongruent Go-RTs as this is the largest difference in RTs.

[14] There might be an alternative explanation for the observed results. Perhaps, participants interpreted the double-arrow half of the time as right-pointing, and half of the time as left-pointing arrow. According to this, one half of the NoGo_Go trials would have been congruent trials and the remaining half would have been incongruent trials. In this perspective, the RTs of the NoGo_Go condition would be expected to be between the congruent and the incongruent Go RTs, which is exactly what was found. However, this alternative account could not explain the trend in Experiment 1. Moreover, the error rates in the Go and NoGo conditions did not support this thesis

the inhibition of the wrong response had to take place before the right response could be performed.

The Cz-amplitudes in the four Go conditions in the P300 time frame of the target mirrored the results for the Go RTs. When RTs in a condition were fast, the Cz-amplitude was small and vice versa. The results for the Pz-amplitudes seem to reflect the amount of information contained in the primes about the required response. The Pz-amplitude was small, when the prime was a good predictor of the required response as in the Congruent_Go condition. However, the Pz-amplitude was increased, when the prime was not a good predictor of the required response as in the NoGo_Go and in the incongruent Go condition.

The Cz-amplitudes in the three NoGo conditions in the P300 time frame of the target replicated the findings of Experiment 1. The Cz-amplitude in the Go_NoGo condition was increased compared to that in the neutral and the congruent NoGo condition.

The centroid of the NoGo primed Go condition was located more anteriorly than those of the congruent or the neutral Go condition. These results clearly support the assumption of a central process that is triggered by a NoGo signal.

The centroid of the incongruent Go condition was located more anteriorly than those of the congruent and the neutral Go condition. Thus, similar processes seemed to be at work during the NoGo primed Go condition and the incongruent Go condition. This is well in line with the findings of Kopp, Rist, and Mattler (1996). Kopp et al. investigated the influence of incongruent flankers within a flanker-task on the P300-amplitude. In fact, they found a more anteriorly located P300-maximum in the condition with incongruent flankers compared to that in the condition with neutral flankers (for similar results, see Kopp, Mattler, Goertz, and Rist, 1996).

Like in Experiment 1, the centroid of the Go primed NoGo condition was not located more posterior than that of the congruent or the neutral NoGo condition. In fact, it was located more anterior than that of the congruent and the neutral NoGo condition. Therefore, the assumption that motor activation could erase frontal positivity in Go trials is rejected.

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either. If the alternative explanation held true, there should be more errors in the NoGo primed conditions than in the neutrally primed conditions. This is clearly not the case.

Taken together, Experiment 2 showed that the NGA could not be explained by central negative motor potentials in the Go conditions. On the contrary, the presentation of a NoGo signal triggers a central process that leads to a greater, central positivity in the NoGo conditions.

3.3 Conclusion

Experiment 1 and Experiment 2 showed that the phenomenon of the NoGo-Anteriorization (NGA) is not an artefact of negative motor potentials. Moreover, Experiment 2 showed that there is in fact a central mechanism triggered by a NoGo stimulus. Now, questions arise concerning the nature of this central process. De Jong, Coles, Logan, and Gratton (1990) assumed that this central process reflects a central inhibitory mechanism (see section 1.4.1). However, the findings of Fallgatter, Bartsch, and Herrmann (2002) suggested that the central process could be the monitoring of conflict and or the monitoring of mental effort (see section 2.1.4). In the following this alternative will be called the conflict assumption. Figure 12 shows illustrations of the inhibition (A) and the conflict assumption (B). Under the inhibition assumption the NGA is connected to the inhibitory process. The NGA in the incongruent Go condition is due to the inhibition of the wrong response. Under the conflict assumption, the NGA reflects conflict. The conflict is due to the coexistence of two competing processes, for example a response and an inhibitory process. The conflict emerges because only one process can determine performance. The arising conflict is described by the double arrow in Figure 12. The conflict assumption also predicts an NGA in the incongruent Go condition in which a conflict would occur between two response processes.

Unfortunately, neither Experiment 1 nor Experiment 2 can answer whether the NoGo-Anteriorization is connected to inhibition or conflict. The inhibition assumption as well as the conflict assumption would predict the same topographies. When a huge amount of inhibition is necessary there is also a huge amount of conflict. When there is only little conflict there is also only little need for inhibition. For example, the centroid of the congruent NoGo condition in Experiment 1 was not located more anterior than that of the Go conditions. According to the conflict assumption this should be the case because there is no conflict in the congruent NoGo condition. On the other hand, the inhibition assumption would also predict a very weak NGA in the congruent NoGo condition. The NoGo prime has already inhibited any response preparation. Therefore, there should be only little if any additional inhibition.

Therefore, Experiment 3 was conducted to investigate whether the NoGo-Anteriorization reflects conflict on a response level or the central inhibitory mechanism.

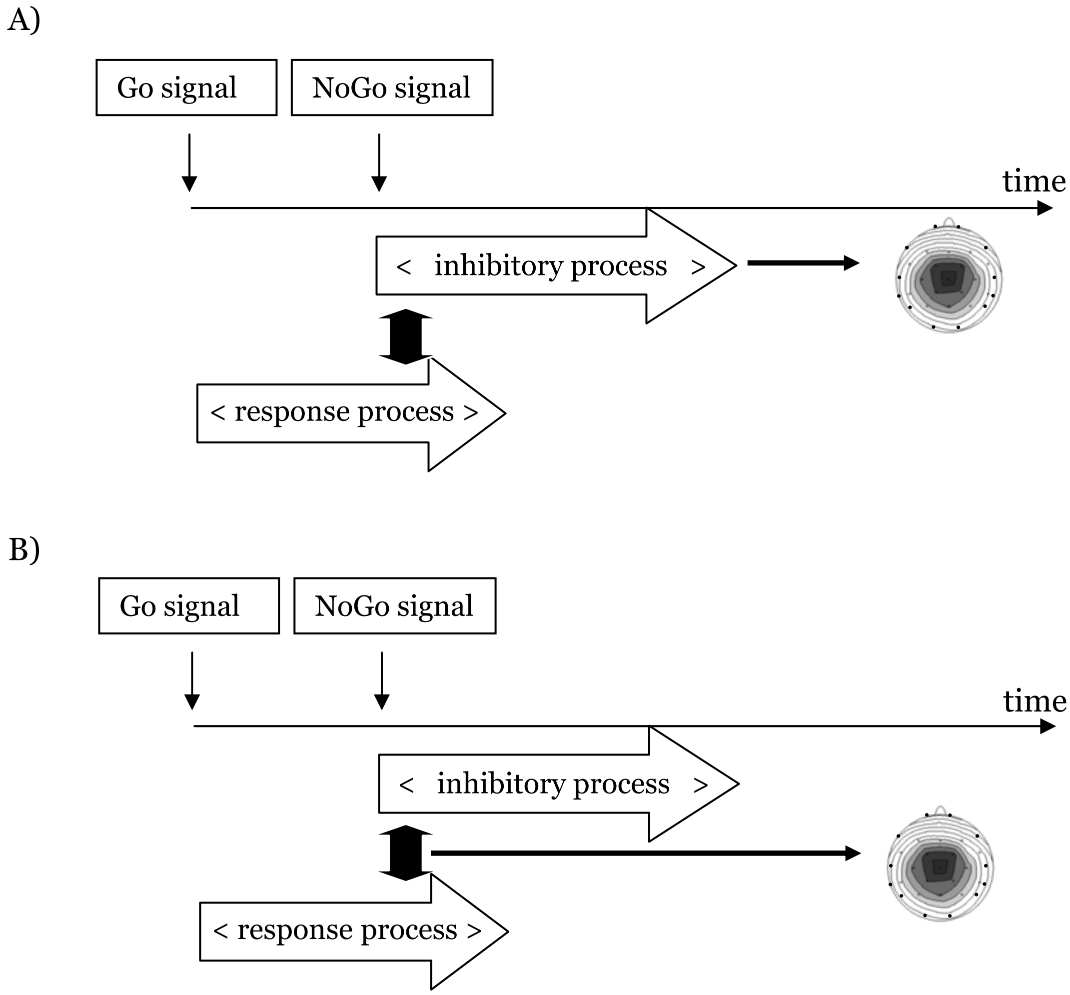


Figure 12. Illustration of the inhibition assumption (A) and the conflict assumption (B).

4 Does NoGo-Anteriorization represent response conflict?

The results of Experiment 1 and those of Experiment 2 are well in line with the conflict assumption as well as with the inhibition assumption. All conditions that contained an inhibitory process also contained conflict and vice versa. Experiment 3 was designed to test if the NGA is the result of an inhibitory process or the occurrence of conflict. Therefore, Go and NoGo conditions were compared that contained the same kind of conflict. If the topographic difference between the Go and the NoGo condition resulted from more conflict in NoGo conditions, the NGA should disappear when comparing conflict-laden Go and NoGo conditions. Conflict in this case means conflict arising as a result of the simultaneous occurrence of two competing processes (response and inhibitory process). Figure 13 depicts how the same kind of conflict was created in Go (B) and in NoGo conditions (A). In each condition two signals are presented sequentially. The first signal triggers a process. For instance, in the NoGo condition with high conflict (A) the first signal triggers a response process, whereas in the Go condition with high conflict (B) the first signal triggers an inhibitory process. After a SOA of 100 ms the first signal is replaced by the second signal. In the NoGo condition (A) the second signal triggers an inhibitory process whereas in the Go condition (B) the second signal triggers a response process. Unlike in the Go_NoGo and the NoGo_Go condition of Experiment 1 and Experiment 2 both signals are presented supraliminal in Experiment 3. The supraliminal presentation generates a reasonable P300 for both stimuli (first P300 after the first signal, second P300 after the second signal).

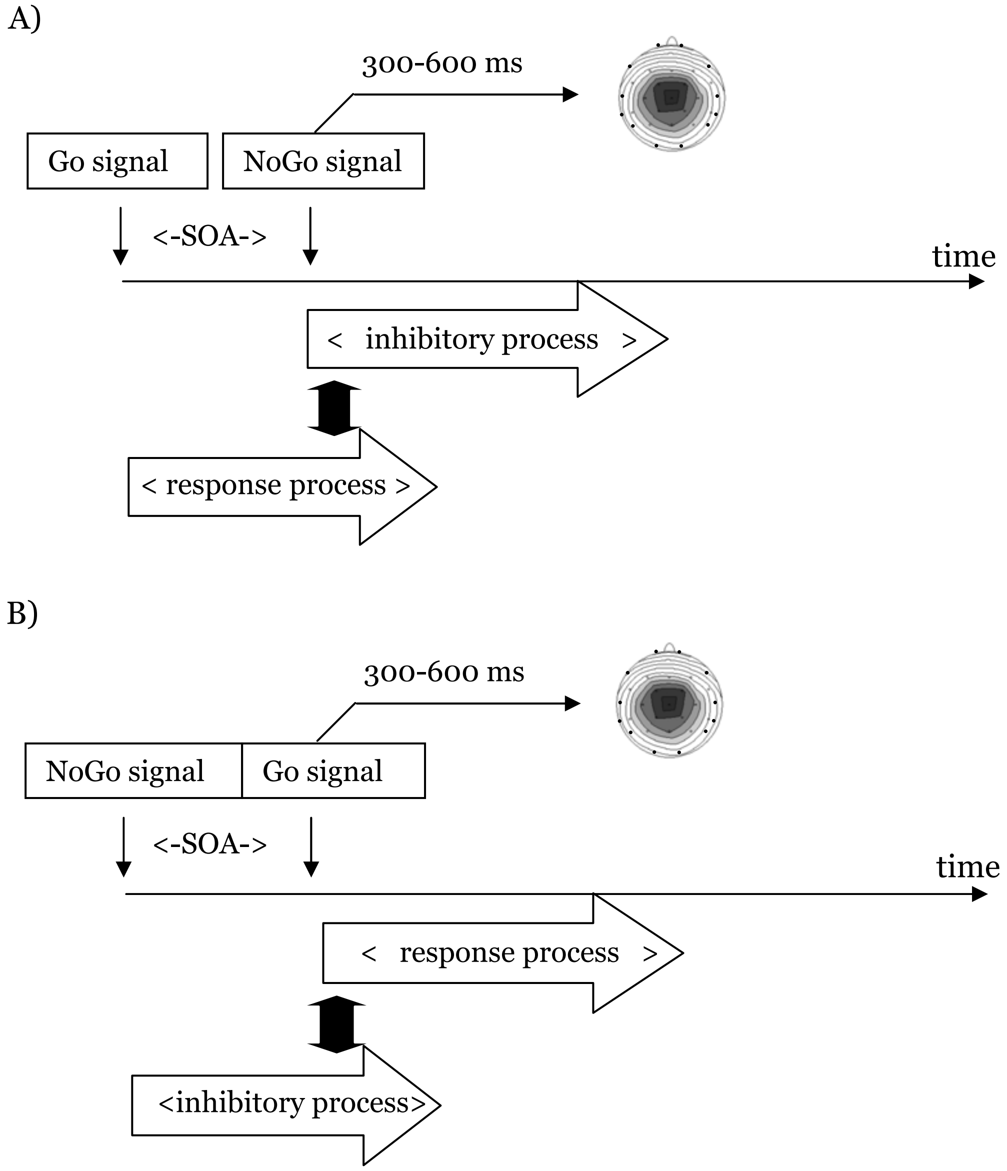
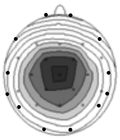
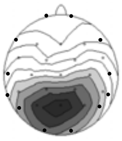
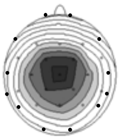
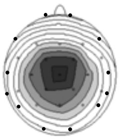
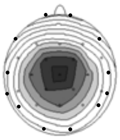
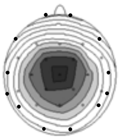


Figure 13. The NoGo-Anteriorization as a result of response conflict in NoGo (A) and Go conditions (B) that contain the same kind of conflict.

The conflict in the Go and the NoGo condition arises with the presentation of the second signal, because then two rivaling processes are simultaneously active.

Table 5 summarizes the expected topographies in case the NGA is connected to response conflict (right side) or in case it is connected the inhibitory mechanism (left side).

Table 5. Summary of the expected topographies under the hypothesis that the NGA is a correlate of response conflict or that it is a correlate of the inhibitory mechanism.

NGA = correlate of response conflict		NGA = correlate of the inhibitory mechanism	
First signal	Second signal	First signal	Second signal
			
Go______NoGo		Go______NoGo	
			
NoGo______Go		NoGo______Go	

If the NGA is connected to conflict on a response level, the P300 after the first Go signal in the Go_NoGo condition should resemble the typical Go-P300 (top left). The P300 after the following NoGo signal should resemble a NoGo-P300, because the response process which was triggered by the first Go signal is now simultaneously active with the inhibitory mechanism. In the NoGo_Go condition, the P300 after the first NoGo signal should resemble a NoGo-P300, because there is a conflict between prepotent tendencies to respond as fast as possible and the inhibitory mechanism, which is triggered by the NoGo signal (lower left). The P300 after the following Go signal should have the topography of the NoGo-P300, because the response process triggered by the Go signal conflicts with the inhibitory mechanism which is still active. Accordingly, the NGA should disappear in Go and NoGo conditions that contain the same kind of conflict (see topographies after the second signal on the left side of Table 5).

However, if the NGA is connected to the inhibitory process, only P300s after a NoGo signal should have the topography of the NoGo-P300 (right side of Table 5).

Of course the duration of the interval between the first and the second signal is crucial because a conflict between an inhibitory and a response process only occurs when both processes overlap in time. Therefore, it had to be assured that the inhibitory process in the NoGo_Go condition is still at work when the Go signal triggers the response process. Since Logan (1994) stated that an inhibitory process in a normal adult approximately lasts 200-250 ms, the Go signal was presented 100 ms after the onset of the NoGo signal. Under these conditions, the inhibitory and the response process should be concurrently active.

The Go-NoGo task implemented for this experiment required four different symbols (circle, square, rhomb, or drop) that changed their color from green to red or the other way around in fifty percent of the trials (see also Figure 14). The participants' task was to press a key every time a circle appeared, no matter whether its color changed (Go_gr) or not (Go_gg). The square required to withhold any key press, irrespective of occurring color-changes (NoGo_rr and NoGo_rg). For the rhomb symbol the participants were instructed to press the key as soon as the color changed (NoGo_Go) but to withhold the key press as long as the color remained unchanged (NoGo_ambi). For the drop symbol they should withhold their response in case of a color-change (Go_NoGo) but press the key when the drop remained green (Go_ambi).

This rather complicated setting served the purpose of comparing NoGo_Go and Go_NoGo conditions with a Go and a NoGo condition that had the same color-change (Go_gr and NoGo_rg). To be sure, that a rapid color-change by itself does not alter the main characteristics of the Go- and the NoGo-P300 respectively, these conditions were compared to a Go and a NoGo condition without color-change (Go_gg and NoGo_rr).

4.1 Method

4.1.1 Participants

A total of 12 women and 8 men aged between 19 and 48 years (mean age 25.5 years) took part in the experiment. None of them suffered from neurological or psychiatric diseases (self-reported). All had normal or corrected-to-normal vision.

Two persons were left-handed; the others were right-handed (self-reported). They received partial course credit or were paid for participation. All participants gave their informed consent prior to their inclusion in the study. They were not informed about the study's specific hypotheses.

Five participants had to be excluded from the EEG data set because there remained less than 20 artifact-free segments per condition for each one of them after the artifact rejection procedure. Therefore, the EEG-data set consisted of 9 women and 6 men aged between 19 and 29 years (mean age 23.5 years). All but one of those remaining participants were right-handed.

4.1.2 Apparatus and stimulus materials

Participants performed the experiment in a dimly illuminated and sound attenuated room. An IBM-compatible computer (Pentium IV with 1.8 GHz) with a 17 Inch VGA-Display (85 Hz) and the software E-Prime (Schneider, Eschman, & Zuccolotto, 2002) served for stimulus presentation and data acquisition. The viewing distance was 60 cm. Responses were executed on the space-bar of a standard keyboard.

Targets were presented in green or red on a black background. Figure 14 shows an illustration of the stimuli. As targets served the four symbols (circle, square, rhomb or drop), which represent the lowercase letters l, n, t, and the uppercase letter S in the wingdings font (size: 60 points). In half of the trials the color of a symbol remained unchanged whereas in the other half the color changed after 100 ms. The circle was presented in green and remained green (Go_gg) or changed to red (Go_gr), the drop was presented in green and remained green (Go_ambi) or changed to red (Go_NoGo), the rhomb was presented in red and remained red (NoGo_ambi) or changed to green (NoGo_Go) and the square was presented in red and remained red (NoGo_rr) or changed to green (NoGo_rg). In addition to the targets a white fixation cross was used in the Arial font (size: 30 points).

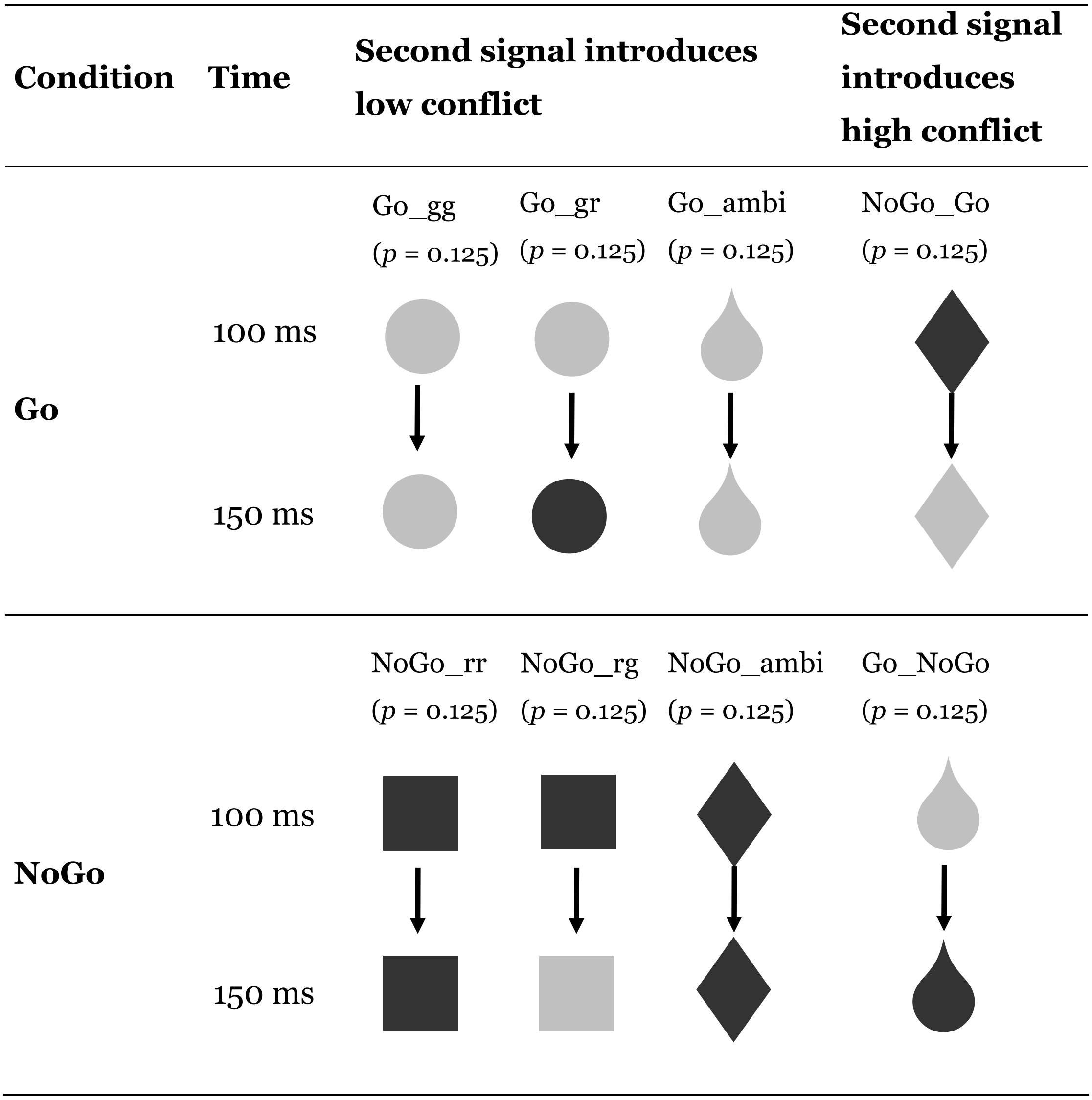


Figure 14. Illustration of the stimuli (lighter gray symbolizes green, darker gray symbolizes red).

4.1.3 Procedure

Participants were instructed to press the space-bar with their right index finger every time a circle appeared, no matter if the color changed (Go_gr) or remained (Go_gg). If they saw a square they should never press the key (NoGo_rr and NoGo_rg). For the rhomb they were to press the key when the color changed (NoGo_Go) but not press it when the color remained (NoGo_ambi). When the drop was presented they should withhold their response in the case of a color-change (Go_NoGo) but press the key when the drop remained green (Go_ambi).

Each trial started with the presentation of the fixation cross for 200-ms. Subsequently, the target was presented for 250 ms. Within these 250 ms, the target changed its color in 50% of the trials after 100 ms. Response times were recorded from target onset to response onset. The next trial started after a randomly determined intertrial interval of 600 ms, 1100 ms, or 1600 ms. This randomly chosen intertrial interval served to avoid the development of a habitual response pattern.

Before the experimental blocks, the participants completed a short practice block. During this practice block participants received feedback about the accuracy and the speed of their response. Participants had to respond within a 800 ms time window following target onset; otherwise the German word for “faster” was displayed on the monitor. The practice block contained 48 trials.

During the experimental blocks, no feedback was given in order to avoid any influences of feedback on the EEG. Participants performed three blocks with 160 trials each. Within each block the trials were intermixed randomly. Each of the eight trial types was presented 20 times in each block.

4.1.4 EEG-Recordings and data analysis

Electrophysiological recordings were identical to Experiment 1, except for the following: Since a second P300 was expected to be elicited by the perception of the second signal, a second P300 was searched for the time window 400-700 ms after the presentation of the first signal.

4.2 Results

4.2.1 Behavioral data

Mean RTs for the Go conditions and error rates for Go and NoGo conditions are listed in Table 6. In an ANOVA on the RTs of correct Go trials the within-subjects factor condition (Go_gg, Go_gr, Go_ambi, NoGo_Go) was significant, $F(3, 57) = 179.52$, $p < .001$,. $\eta^2 = .90$.

Table 6. Mean RTs of the four Go conditions and error rates for Go and NoGo conditions.

Condition	Mean RT (SD)	Error rates (SD)
Go_gg	433 ms (64 ms)	0.08 % (0.3%)
Go_gr	510 ms (76 ms)	1.8% (2.9%)
Go_ambi	464 ms (77 ms)	0.08% (0.4%)
NoGo_Go	583 ms (71 ms)	1.3% (1.8%)
NoGo_rr		0.3% (1.5 %)
NoGo rg		13.3% (22.5%)
NoGo_ambi		1.5% (3.0%)
Go_NoGo		24.1% (13.3%)

Note. RT = reaction time, SD = standard deviation

RTs in the Go condition without color change (Go_gg) were shorter than those in the Go_ambi condition, in which the symbol could also instruct a NoGo trial when its color changes, $t(19) = 6.86$, $p < .001$. RTs in the Go_ambi condition were in turn shorter than those in the Go condition with color change (Go_gr), $t(19) = 5.38$, $p < .001$. Participants responded slowest in the NoGo_Go condition compared to all other Go conditions, ps < .001.

Please note that response times were measured from the onset of the first stimulus, so in principle response initiation in the NoGo_Go condition was at least as fast as in the Go_ambi condition as it could only start 100 ms later when the second signal was presented. Interestingly, even after subtracting 100 ms from the NoGo_Go RTs, the mean reaction time in the NoGo_Go condition was still significantly slower than in the simple Go condition without color change, $t(19) = 8.51$, $p < .001$.

Condition was also a significant main factor in the ANOVAs on the errors in Go, $F(3, 57) = 7.66$, $p < .01$,. $\eta^2 = .29$, and in NoGo conditions, $F(3, 57) = 13.87$, $p < .001$,. $\eta^2 = .42$. In Go conditions, participants performed more errors in the Go condition with color change (Go_gr) and in the NoGo_Go condition than in any other Go condition, ps < .001. The error rates in Go_gr and NoGo_Go trials did not differ not significantly from each other, $p >.30$. In NoGo conditions, the highest error rates were found in the NoGo condition with color change (NoGo_rg) and in the Go_NoGo condition, *p*s < .05. Numerically, participants committed more errors in the Go_NoGo condition than in the NoGo_rg condition but statistically this difference just missed the conventional level of significance, $p < .10$.

4.2.2 ERP-data

(1) Effects of color-change

The first analysis investigated the influence of the color-change on the ERP-data due to perceptual overlays. Figure 15 shows the ERPs at Cz and Pz for the Go and NoGo conditions with (Go_gr and NoGo_rg) and without color-change (Go_gg and NoGo_rr). The ERPs start with the onset of the first signal. To facilitate reading of the data, in the following all findings in ERPs are reported relative to the onset of the first signal.

Graphical inspection reveals that Go and NoGo conditions with color-change differ from those without color-change in the P300 time frame at the Cz- and the Pz-electrode.

Since the main interest of this study was in the P300 amplitude, statistical analyses were limited to the segments in the P300 time frames of the first (300-600 ms) and the second stimulus (400-700 ms).

For the Cz-electrode data, an ANOVA with repeated measures on the factors task (Go, NoGo), color-change (with, without), and time (50 ms-segments spanning the period from 300-700 ms after the first stimulus' onset) was conducted for the conditions Go_gg, Go_gr, NoGo_rr, and NoGo_rg.

The ANOVA revealed a significant main effect for time, $F(7, 98) = 10.23$, $p < .001$,. $\eta^2 = .42$. The main effect of task just missed the conventional level of significance, $F(1, 14) = 4.44$, $p < .06$,. $\eta^2 = .24$. Additionally, there were significantly interactions between the factors task and time, $F(7, 98) = 9.18$, $p < .001$,. $\eta^2 = .40$, between color-change and time, $F(7, 98) = 12.36$, $p < .001$,. $\eta^2 = .47$, and between task, color-change, and time, $F(7, 98) = 7.74$, $p < .01$,. $\eta^2 = .29$.

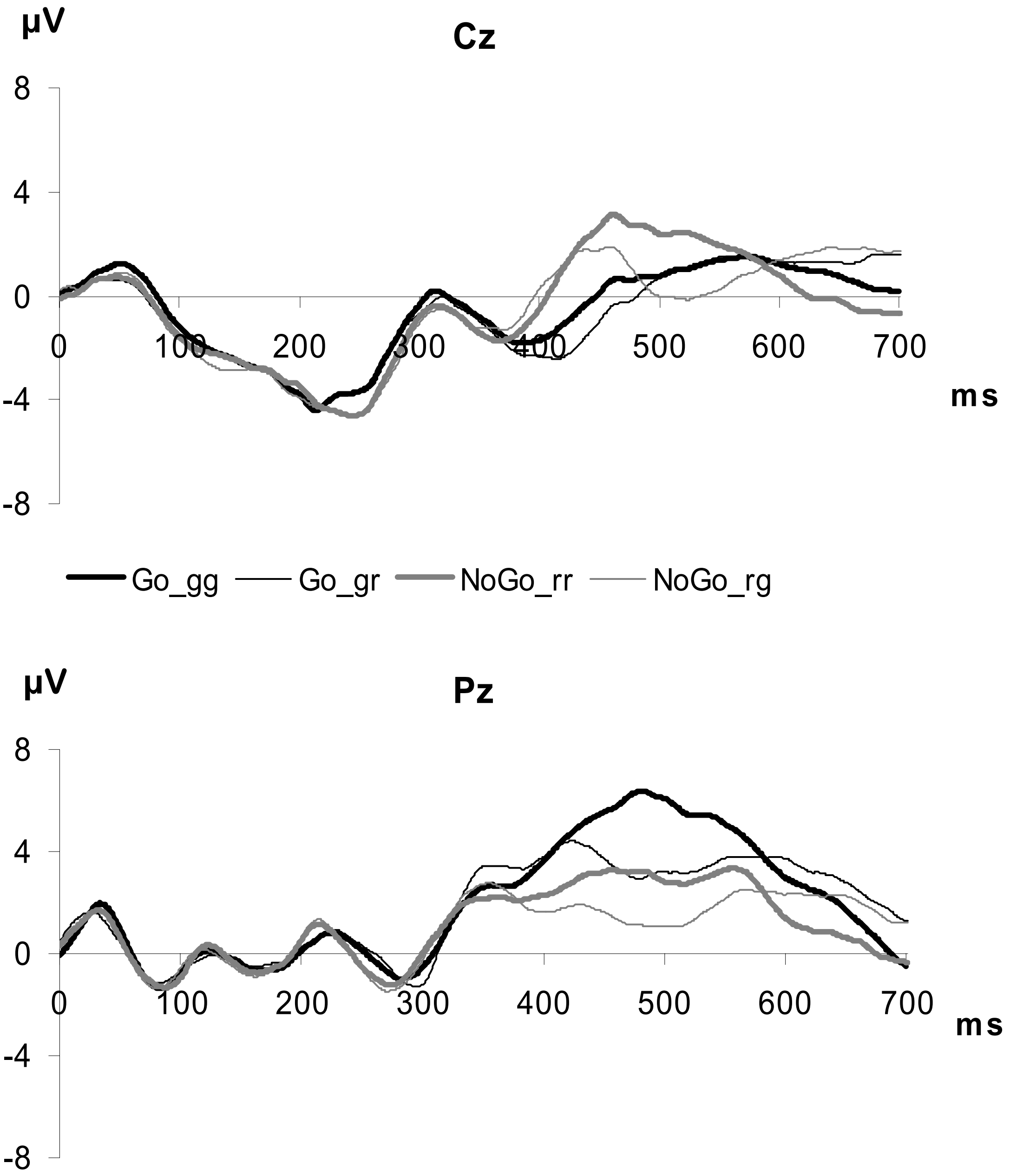


Figure 15. Grand-averaged event-related potentials (ERPs) over 16 participants for both Go conditions (with and without color-change) and for both NoGo conditions (with and without color-change). ERPs are shown for the Cz- (above) and the Pz-electrode (below).

The ANOVAs for the single segments with repeated measures on the factors task (Go, NoGo) and color-change (with or without color-change) showed a significant main effect of task in the segments 400-500 ms after the onset of the first stimulus, all *p*s < .01, η^2s > .53. However, there was also a significant main effect of color-change in the segments 450-550 ms and 650-700 ms, all *p*s < 01,

η^2s > .42. This main effect just missed significance in the segments 400-450 ms and 600-650 ms, all *p*s < .08. Additionally, there were significant interactions between task and color-change in the segments 450-550 ms and 600-700 ms, all *p*s < .05, η^2s > .25. This interaction just missed significance in the segments 350-450 ms, all *p*s < .10.

Further analyses revealed that in the segments 450-550 ms after the onset of the first stimulus the Cz-amplitude in NoGo_rr trials was significantly increased compared to that in Go_gg trials, all *p*s < .01. In the segment 600-650 ms the greater Cz-amplitude in NoGo_rr trials compared to that in Go_gg trials just missed significance, *t*(14) = 1.79, *p* < .10. The Cz-amplitude in NoGo_rg trials was increased compared to that in Go_gr trials in the segment 450-500 ms, *t*(14) = 2.43, *p* < .05, whereas the Cz-amplitude in Go_gr trials was increased compared to that in NoGo_rg trials in the segment 500-550 ms, *t*(14) = 2.14, *p* = .05. Furthermore, the Cz-amplitude in NoGo_rr trials was significantly increased compared to that in NoGo_rg trials in the segments 450-550 ms, all *p*s < .01, whereas the Cz-amplitude in NoGo_rr trials was significantly smaller than that in NoGo_rg trials in the segments 600-700 ms, all *p*s < .05. Finally, the Cz-amplitude in Go_gr trials was increased compared to that in Go_gg trials in the segment 650-700 ms, *t*(14) = 2.16, *p* = .05.

For the Pz-electrode data, an ANOVA with repeated measures on the factors task (Go, NoGo), color-change (with, without), and time (50 ms-segments spanning the period from 300-700 ms after the first stimulus' onset) was conducted for the conditions Go_gg, Go_gr, NoGo_rr, and NoGo_rg.

The factor task, *F*(1, 14) = 17.61, *p* < .01,. η^2 = .56, and the factor time were significant main effects, *F*(7, 98) = 8.53, *p* < .001,. η^2 = .38. Additionally, there were significant interactions between task and time, *F*(7, 98) = 5.07, *p* < .05,. η^2 = .27, as well as between color-change and time, *F*(7, 98) = 27.06, *p* < .001,. η^2 = .66.

The ANOVAs for the single segments with repeated measures on the factors task (Go, NoGo) and color (with or without color-change) revealed a significant main effect of task in the segments 350-550 ms after the onset of the first stimulus, all *p*s < .05, η^2s > .34. This main factor just missed significance in the segments

550-600 ms, all $ps < .08$. However, there was also a significant main effect of color-change in the segments 350-550 ms and 600-700 ms, all $ps \leq .05$, η^2s > .24. Additionally, there were significant interactions between task and color-change in the segment 450-500 ms, $F(1, 14) = 6.30$, $p < .05$,. $\eta^2 = .31$.

Further analyses revealed that in the segments 400-550 ms after the onset of the first stimulus the Pz-amplitude in Go_gg trials was significantly increased compared to that in NoGo_rr trials, all $ps < .01$. The Pz-amplitude in Go_gr trials was increased compared to that in NoGo_rg trials in the segment 350-550 ms, all $ps < .05$ Furthermore, the Pz-amplitude in Go_gr trials was significantly increased compared to that in Go_gg trials in the segments 350-400 ms, $t(14) = 2.39$, $p < .05$, and 650-700, all $ps < .01$. Contrary, the Pz-amplitude in Go_gg trials was significantly larger than that in Go_gr trials in the segments 450-550 ms, all $ps < .01$. Finally, the Pz-amplitude in NoGo_rr trials was increased compared to that in NoGo_rg trials in the segments 400-550 ms, all $ps < .01$, whereas the Pz-amplitude in NoGo_rg trials was increased compared to that in NoGo_rr trials in the segments 600-700 ms, all $ps < .05$.

(2) Effects of the second signal in the Go_NoGo and the NoGo_Go condition

The second analysis tested if the presentation of the second signal in the Go_NoGo and the NoGo_Go condition had an additional influence on the ERPs that exceeded the effects of the color-change. Therefore, the ERPs at Cz and Pz in those conditions were compared that contained the same color-change. Accordingly, the ERP in the Go condition with color-change (Go_gr) was compared to that in the Go_NoGo condition and the ERP in the NoGo condition with color-change (NoGo_rg) was compared with that in the NoGo_Go condition. Figure 16 presents the ERPs for these four conditions. Those ERPs that were compared in the statistical analysis are printed in the same color. The ERPs start with the onset of the first signal. To facilitate reading of the data, in the following all findings in the ERPs are reported relative to the onset of the first signal.

The first 500 ms in Go_gr and Go_NoGo trials as well as in NoGo_rg trials and NoGo_Go trials are quite similar. After 500 ms there is a positive maximum at the Cz-electrode for Go_NoGo and a positive maximum at the Pz-electrode for

NoGo_Go. There is nothing comparable in the Go and NoGo conditions with color-change (Go_gr, NoGo_rg).

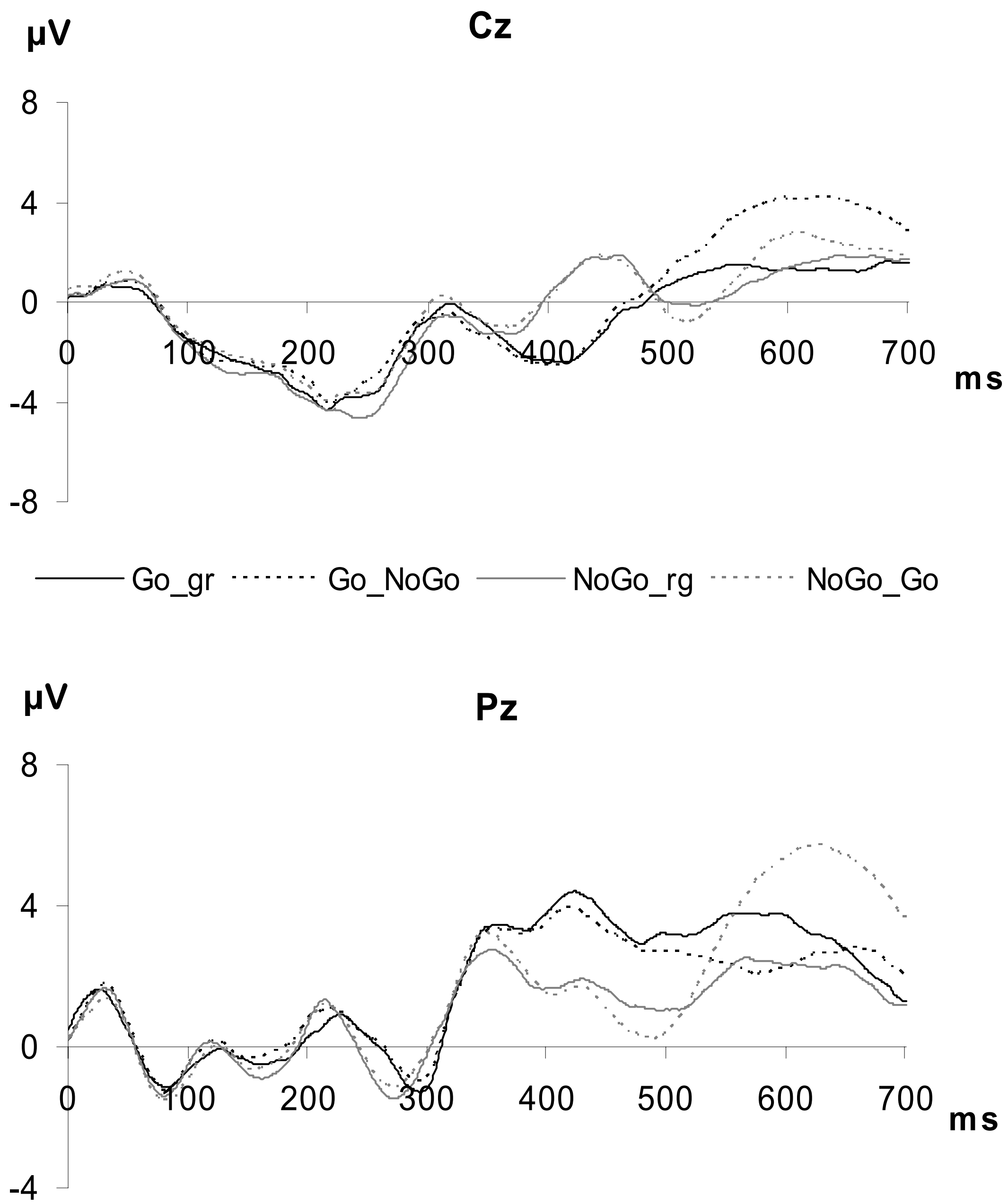


Figure 16. Grand-averaged event-related potentials (ERPs) over 16 participants for the Go condition with color-change (Go_gr), the NoGo condition with color-change (NoGo_rg), the Go_NoGo condition, and the NoGo_Go condition. ERPs are shown for the Cz- (above) and the Pz-electrode (below).

The 8 segments in the conditions Go_gr and Go_NoGo were entered into an ANOVA with repeated measures on the factors electrode (Cz, Pz), stimulus (Go, NoGo), and time (50 ms-segments spanning the period from 300-700 ms after the first stimulus' onset). The ANOVA revealed significant main effects of electrode, $F(1, 14) = 14.95$, $p < .01$,. $\eta^2 = .52$, and time, $F(7, 98) = 13.91$, $p < .001$,. $\eta^2 = .50$. The main effect of stimulus just missed the conventional level of significance, $F(1, 14) = 3.31$, $p < .10$,. $\eta^2 = .19$. Additionally, there were significant interactions between the factors electrode and stimulus, $F(1, 14) = 18.34$, $p < .01$,. $\eta^2 = .57$, electrode and time, $F(7, 98) = 24.16$, $p < .001$,. $\eta^2 = .63$, and between the factors electrode, stimulus, and time, $F(7, 98) = 12.63$, $p < .001$,. $\eta^2 = .47$. The interaction between the factors stimulus and time just missed the conventional level of significance, $F(7, 98) = 2.49$, $p < .09$,. $\eta^2 = .15$.

Further analyses of the ERPs at Cz revealed that the Go condition with color-change (Go_gr) and the Go_NoGo condition did not differ significantly until 500 ms after presentation of the first stimulus, all ps > .17. In subsequent segments, Go_NoGo had an increased amplitude compared to Go_gr, which nearly reached significance in the segment 550-600 ms, $t(15) = 2.06$, $p = .06$, and was significant in the segments 600-700 ms, all ps < .01.

At the Pz-electrode, the amplitude in the Go_NoGo condition was significantly smaller than that in the Go_gr condition in the segment 550-600 ms, $t(15) = 2.34$, $p = .05$. Both conditions did not differ significantly in any other segment at Pz, all ps > .14.

In order to compare ERPs between the NoGo condition with color-change (NoGo_rg) and the NoGo_Go condition an ANOVA was conducted with repeated measures on the factors electrode (Cz, Pz), stimulus (Go, NoGo), and time (50 ms-segments spanning the period from 300-700 ms after the first stimulus' onset). Electrode, $F(1, 14) = 11.08$, $p < .01$,. $\eta^2 = .44$, stimulus, $F(1, 14) = 7.25$, $p < .05$,. $\eta^2 = .34$, and time, $F(7, 98) = 14.26$, $p < .001$,. $\eta^2 = .51$, were significant main effects. Additionally, there were significant interactions between the factors electrode and stimulus, $F(1, 14) = 6.86$, $p < .05$,. $\eta^2 = .33$, electrode and time, $F(7, 98) = 6.06$, $p < .001$,. $\eta^2 = .30$, stimulus and time, $F(7, 98) = 13.30$, $p < .001$,. $\eta^2 = .49$, and between the factors electrode, stimulus, and time, *F*(7, 98) = 14.10, $p < .001$,. $\eta^2 = .50$.

At the Cz-electrode, the amplitude in NoGo_rg trials was significantly smaller than that in NoGo_Go trials in the segments 200-350 ms, all *p*s < .05. In the segments 550-650 ms, the amplitude in Go_NoGo trials was slightly more increased than that in NoGo_rg trials. However, this difference did not reach significance, all *p*s < .10.

The analysis of the ERPs at Pz revealed that the NoGo condition with color-change (NoGo_rg) and the NoGo_Go condition did not differ significantly until 450 ms after presentation of the first stimulus, all *p*s > .32. In the segment 450-500 ms after the presentation of the first stimulus, NoGo_rg trials displayed an increased amplitude compared to that in NoGo_Go trials, *t*(15) = 2.62, $p < .05$. In contrast, there was an increased Pz-amplitude in NoGo_Go trials compared to that in NoGo_rg trials in the segments from 550-700 ms after the presentation of the first stimulus, all *p*s < .01.

4.2.3 Topographical mapping

Figure 17 shows the scalp topographic voltage maps of the Go_NoGo and the NoGo_Go condition at the global field power peaks of the first and the second stimulus' P300 time frames. The first P300 centroid of the NoGo_Go condition and the second P300 centroid of the Go_NoGo condition are more anterior than the first P300 centroid of the Go_NoGo condition and the second P300 centroid of the NoGo_Go condition.

This was confirmed in an ANOVA with repeated measures on the factors condition (Go, NoGo) and P300-centroid (after first and second stimulus) which revealed a significant interaction between condition and P300 centroid, *F*(1, 14) = 12.13, $p < .01$, $\eta^2 = .46$.

Additional t-tests showed that the first centroid of the NoGo_Go condition was significantly more anterior than the first centroid of the Go_NoGo condition, *t*(15) = 3.03, $p < .01$. In contrast, the second centroid of the Go_NoGo condition was more anterior than the second centroid of the NoGo_Go condition, *t*(15) = 2.1

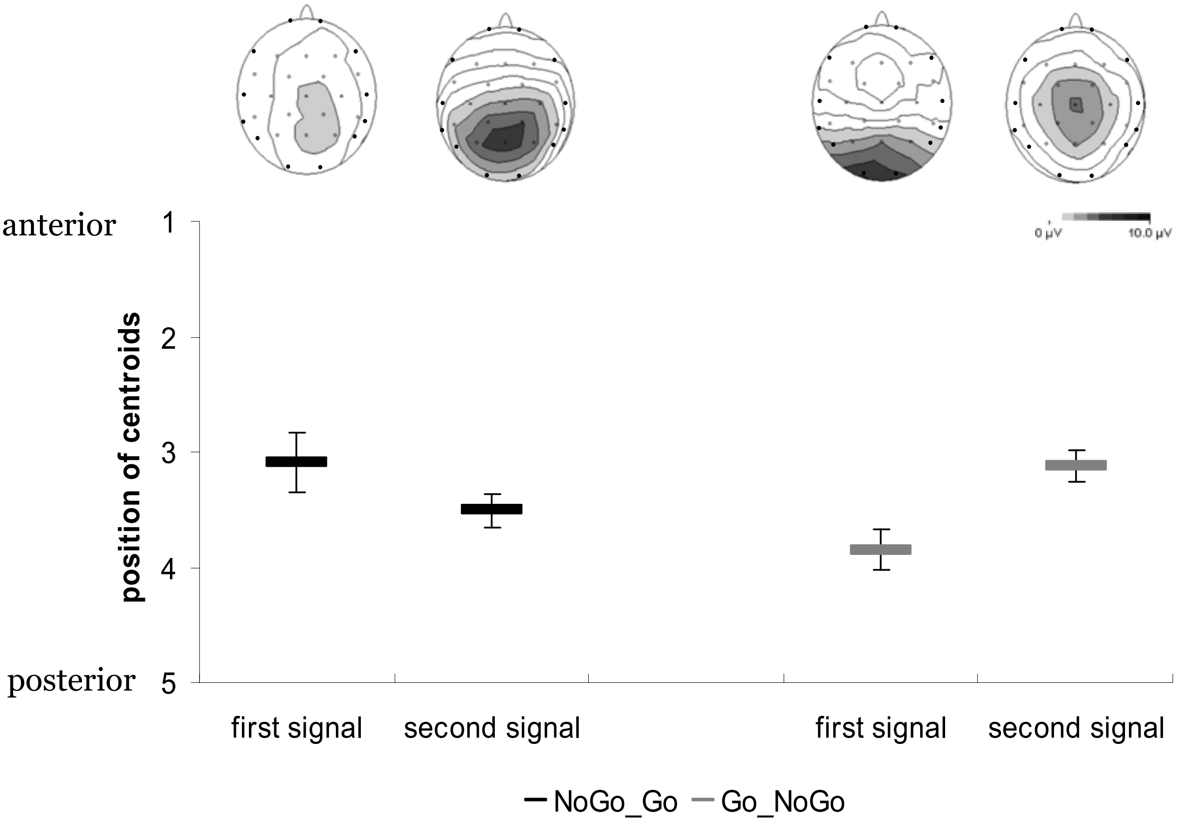


Figure 17. Positions of the NoGo_Go and Go_NoGo centroids at the peak of the global field power in the P300 time frames for the first and the second signal. Upper panel: Corresponding scalp topographic voltage maps.

4.3 Discussion

Experiment 3 investigated whether the typical topography of the NoGo-P300 is specific to inhibition or whether it could be explained in the context of response conflict. Therefore, Go and NoGo conditions were compared that contained the same kind of conflict. To implement this, two signals with different requirements were presented in a Go and a NoGo condition. In the NoGo_Go condition the requirements changed from inhibition of a key press (first signal) to its execution (second signal). In the Go_NoGo condition the requirements changed from the activation of a key press (first signal) to its inhibition (second signal). Therefore, presenting the second signal triggered a competing process with the ongoing process, which was triggered by the first stimulus. Accordingly, the second signal introduced conflict regardless of whether it was a Go or a NoGo signal.

The behavioral data showed that compared to the Go condition without color-change (Go_gg) the mean reaction time (RT) was significantly slower in the Go condition (Go_ambi), in which one symbol was linked to two different response requirements (Go_ambi vs. Go_NoGo). However, it seems safe to assume that participants did not change their response strategy in the Go_ambi condition such that they waited with the key press until they were sure not to perform an erroneous response. The mean RT in the NoGo_Go condition was more than 100 ms longer than that in the Go_ambi condition. Furthermore, even when considering the delayed onset of the Go signal in the NoGo_Go condition, the mean RT was significantly slower than that in the simple Go_gg condition. These findings are well in line with the results of Experiment 2, in which the mean RT in the NoGo primed Go condition was slower than that in the neutrally primed Go condition. It seems that in fact the inhibition process disturbed the response process in the NoGo primed Go condition of Experiment 2 as well as in the NoGo_Go condition of Experiment 3. Interestingly, the mean RT in the Go condition with color-change (Go_gr) was also significantly longer than that in the Go condition without color-change (Go_gg). Maybe, the color-change from green to red disturbed the processing of the Go signal, because the red color occurred twice as much in NoGo trials than in Go trials.

Moreover, the increased error rates in the Go_gr condition and in the NoGo_Go condition compared to those in the other Go conditions support the idea that the response process was disturbed in these Go conditions.

Among the NoGo conditions participants committed the most errors in the Go_NoGo condition, as could be expected from the assumptions of the Horse Race Model (see section 1.2); if the response process, which is triggered by the Go signal, wins the race against the inhibitory process triggered by the NoGo signal a response is performed erroneously. However, participants committed a lot of errors in the NoGo condition with color-change (Nogo_rg) as well. Probably, the color-change from green to red caused the high error rate because the green color occurred twice as much in Go trials than in NoGo trials. Therefore, the green color could have triggered a response process leading to error.

The effects of the color-change on RTs also suggest an effect on ERPs. In fact, the comparison between the Go conditions with and without color-change

(Go_gg and Go_gr) and the NoGo conditions with and without color-change (NoGo_rr and NoGo_rg) showed that the color-change had a significant influence on ERPs in the P300 time frame of the first (300–600 ms) and the second signal (400–700 ms) at the Cz-and the Pz-electrode. The Cz-amplitude in NoGo_rr trials was larger than that in NoGo_rg trials in the P300 time frame of the first signal and it was smaller than that in NoGo_rg trials in the P300 time frame of the second signal. It seems that the second signal in the NoGo_rg condition elicited a second P300, whereas this was not the case in the NoGo_rr condition. A similar finding was seen in the Go conditions. The Pz-amplitude in Go_gr trials was increased compared to that in Go_gg trials in the P300 time frame of the first and the second signal. However, in the segments 450-550 ms after onset of the first signal the Pz-amplitude in Go_gg trials was larger than that in Go_gr trials indicating that the color-change in Go_gr and NoGo_rg trials elicited a second P300.

However, the typical Go/NoGo differences were mostly unaffected by color-changes. Firstly, the Cz-amplitude in the NoGo conditions was increased compared to that in the Go conditions, except for the segment 500-550 ms after onset of the first signal, in which the Cz-amplitude in Go_gr trials was larger than that in NoGo_rg trials. Secondly, the Pz-amplitude in the Go conditions was enhanced compared to that in the NoGo conditions. One explanation for the larger Cz-amplitude in the Go_gr condition compared to that in the NoGo_rg condition could lie in the differential probabilities of the color green and the color red in Go and in NoGo conditions. Participants could have processed the color green as a feature of Go signals, whereas they could have processed the color red as a feature of NoGo signals; especially as this coherence comes up to the meaning of traffic lights. Accordingly, each green-colored signal could have triggered a response process while each red-colored signal could have triggered an inhibitory process. Therefore, similar processes could be at work in the Go and NoGo conditions with color-change and in the Go_NoGo and NoGo_Go conditions. However, the comparison between the conditions, which contained the same color-change (Go_gr vs. Go_NoGo and NoGo_rg vs. NoGo_Go) revealed that the presentation of the second signal in the Go_NoGo and the NoGo_Go condition had an additional influence on the ERPs at the Cz- and at the Pz-electrode.

The NoGo signal in the Go_NoGo condition elicited a second P300 with maximum at the Cz-electrode. In the Go condition with the same color-change there was no such event-related potential (ERP) at the Cz-electrode. Conversely, the Pz-amplitude after the color-change in the Go condition with color-change was increased compared to that in the Go_NoGo condition. Therefore, it seems that the NoGo signal in the Go_NoGo condition elicited a NoGo-P300, whereas the same color-change acting as the second Go signal in the Go condition with color-change elicited a second Go-P300.

Interestingly, the Cz-amplitude in the NoGo condition with color-change is smaller than that in the NoGo_condition in the segment 200-350 ms after the onset of the first signal. This means that the NoGo condition with color-change elicited a larger N200 after the first signal than the NoGo_Go condition. Maybe, the presentation of the second NoGo signal appearing in the “Go color” green increased the mental effort of evaluating the first signal in the NoGo condition with color-change. Furthermore, the Cz-amplitude in the NoGo_Go condition was increased compared to that of the NoGo condition with color-change in the segments from 550-650 ms after the presentation of the first stimulus. This result could be explained by the occurrence of a second P300 in the NoGo_Go condition with a maximum at the Pz-electrode. Nothing comparable was observed in the NoGo condition with color-change[15].

The results concerning the topography of the P300 after the second signal in the Go_NoGo and NoGo_Go conditions contradicted the assumption that the NGA results from conflict on a response level. The Go_NoGo and the NoGo_Go condition contained the same kind of conflict between a response and an inhibitory process. However, the characteristics of the NGA remained: The centroid of the P300 after a Go signal was located more posterior than that after a NoGo signal, independently of the presence or absence of conflict.

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[15] An additional finding at the Pz-electrode was that the Go signal of the NoGo_Go condition elicited a negative potential in the segment 450-500 ms after the presentation of the first stimulus. Maybe, this negative potential is a second N200 elicited due to the conflict triggered by the Go signal. However, this negative potential occurrs relatively late (350-400 ms after the presentation of the Go signal) and its topography (parietal maximum) is unusual for a N200.

Taken together, there was no evidence that conflict on a response level is responsible for the NGA. Rather, the results point to a link between the NoGo-P300 and inhibition.

How can these results be reconciled with the findings of Fallgatter, Bartsch, and Herrmann (2002) who claimed that the anterior cingulate cortex (ACC) is the location with the most prominent activation difference between Go and NoGo conditions? This question is relevant, because recent studies have implicated that the ACC plays a crucial role for conflict on a response level (Milham et al., 2001; vanVeen, Cohen, Botvinick, Stenger, & Carter, 2001) or have shown that it is active during mental effort (Bottvinick, Cohen, & Carter, 2004).

Two possible explanations for Fallgatter et al.'s (2002) results are imaginable. First of all, the higher activation of the ACC in the NoGo condition might be due to the characteristics of Fallgatter et al.'s Go and NoGo conditions. In their study, they used a special version of the Continuous Performance Test (see Rosvold, Mirsky, Sarason, Bransome, & Beck, 1956, for the classical version). Different letters were presented consecutively for 200 ms each with an interstimulus interval of 1650 ms. Participants were instructed to press a key each time the letter X appears after the letter O (Go condition). There were also trials in which the letter O was followed by another letter. These trials constituted the NoGo condition. Letters, which were not preceded by O, were regarded as distractors. The characteristics of this task are such that each Go condition was preceded by a cue (the letter O). Consequently, it was a good strategy for the participants to prepare a motor response after the occurrence of this cue. With this strategy they would hardly make errors (e.g., miss responses) in Go trials as they would have to simply execute the prepared response if the letter X appears in the next trial. Each NoGo signal was also preceded by a cue that triggered a response process. In this case however, the preparation of a response is inadequate because participants were not allowed to execute the prepared response in NoGo trials. That means that in Fallgatter et al.'s study the NoGo condition contained much more conflict on a response level than the Go condition, which would explain differences in activation of the ACC in the Go and the NoGo condition.

Secondly, the ACC could be connected to the inhibitory mechanism. Table 1 (section 1.5) contains an ordered list of brain regions that have been found to be active during tasks requiring inhibition. The more studies named a particular region the higher was its rank in the list. The ACC is listed in third place. Moreover, Carter et al. (2000) investigated ACC function in a Stroop task (Stroop, 1935) during fMRI. In the Stroop task participants see names of colors printed in various colors. The participants alternate between two instructions. Under one instruction they have to read the word, whereas under the other instruction participants have to name the color in which the word is printed. Carter et al. distinguished two conditions under the color-naming instruction. In the congruent condition the word denoted the same color in which it was printed. In the incongruent condition the word denoted a color different from the one it was printed in. Therefore, in the incongruent condition the meaning of the word differed from the color in which the word was printed. Carter et al. implemented two kinds of blocks. In one block the ratio of congruent to incongruent trials was 4 to 1, while it was reversed in the other block. Therefore, the expectancies concerning the next trial varied between the two blocks. Accordingly, RTs in incongruent trials in the block with high expectancy for congruent trials were longer than those in the block with high expectancy for incongruent trials. Interestingly, the ACC activity in incongruent trials in the block with high expectancy for congruent trials was larger than that in incongruent trials in the block with high expectancy for incongruent trials. Carter et al. interpreted these findings as evidence for an evaluative function of the ACC detecting cognitive states such as response competition. However, the findings are also well in line with the assumption that the ACC is connected to the inhibitory mechanism. In the incongruent condition in the block with high expectancy for incongruent trials the low expectancy for congruent trials weakens the response process triggered by reading the word. This is the case, because in most of the trials the color denoted by the word does not correspond to the color in which the word is printed. Therefore, little inhibition is necessary to overcome false response tendencies. Conversely, in the incongruent condition in the block with high expectancy for congruent trials the response process resulting from reading the word is stronger. This is the case, because in most of the trials the color denoted by the word does correspond to the color in which the word is printed. Consequently, more inhibition is needed to overcome false response tendencies. Because weak inhibition should require less activity than strong inhibition, Carter et al.'s results could be interpreted as being in agreement with the notion that the ACC is connected to inhibition.

Even though Experiment 3 ruled out the possibility that the NGA is a result of conflict, Experiment 3 could not prove that the NGA is specific to the inhibitory mechanism. Falkenstein, Koshlykova, Kiroj, Hoormann, and Hohnsbein (1995) assumed that the P300 is the result of two overlapping event-related potentials (ERPs) in Go and NoGo trials[16]. In this conception the NGA results from more cognitive response selection in Go trials than in NoGo trials. Experiment 4 investigated this possibility.

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[16] One ERP should have its maximum at the Cz-electrode and is assumed to reflect stimulus identification (P-SR) and another ERP should have its maximum at the Pz-electrode and is assumed to reflect cognitive response selection (P-CR).

5 Does the NoGo-Anteriorization represent less cognitive response selection in NoGo trials?

Experiment 4 tested if the NoGo-Anteriorization (NGA) is the result of less cognitive response selection in NoGo trials than in Go trials. Falkenstein, Koshlykova, Kiroj, Hoormann, and Hohnsbein (1995) proposed that the P300 results from two overlapping event-related potentials (ERPs), one ERP (P-SR) reflects stimulus identification (Hohnsbein, Falkenstein, & Hoormann, 1998) and the other ERP (P-CR) reflects cognitive response selection (Falkenstein et al., 1995; see also section 2.2.3). The P-SR is assumed to have its maximum at the Cz-electrode while the P-CR should have its maximum at the Pz-electrode. According to this account the NGA results from a larger P-CR in Go trials than in NoGo trials.

One possibility of testing Falkenstein et al.'s (1995) assumptions is to investigate Go and NoGo trials without an exogenous signal that had to be identified. According to Falkenstein et al., no P-SR should occur under such conditions. In the absence of the P-SR the P-CR in Go and NoGo trials should be directly comparable. Furthermore, to estimate the influence of the P-SR on the Go- and the NoGo-P300, the Go and NoGo trials without exogenous signals were compared to Go and NoGo trials that contained a Go and a NoGo signal, respectively.

In order to implement a Go condition without an exogenous signal participants pressed a key in a self-paced manner without a signal instructing them when to do it.

In order to implement a NoGo condition without an exogenous signal participants had to perform a fixed sequence of key presses but sometimes were instructed to omit one key press of the sequence. These instructions were given prior to execution of the whole sequence by presenting the depiction of a pair of hands (see Figure 19 (B)). A finger colored in red instructed participants to leave out the key press with the respective finger. Therefore, there was no exogenous NoGo signal that had to be evaluated when participants withheld the key press.

Figure 18 summarizes the predictions concerning the P300-topography in case the NGA does indeed result from less cognitive response selection in NoGo trials.

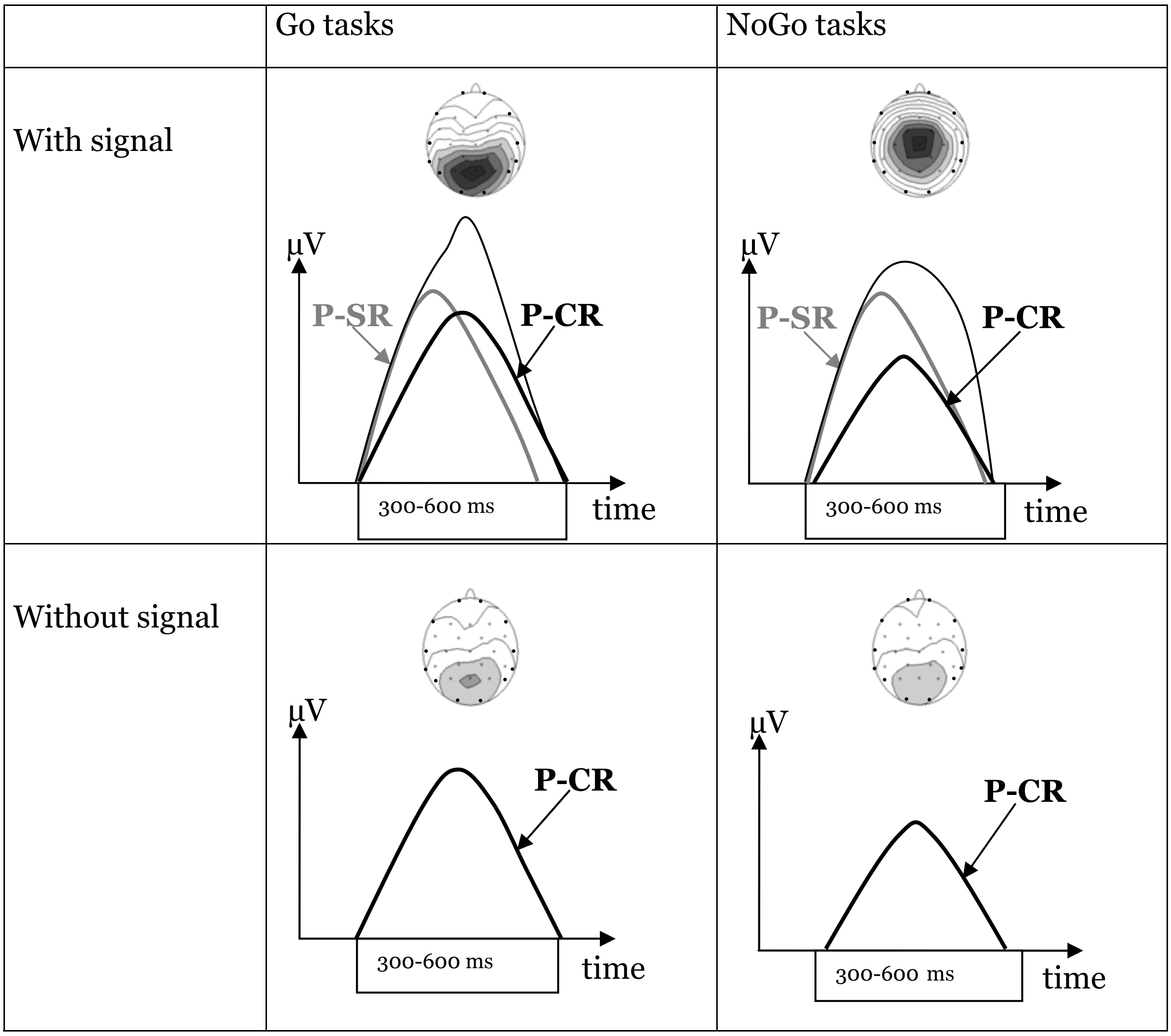

Figure 18. Summary of the expected topographies in Experiment 4 under the hypothesis that the P300 is the result of two overlapping event-related potentials (P-SR and P-CR) and the NGA originates from a smaller P-CR in NoGo trials.

In a Go task with Go signal participants have to identify the Go signal and they have to choose the correct response. Therefore, both P-SR and P-CR should occur. Moreover, the greater influence of the P-CR on the Go-P300 should result in a P300 with a parietal maximum (top left panel in Figure 18). In a NoGo task with NoGo signal there should also be both P-SR and P-CR. However, the smaller amplitude of the P-CR in NoGo trials than in Go trials should strengthen the influence of the P-SR on the NoGo-P300 (top right panel in Figure 18)[17]. Therefore, the maximum of the NoGo-P300 should be located more anterior than that of the Go-P300. In the absence of a signal that has to be identified there is no need for stimulus identification. Therefore, in Go trials without Go signal (lower left panel in Figure 18) as well as in NoGo trials without NoGo signal (lower right panel in Figure 18) only the P-CR (with a parietal maximum) representing cognitive response selection should be present. Because there is no P-SR-overlapping with the P-CR in Go and NoGo trials without signals the P300s should be smaller and located more posteriorly than those in tasks with signal. Furthermore, according to Falkenstein et al. (1995), the P-CR in NoGo trials without signal should be smaller than that in Go trials without signal.

In contrast, there should be a P300 with a posterior maximum in the Go tasks and a P300 with the maximum over the Cz-electrode in the NoGo tasks, if the NGA is linked to the inhibitory mechanism (see Table 7).

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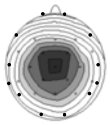
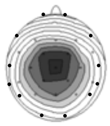
[17] One explanation for a smaller P-CR in NoGo conditions compared to that in Go conditions would be that the cognitive response selection in NoGo trials is easier than in Go trials.

Table 7. Summary of the expected topographies in Experiment 4 under the hypothesis that the P300 is linked to the inhibitory mechanism.

	Go tasks	NoGo tasks
With signal		
Without signal		

In the absence of exogenous signals the P300 in Experiment 4 was measured response-locked. That means that for Go trials the EEG-data around the onset of the response in each trial were averaged to obtain the P300. For NoGo trials the P300 was searched in the EEG-data between the two key presses that surrounded the key press that had to be left out.

5.1 Method

5.1.1 Participants

A total of 14 women and 4 men aged between 19 and 30 years (mean age 23.1 years) took part in the experiment. None of them suffered from neurological or psychiatric diseases (self-reported). All had normal or corrected-to-normal vision. All but three were right-handed (self-reported). All received partial course credit or were paid for participation. Participants gave their informed consent prior to their inclusion in the study. They were not informed about the study's specific hypotheses. Half of the participants first completed the Go tasks and then the NoGo tasks while the order was reversed for the other half.

All participants had more than 20 artifact-free Go trials after the artifact rejection procedure. However, 4 participants had to be excluded from the EEG-data-set of the NoGo task with NoGo signal, and 6 participants had to be excluded from the EEG-data set for the NoGo task without NoGo signal due to less than 20 artifact-free NoGo trials after the artifact rejection procedure. The EEG-data set for the NoGo task with NoGo signal consisted of 11 women and 3 men aged between 19 and 30 years (mean age 23.3 years) and the EEG-data set for the NoGo task without NoGo signal consisted of 9 women and 3 men aged between 19 and 30 years (mean age 23.0 years). In both data sets, all but three participants were right-handed.

To permit statistical comparisons between conditions, only participants were included in the analysis that had at least 20 artifact-free trials after the artifact rejection procedure in the respective conditions. For instance, the comparison between the NoGo task with and without signal included only participants who had at least 20 artifact-free NoGo trials in both NoGo tasks, which resulted in a data set of 12 participants.

5.1.2 Apparatus and stimulus materials

Participants were seated in a dimly illuminated and sound attenuated room. An IBM-compatible computer (Pentium IV with 2.60 GHz) equipped with a 17 inch VGA-Display (75 Hz) and the software E-Prime (Schneider, Eschman, & Zuccolotto, 2002) served for stimulus presentation. The viewing distance was 60 cm. Response keys in the Go tasks were the keys “1” and “3” of a standard keyboard. In the NoGo tasks, participants performed their response on the keys “s”, “d”, “f”, “j”, “k”, and “l” of the same keyboard.

Stimuli were crosses in the Arial font (size: 30 points) centered on the screen in white or red color on a black background. In the NoGo task without NoGo-signal additionally, two grey hands were presented (6.3 cm x 9.0 cm) on a black background. One of the index, middle, or ring fingers could be colored in red (see Figure 19).

In the short practice session before the NoGo task, a 250-ms tone was presented (44 kHz) through external loudspeakers with cross-onset.

5.1.3 Procedure

In the Go task with Go signal, participants were instructed to decide whether to press a key with the left or the right index finger in response to the appearance of a red cross. They had to choose each response approximately equally often and should not follow a fixed scheme (like alternating between left and right responses).

Each Go trial with Go signal started with the presentation of a white cross. After a randomly chosen interval of either 1700, 1900, 2100, 2300, or 2500 ms duration the white cross turned red and remained red until participants performed their response. Response times were recorded from the onset of the red cross. Afterwards, the next trial began. After each block participants were informed how often they had chosen each response so as to aid them in choosing both responses equally often without them having to count the responses themselves.

In the Go task without Go signal, participants were instructed similar except that they could choose when to press a key. The only constraint was that the time between two key presses should be at least 1 second and the intervals between presses should vary. During the entire block, participants saw a white cross in the center of the monitor. Like in the Go task with Go signal, participants were informed about the number of right and left responses they had performed at the end of each block.

Half of the participants first performed the Go task with Go signal and half of the participants started with the Go task without Go signal. Participants performed six blocks with 20 trials each.

The NoGo tasks started with a short practice block, in which participants practiced pressing the six keys “s”, “d”, “f”, “j”, “k”, and “l” in a sequence from left to right starting with the left ring finger and ending with the right ring finger. Participants started each sequence self-paced by pressing the space-bar. During the sequences, a white cross was presented in the middle of the monitor, and a tone indicated when to press a key. The interval between two tones was 1500 ms. Participants practiced the sequence 10 times.

Figure 19 (A) shows an illustration of the NoGo task with NoGo signal. Each sequence started with a white cross centered on the monitor. Participants had to start the trained sequence with at least 1 second between any two key presses. At some point in the NoGo sequence the white cross turned red after a key press. This was the sign for the participants to leave out the next key press. After 250 ms the cross turned white again. All sequences were divided by the presentation of a

2000-ms black screen. Participants were instructed to perform the trained sequence. No tones were presented in the experimental trials. Participants were asked to wait at least 1 second between the key presses of a sequence. If a red cross appeared, the next key press in the sequence should be skipped. By the time the red cross appeared, participants had to withdraw the key press which would be the next in the sequence. Participants were further instructed to wait twice as long as usual (i.e., 2 s instead of at least 1 s) before executing the response following the withheld response in the sequence.

There were five types of sequences: Sequences in which no key press was to be left out and NoGo sequences in which either the second, the third, the fourth, or the fifth key press was to be skipped. In none of the NoGo sequences was the first or the last key press to be left out.

Figure 19 (B) shows an illustration of the NoGo task without NoGo signal. Before executing a sequence, participants saw a drawing of a pair of two hands. One of the fingers in the drawing was depicted in red indicating which response in the sequence was to be skipped. Pressing the space-bar terminated the presentation of the drawing and started the sequence. The cross in the middle of the screen remained white throughout execution of the sequence. After each sequence black screen was presented for 2000 ms. Instructions were similar to the ones given for the NoGo task with NoGo signals, except that in this task, the drawing before each sequence informed participants which response to withhold.

There were the same types of sequences as in the NoGo task with NoGo signal.

Half of the participants first performed the NoGo task with NoGo signal followed by the NoGo task without NoGo signal while the order was reversed for the other half of participants. Participants completed three blocks with 20 sequences, each presented in a random order, and skipped a response 48 times within each NoGo task.

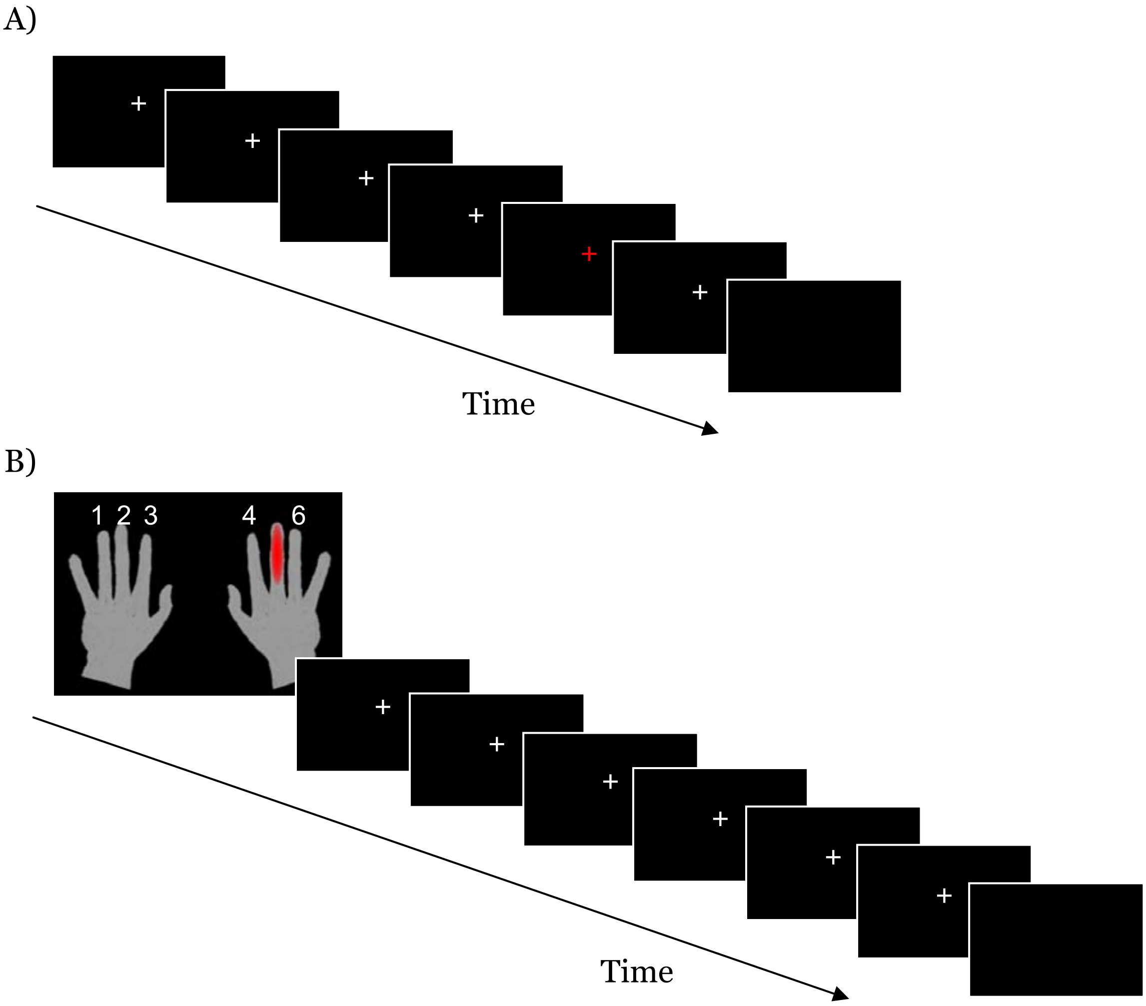


Figure 19. Illustration of a sequence in the NoGo task with signal (A) and in the NoGo task without signal (B).

5.1.4 EEG-Recordings and data analysis

Electrophysiological recordings were identical to Experiment 1, except for the following: An epoch in a Go trial lasted from 400 ms before a key press until 300 ms after that key press. An epoch in a NoGo trial started with the key press before a key press was skipped and ended 2000 ms later.

5.2 Results

5.2.1 Behavioral Data

In the Go tasks, the number of responses executed with the right hand was subtracted from the number of responses executed with the left hand. On average their difference did not significantly differ from zero, *p*s > .11.

Table 8 shows the mean interval between two key presses in the Go tasks and in the NoGo tasks with and without a key press left out. There were no errors in the NoGo task with NoGo signal and 2.41% errors in the NoGo task without NoGo signal.

Table 8. Mean interval between two key presses in Go and NoGo tasks

Condition	Mean interval between key presses (SD)	
	Without a key press left out	With a key press left out
Go task with Go signal	2492 ms (104 ms)	
Go task without Go signal	2064 ms (619 ms)	
NoGo task with NoGo signal	1565 ms (350 ms)	2555 ms (565 ms)
NoGo task without NoGo signal	1606 ms (348 ms)	2506 ms (551 ms)

Note. SD = standard deviation

5.2.2 ERP-data

Figure 20 shows the ERPs at the Cz- and the Pz-electrode for the two Go-conditions. Graphical inspection of the ERPs suggests differences between the Go conditions in the time frame from -250 to 150 ms at the Pz-electrode.

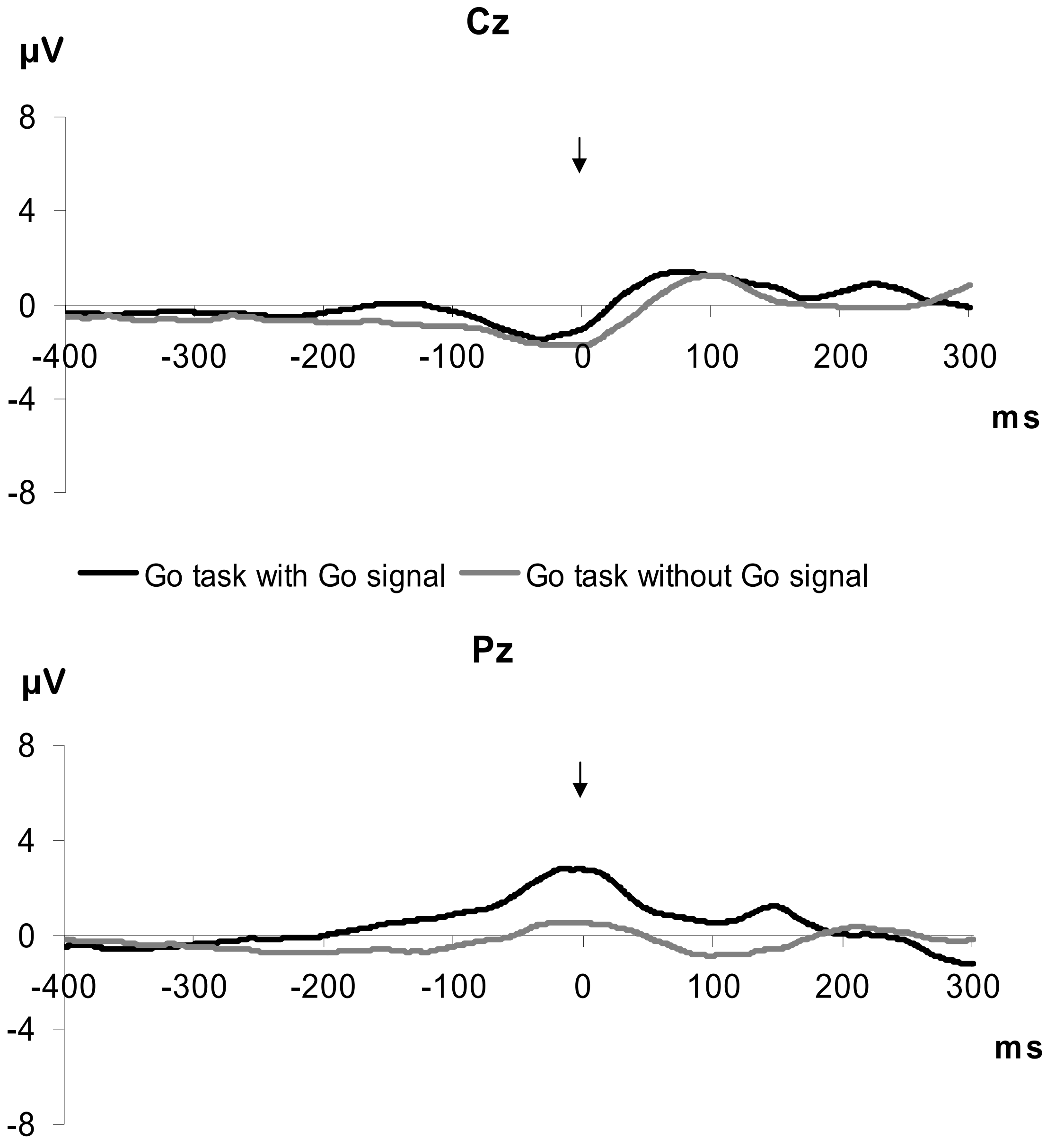


Figure 20. Grand-averaged event-related potentials (ERPs) over 18 participants. Comparisons between the Go task with Go signal and the Go task without Go signal. ERPs are shown for the Cz- (above) and the Pz-electrode (below). The arrow indicates the execution of the key press.

For the Cz-electrode data, an ANOVA with repeated measures on the factors condition (with signal and without signal) and time (14 segments of 50 ms-length) revealed significant main effects of condition, $F(1, 17) = 5.94$, $p < .05$, $\eta^2 = .26$, and time, $F(13, 221) = 17.57$, $p < .001$, $\eta^2 = .51$. The amplitude in the Go task with Go signal was increased compared to that in the Go task without Go signal in the segments from -150 to -100 ms, 0-50 ms, and 200-250 ms, all *p*s < .05. In the segments from -200 to -150 ms and from 50 to 100 ms, the increased amplitude in the Go task with Go signal just missed the conventional level of significance, all *p*s < .09.

For the Pz-electrode data, an ANOVA with repeated measures on the factors condition (with signal and without signal) and time (14 segments of 50 ms-length) revealed significant main effects of condition, $F(1, 17) = 31.50$, $p < .001$, $\eta^2 = .65$, and time, $F(13, 221) = 15.21$, $p < .001$, $\eta^2 = .47$. Additionally, there was a significant interaction between both factors, $F(13, 221) = 18.25$, $p < .001$, $\eta^2 = .52$. Additional t-tests showed that the amplitude in the Go task with Go signal was increased compared to that in the Go task without Go signal in the segments from -250 to 200 ms, all *p*s < .05. In the segment 250-300 ms after a key press, the amplitude in the Go task without Go signal was enhanced compared to that in the Go task with Go signal, $t(17) = 3.46$, $p < .01$.

Figure 21 shows the ERPs at the Cz- and the Pz-electrode for the two NoGo-conditions. Graphical inspection of the ERPs suggests differences between the NoGo conditions in the time frame 0-600 ms at the Cz and at the Pz-electrode.

For the Cz-electrode data, an ANOVA with repeated measures on the factors condition (with signal and without signal) and time (14 segments of 50 ms-length) revealed a significant main effect of time, $F(39, 429) = 19.34$, $p < .001$, $\eta^2 = .64$, and a significant interaction between the factors condition and time, $F(39, 429) = 21.03$, $p < .001$, $\eta^2 = .66$. The amplitude in the NoGo task with NoGo signal was increased compared to that in the NoGo task without NoGo signal in the segments 100-550 ms, all *p*s < .05. The increased amplitude in the NoGo task without NoGo signal compared to that in the NoGo task with NoGo signal missed the conventional level of significance in the segments 700-800 ms, both *p*s < .08, but was statistically significant in the segments 800-2000 ms, all *p*s < .05.

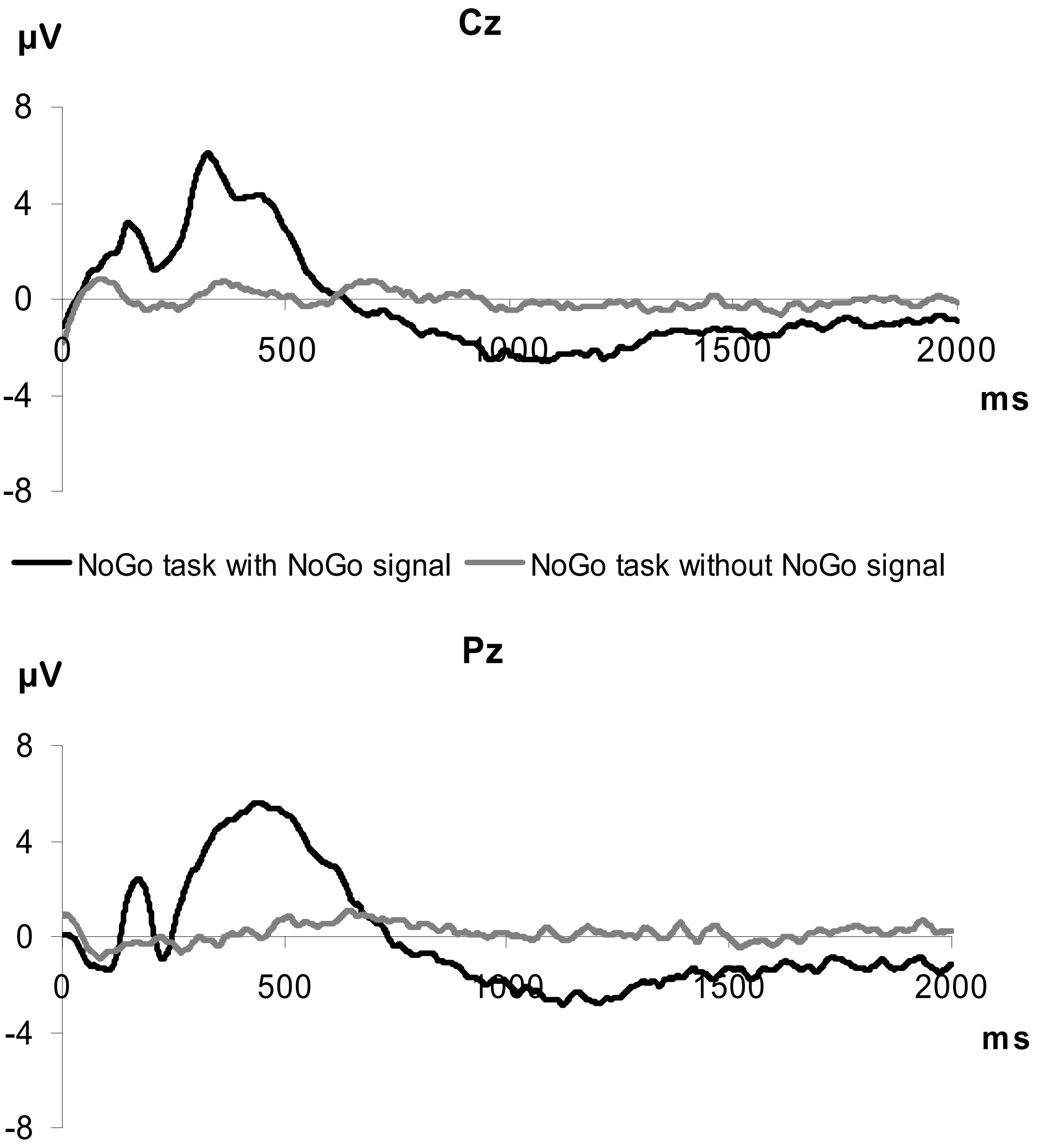


Figure 21. Grand-averaged event-related potentials (ERPs) over 12 participants. Comparison between the NoGo task with NoGo signal and the NoGo task without NoGo signal. ERPs are shown for the Cz- (above) and the Pz-electrode (below). The time scale starts with the onset of the last key press in the sequence before one key press was skipped.

For the Pz-electrode data, the ANOVA with repeated measures on the factors condition (with signal and without signal) and time (14 segments of 50 ms-length) revealed a significant main effect of time, $F(39, 429) = 21.08$, $p < .001$, $\eta^2 = .66$. The main effect of condition just missed significance, $F(1, 11) = 3.32$, $p < .10$.

Additionally, there was a significant interaction between the factors condition and time, $F(39, 429) = 23.30$, $p < .001$, $\eta^2 = .68$. The amplitude in the NoGo task with NoGo signal was increased compared to that in the NoGo task without NoGo signal in the segments 150-200 ms and 250-650 ms, all $ps < .01$. In the segments 750-2000 ms, however, the amplitude in the NoGo task without NoGo signal was significantly increased compared to that in the NoGo task with NoGo signal, all $ps < .05$.

5.2.3 Topographical mappings

Figure 22 shows the scalp topographic voltage maps for the two Go tasks and the NoGo task with NoGo signal at the global field power peak in the P300 time frame. Because there was no peak for the NoGo task without NoGo signal, it was not possible to produce a corresponding topographical map. The P300 centroids of the two Go tasks are very similar, whereas the centroid of the NoGo task with Nogo signal has a more anterior topography.

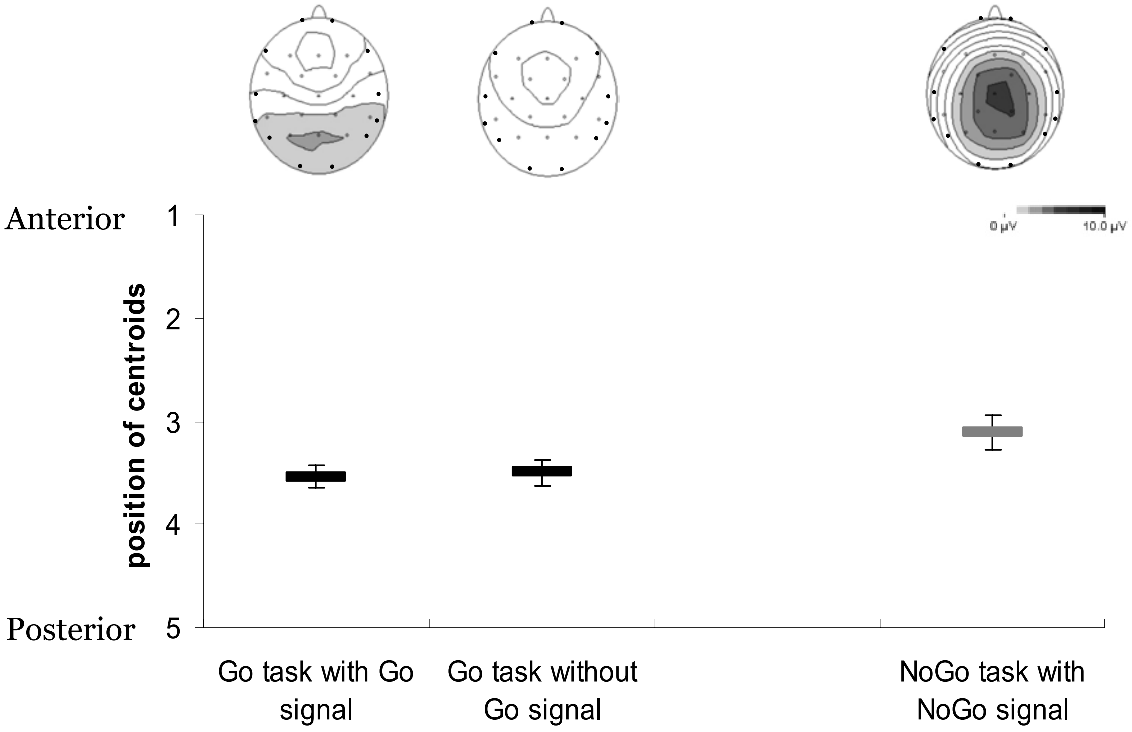


Figure 22. Positions of the centroids at the peak of the global field power in the P300 time frames for the Go tasks (with Go signal and without Go signal) and the NoGo task with NoGo signal as well as corresponding scalp topographic voltage maps (upper panel).

T-tests showed, that the centroids of the Go tasks did not differ in location, $t(17) = 0.41$, $p = .68$. However, the centroid of the NoGo task with NoGo signal was located more anteriorly than that of the Go task with Go signal, $t(13) = 2.83$, $p < .05$. The corresponding difference between the centroids of the NoGo task with NoGo signal and the Go task without Go signal missed marginally the conventional level of significance, $t(13) = 1.92$, $p < .08$.

5.3 Discussion

Experiment 4 investigated, whether the typical topography of the NoGo-P300 is specific to inhibition or whether it can be explained by less cognitive response selection in NoGo trials compared to Go trials. The idea that the NoGo-Anteriorization (NGA) is the result of less cognitive response selection in NoGo than in Go trials rests on the assumption that the P300 is composed of two event-related potentials (P-SR and P-CR; Falkenstein, Koshlykova, Kiroj, Hoormann, & Hohnsbein, 1995). According to Hohnsbein, Falkenstein, and Hoormann (1998) the P-SR represents stimulus identification while the P-CR represents cognitive response selection (Falkenstein et al.). A smaller amplitude of the P-CR in NoGo trials compared to Go trials is assumed to be the reason for the NGA. To investigate P-CRs in Go and NoGo trials Experiment 4 introduced a Go and a NoGo task without exogenous signals that had to be identified. These tasks were compared to Go and NoGo tasks that included exogenous signals.

The behavioral data showed that for the Go tasks participants complied with instructions to execute an equal number of responses with the index finger of their left and right hand, respectively. In the NoGo trials participants also followed successfully instructions to extend the time-period between two responses when skipping one response in the sequence; although, on average that time was not double the inter-response interval between regular consecutive responses.

The Go task in which participants had to identify a Go signal (Go task with Go signal) has an increased P300-amplitude compared to that in the Go task without Go signal. This is well in line with Falkenstein et al.'s (1995) assumptions (see Figure 18). However, although there is a NoGo-P300 for the NoGo task in which stimulus identification should have taken place (NoGo task with NoGo signal), there is no peak at all in the NoGo task without NoGo signal. This finding is at odds with Falkenstein et al.'s assumption that a P-CR would occur in NoGo trials. However, it also contradicts the idea that the NoGo-P300 is connected to an inhibitory mechanism. Otherwise, there should be a peak at the Cz-electrode when participants omitted a key press in the sequence.

The centroids of both Go tasks were located close to the Pz-electrode and they were located more posteriorly than that of the NoGo task with NoGo signal, although, this difference was just marginally significant for the Go task without Go signal. This pattern of differences in centroid locations was expected on the basis of the assumption that the topography of the Go-P300 would be mainly influenced by the P-CR. The fact that the centroids of the Go tasks did not differ in position, however, is not in line with Falkenstein et al.'s assumptions: The centroid of the Go task without Go signal should have been located more posteriorly than that of the Go task with Go signal, due to the absence of the P-SR in the Go task without Go signal.

The centroid of the NoGo task with NoGo signal was located close to the Cz-electrode. This finding is well in line with the idea that the topography of the NoGo-P300 is influenced mainly by the topography of the P-SR. As a result of missing peaks in the NoGo task without NoGo signal it was not possible to determine a P300 centroid for this condition.

Does this mean that the NoGo-P300 is not a composit of P-SR and P-CR and not connected to an inhibitory mechanism?

Problematic to this view is the significantly increased Cz- and Pz-amplitude in the NoGo task without NoGo signal compared to that in the NoGo task with NoGo signal in the segments from approximately 600 to 2000 ms after the last key press in a sequence before a key press was skipped. Maybe, the missing peak in the NoGo task without NoGo signal results from a methodical problem. In absence of an external signal that indicates when to inhibit the key press, the inhibition of the key press could have taken place at different moments in time in each trial for each participant. An averaging across trials would then diminish any peaks due to their variable occurrence within trials. In contrast, the NoGo signals in the NoGo task with NoGo signal are in a way pacemakers for the inhibition of the key press. If this holds true, the NoGo-P300 in the NoGo task with NoGo signal should also be diminished when the ERP is locked to the point in time when participants restart the sequence after omitting a key press.

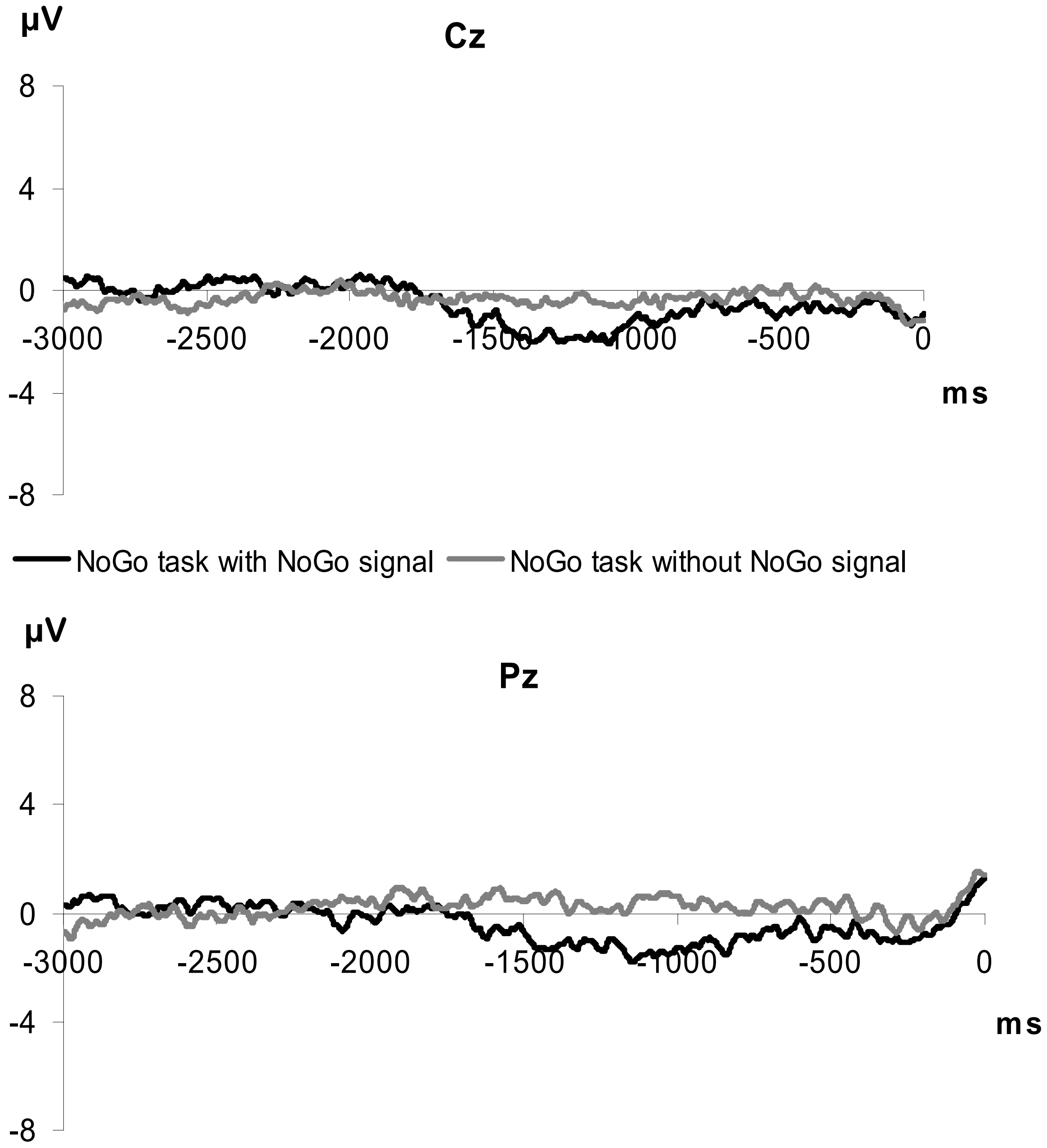


Figure 23. Grand-averaged event-related potentials (ERPs) over 12 participants. Comparisons between the NoGo task with NoGo signal and the NoGo task without NoGo signal. ERPs are shown for the Cz- (above) and the Pz-electrode (below). The time scale ends with the onset of the first key press in the sequence after one key press was skipped.

In order to investigate this possibility, both NoGo tasks were segmented such that they began 3000 ms before participants restarted the sequence. This time frame was chosen because behavioral data showed that, on average, the execution of the two key presses (before and after skipping a key press) took place within this interval. The graphical results are shown in Figure 23.

No peaks were apparent in either of the two NoGo tasks. Therefore, it seems reasonable to assume that the standard analysis of event-related potentials averaged across trials is not appropriate in this setting. Rather, a method should be chosen that allows detecting Cz-activity at point in time in each trial. Accordingly, the four conditions of Experiment 4 (Go task with Go signal, Go task without Go signal, NoGo task with NoGo signal, and NoGo task without NoGo signal) were analyzed with a Fast Fourier Transformation (FFT).

5.4 Fast Fourier Transformation

The recorded EEG-signal consists of different frequency ranges between 0.5 and 50 Hz. A Fast Fourier Transformation (FFT) analyzes the amount of activity in each frequency range in the EEG (see for instance Skrandies, 2000). Because many studies found a P300 lasting approximately 300 ms (e.g., Eimer, 1993; Bruin, Wijers, & van Staveren, 2001; Kopp, Mattler, Goertz, & Rist, 1996; Kopp, Rist, & Mattler, 1996; Falkenstein, Hoormann, & Hohnsbein, 1999) the frequencies in a range between 0.5-3.5 Hz (delta range) seem to contribute especially to the characteristics of the P300. Furthermore, Fell et al. (2004) observed a direct relationship between the P300 and the delta frequency (see also Demiralp, Ademoglu, Comerchero, & Polich, 2001). In their study patients with unilateral temporal lobe epilepsy performed a Go-NoGo task with infrequent Go stimuli. Fell et al. recorded the P300, which was elicited by the infrequent Go stimuli, via a depth electrode from the patients' healthy medial temporal lobe and wavelet transformed[18] the data to analyze the origin of the P300 in the frequency domain. The Wavelet Analysis revealed that the P300 was produced by stimulus-related

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[18] The Wavelet Analysis decomposits temporal and frequential information of the EEG.

phase-locking of ongoing delta activity as well as by increased delta activity in neural assemblies.

Therefore, only the FFT-data for the delta frequency range are reported in the following section. The results of the FFTs concerning the other frequency ranges (theta frequency: 3.5-7.5 Hz, alpha frequency: 7.5-12.5 Hz, and beta frequency: 12.5-30 Hz) are presented in the Appendix. Increased activity in the delta range at the Cz-electrode in both NoGo tasks would support the assumption that there is a NoGo-P300 in the NoGo task without NoGo signal. FFTs were conducted for both Go and NoGo conditions at the Cz-electrode.

5.4.1 Participants

To permit the comparison of Go and NoGo tasks only the data of those participants were included in statistical analyses who had at least 20 artifact-free epochs in all four conditions (Go tasks with and without Go signal and NoGo tasks with and without NoGo signal) after the artifact rejection procedure. The effective EEG-data set consisted of 9 women and 3 men aged between 19 and 30 years (mean age 23.0 years). All but three were right-handed.

5.4.2 EEG-Recordings and data analysis

The Fast Fourier Transformation was conducted with the software Brain Vision Analyzer 1.05 (Brain Products GmbH, 2002). The first steps in the data analysis were the same as described in Experiment 1, except that the average referenced data were filtered digitally with a 40 Hz low pass filter instead of a 30 Hz low pass filter. After the occular artifact correction (Gratton, Coles, & Donchin, 1983) epochs of 1024 ms were segmented in the Go conditions and epochs of 2048 ms were segmented in the NoGo conditions. The lengths of these epochs corresponded to 1024 data points in the Go conditions and 2048 data points in the NoGo conditions. These epochs were chosen because for comparisons between FFT-results that derived from segments of different length the number of data points should ideally be a power of 2 as is recommended in the user manual of the Brain Vision Analyzer software (Brain Products GmbH, 2002, p. 82). ERPs with amplitudes exceeding ±50 µV in any of the electrodes were considered as containing artifacts and therefore discarded. For each epoch the full spectrum and maximal resolution option was used to compute the power density. Additionally, a Hanning window of 10% was applied. The power density (µV²/Hz) specifies the average power of each frequency in the EEG signal. It was calculated because it allows the comparison between FFT analyses that have been carried out with different spectral resolutions. The different length of the Go epochs (1024 data points) and the NoGo epochs (2048 data points) required this, because the maximum resolution depends on the length of the epochs. The power densities of the epochs were averaged for each condition, separately.

5.4.3 Results

Figure 24 shows the results of the FFTs for the delta frequency range (0.5-3.5Hz). Graphical inspection shows that the most activity at the Cz-electrode was found in the NoGo task with NoGo signal. Furthermore, the epochs of conditions in which a signal had to be identified contained more delta frequency than epochs of conditions without signals.

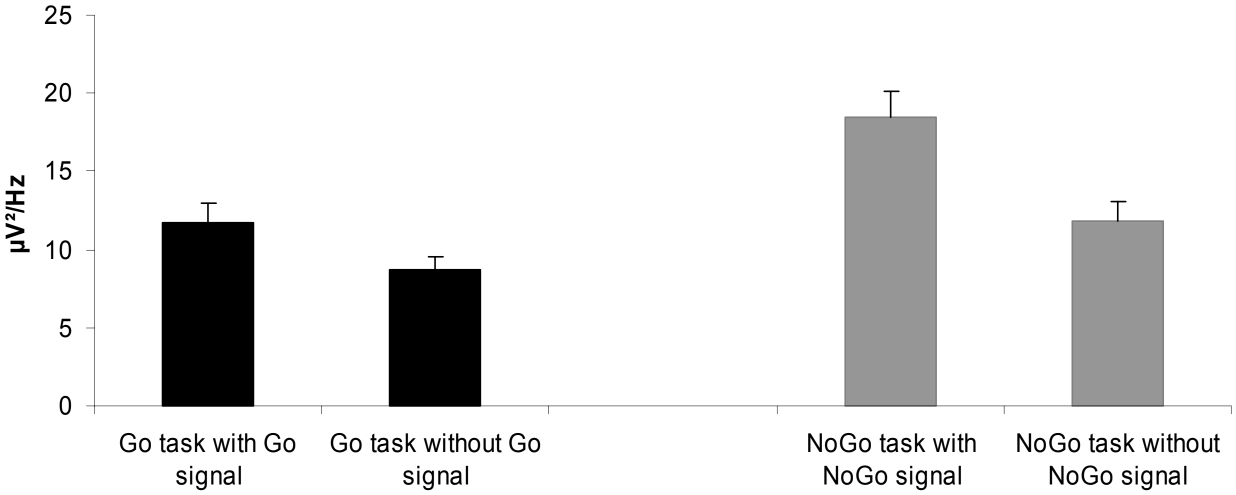

Figure 24. Results of the FFTs in the delta frequency range (0.5-3.5 Hz) for the Go tasks with and without signal and the NoGo tasks with and without signal.

An ANOVA was conducted with repeated measures on the factors condition (Go and NoGo condition) and signal (with signal and without signal) on the amount of delta frequency at the Cz-electrode.

The ANOVA revealed significant main effects of condition, $F(1, 11) = 54.93$, $p < .001$, $\eta^2 = .83$, and signal, $F(1, 11) = 49.21$, $p < .001$, $\eta^2 = .82$. Additionally, there was a significant interaction between condition and signal, $F(1, 11) = 5.55$, $p < .05$, $\eta^2 = .34$.

Additional t-tests showed that the NoGo task with NoGo signal elicited more activity at the Cz-electrode in the delta frequency range than any other condition, ps < .01. The NoGo task without NoGo signal elicited more delta frequency at the Cz-electrode than the Go task without Go signal, $t(11) = 4.94$, $p < .001$, but not significantly more than the Go task with Go signal, $t(11) = 0.15$, $p = .89$. The Go task with Go signal also caused more delta frequency at the Cz-electrode than the Go task without Go signal, $t(11) = 6.22$, $p < .001$.

5.4.4 Discussion

The Fast Fourier Transformation (FFT) was conducted to detect Cz-activity in each trial at any point in time in the NoGo task without NoGo signal. This was necessary because the NoGo task without NoGo signal contained no signal that could act as pacemaker for the inhibitory mechanism. Therefore, it was not clear when inhibition took place. This great variability in the occurrence of the inhibitory mechanism eliminates any connected event-related potential. The FFT results concerning frequencies in the delta range (0.5-3.5 Hz) were of special interest because the P300 arises with similar frequencies.

The FFT revealed a greater amount of frequency in the delta range at the Cz-electrode in the tasks that contain a signal which had to be evaluated than the tasks without signal. This finding supports the idea of Falkenstein, Koshlykova, Kiroj, Hoormann, and Hohnsbein (1995) that activity at the Cz-electrode is connected to stimulus identification. However, the results of the topographical mappings (section 5.2.3) showed that the Go tasks did not differ in topography. Therefore, the activity caused by stimulus identification did not affect topography. Interestingly, the influence of signal presentation on the delta frequency is increased in the NoGo tasks. This finding leads to the question whether the identification of a NoGo signal requires more resources than the identification of a

Go signal. Contrary to this, Falkenstein et al. proposed that the amplitude of the P-SR representing stimulus identification was the same in Go and NoGo tasks.

Moreover, the NoGo task with NoGo signal elicited significantly more activity in the delta frequency range than the Go task with Go signal. Additionally, the NoGo task without NoGo signal elicited significantly more activity in the delta frequency range than the Go task without Go signal. Therefore, the NoGo tasks elicited more activity in the frequency range of the NoGo-P300 than the corresponding Go tasks.

5.5 Validation of the FFT-results

To validate the connection between the results of the frequency analysis and the topographic results, the data of Experiment 2 were analyzed in the same manner as those of Experiment 4. Figure 25 shows the activity in the delta frequency range over the Cz-electrode for the Go (A) and NoGo (B) conditions of Experiment 2. The upper panels of (A) and (B) show the topographies of the P300s.

Graphical inspection suggests that the NoGo conditions elicited more activity in the delta frequency range than the Go conditions. Furthermore, if the centroid of a condition was located more anteriorly than that of another condition, the former condition elicited a greater activity in the delta frequency range than the latter. The different number of Go and NoGo conditions did not allow calculating an ANOVA. However, planned t-tests[19] showed that there was much accordance between activity in the delta frequency range and topography.

The Incongruent_Go condition contained more delta frequency than all other Go conditions, all *p*s < .05 (one-tailed). Furthermore, the NoGo_Go condition elicited more delta frequency than the Congruent_Go condition, $t(15) = 1.82$, $p < .05$ (one-tailed). These results are well in line with the topographical findings (for details see section 3.2.2). However, the NoGo_Go condition did not elicit more delta frequency than the Neutral_Go condition, $t(15) = 0.60$, $p = .28$

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[19] The same t-tests were calculated as for the comparisons between centroids (see section 3.2.2).

(one-tailed), even though the centroid of the NoGo_Go condition was located more anteriorly than that of the Neutral_Go condition.

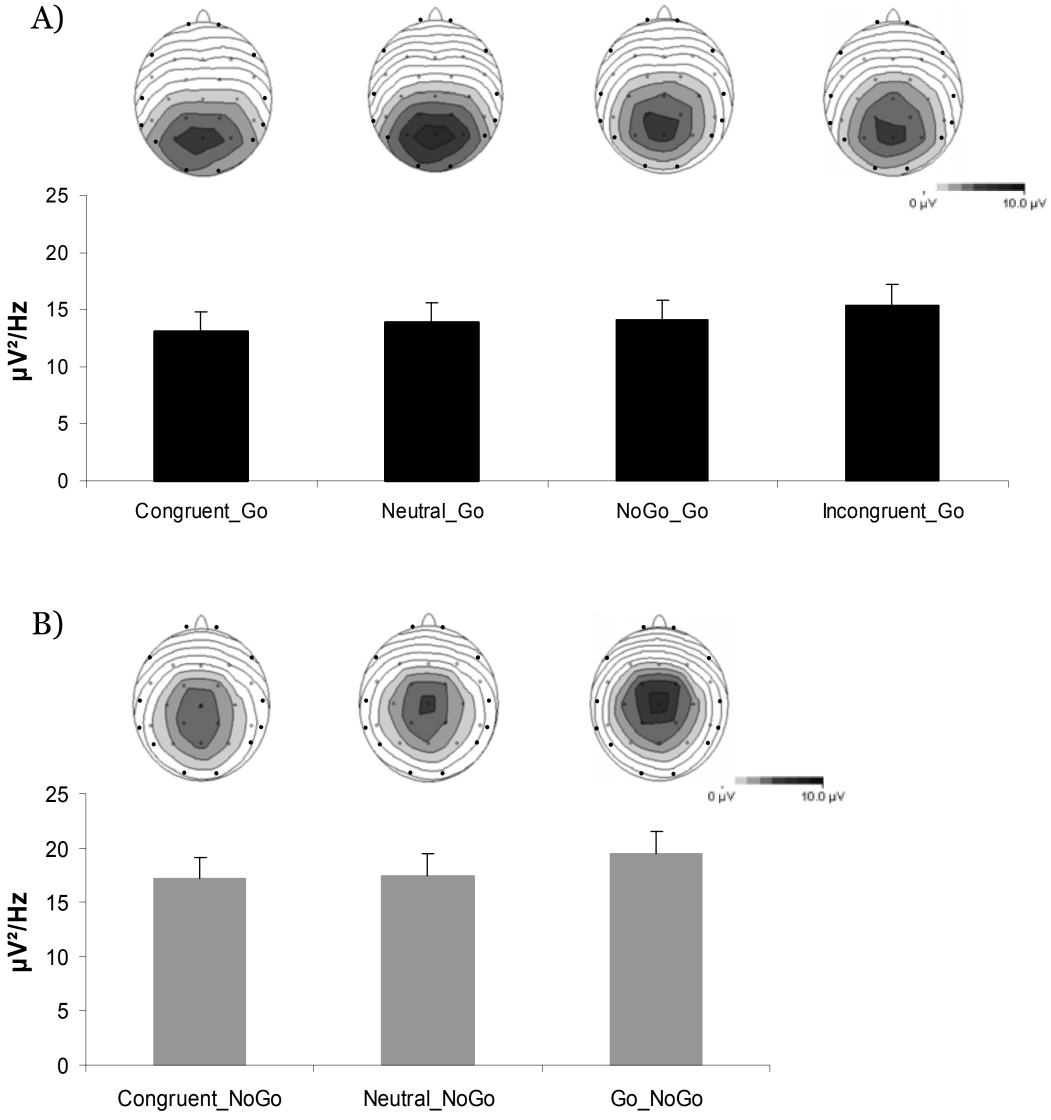

Figure 25. Results of the FFTs in the delta frequency range (0.5-3.5 Hz) for the Go tasks (A) and the NoGo tasks (B) of Experiment 2 and corresponding scalp topographic voltage maps (upper panels).

In the NoGo conditions the amount of delta frequency mirrored the topographical results in the anterior-posterior direction. The Go_NoGo condition elicited more delta activity than the Congruent_NoGo condition, $t(15) = 4.97$, $p < .001$ (one-tailed), and the Neutral_NoGo condition, $t(15) = 3.63$, $p < .001$ (one-tailed). Congruent_Go and Neutral_Go differed neither in the amount of delta frequency, $t(15) = 0.41$, $p = .34$ (one-tailed), nor in topography (see section 3.2.2).

In sum, except for the topographical difference between the NoGo_Go condition and the Neutral_Go condition, the topographical findings are mirrored by significant pairwise comparisons in the delta frequency range. Accordingly, it seems reasonable to assume a connection between P300-topography and amount of delta frequency.

However, the missing topographical difference between the NoGo_Go condition and the Neutral_Go condition has to be explained. One explanation lie in the fact that delta frequency is also elicited in case of more need for stimulus identification (the Go task with Go signal elicited more delta frequency than the Go task without Go signal, see section 5.4.3). Both neutral prime and NoGo prime are different from the Go stimulus, which indicates the required response. Maybe, the stimulus identification of the Go target is hampered by the neutral prime as well as by the NoGo prime and, therefore, in both cases delta frequency is elicited, which covers the difference in delta frequency, which is caused by the NoGo prime.

In conclusion, the result of the FFT analysis in the delta frequency range supports the idea that the NoGo-P300 is connected to an inhibitory mechanism. Each time the inhibitory mechanism is active there is much activity in the P300 frequency range at the Cz-electrode. In contrast, the idea of Falkenstein et al. (1995) that the NoGo-Anteriorization is the result of a smaller event-related potential (P-CR) with a parietal maximum in NoGo trials than in Go trials representing less cognitive response selection in NoGo trials than in Go trials could not be supported.

6 General discussion

The ability to inhibit actions is crucial for human beings. Unfortunately, this ability is difficult to investigate because it is in inhibition's nature to prevent observable behavior. However, event-related potentials (ERPs) offer a possible way of observing underlying processes of inhibition in the brain. The characteristic of one ERP, the NoGo-Anteriorization (NGA; e.g., Fallgatter, Brandeis, & Strik, 1997), has been widely discussed to be connected to inhibition. The NGA describes the topographic difference between Go- and NoGo-P300. However, some researchers proposed alternative explanations for the NGA. These alternatives were tested in four experiments.

The first alternative is that the NGA represents motor potentials during Go trials, which erase the anterior part of the Go-P300 and therefore cause the different topography of the Go- and the NoGo-P300. Experiment 1 and Experiment 2 showed that motor activation through subliminally presented Go primes did not erase the anterior part of the P300 in NoGo trials. Contrary, the prepotent motor activation in NoGo trials caused by the Go primes even resulted in a P300 with a more anteriorly located centroid compared to that of a typical Go-P300. Furthermore, in Experiment 2 NoGo primes that preceded a Go stimulus caused a more anteriorly located P300-centroid in Go trials. In conclusion, the posteriorly located Go-P300 centroid is not the result of a process triggered by Go stimuli. Instead, a process elicited by the NoGo stimuli produces the anteriorly located NoGo-P300 centroid. Therefore, NoGo stimuli instead of Go stimuli trigger a process that causes the NGA.

The second alternative is that the NGA results from higher response conflict and or higher mental effort in NoGo trials than in Go trials. Experiment 3 revealed, however, that the NGA was unaffected by the amount of conflict in Go and NoGo conditions.

The third and final alternative is that the NGA is the result of less cognitive response selection in NoGo trials compared to Go trials. The idea is based on the assumption that the P300 is composed of two event-related potentials (P-SR and P-CR; Falkenstein, Koshlykova, Kiroj, Hoormann, & Hohnsbein, 1995), with the P-SR reflecting stimulus identification and the P-CR reflecting cognitive response selection (Falkenstein et al.). The smaller amplitude of the P-CR in NoGo trials compared to Go trials is supposed to be the reason for the NGA (Falkenstein et al.). However, in Experiment 4, no P300 at all was found in a NoGo condition without exogenous signals that had to be identified. Assuming that this finding was the result of the standard method used for calculating event-related potentials (averaged across trials), an alternative method, a Fast Fourier Transformation (FFT), was employed to analyze brain activity within the P300 frequency-range of the EEG-data (0.5-3.5 Hz). The FFT revealed that there was high activity at the Cz-electrode in NoGo trials in the P300 frequency-range.

Therefore, the four experiments support the assumption that the NGA is connected to an inhibitory mechanism in NoGo conditions. With regard to the nature of this connection one has to consider that the onset of the P300 is too late to mark the start of inhibition (e.g., Falkenstein, Hoormann, & Hohnsbein, 1999). Thus, I suggest that the NGA reflects a late inhibitory mechanism like it is proposed by De Jong, Coles, Logan, and Gratton (1990; see also section 1.4.2). This would be well in line with the idea of Fallgatter, Bartsch, and Herrmann (2002) who assume that the NGA might not be elicited solely by the anterior cingulate cortex (ACC) but reflects activation in inhibitory networks.

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Appendix

Separate ANOVAs were computed with repeated measure on the factors condition (Go and NoGo condition) and signal (with signal and without signal) on the amount of theta, alpha, and beta frequency at the Cz-electrode.

The ANOVA for the amount of theta frequency (see Figure 26) revealed significant main effects of condition, $F(1, 11) = 5.25$, $p < .05$, $\eta^2 = .32$, and signal, $F(1, 11) = 21.86$, $p < .01$, $\eta^2 = .67$. Additionally, there was a significant interaction between condition and signal, $F(1, 11) = 9.36$, $p < .05$, $\eta^2 = .46$. Further analysis revealed that the Go task with Go signal elicited more theta activity than the Go task without Go signal and the NoGo task without NoGo signal, all *p*s < .01. Furthermore, the Go task without Go signal elicited more theta activity than the NoGo task without NoGo signal, $t(11) = 2.93$, $p < .05$, and the NoGo task with NoGo signal elicited more theta activity than the NoGo task without NoGo signal, $t(11) = 5.66$, $p < .001$.

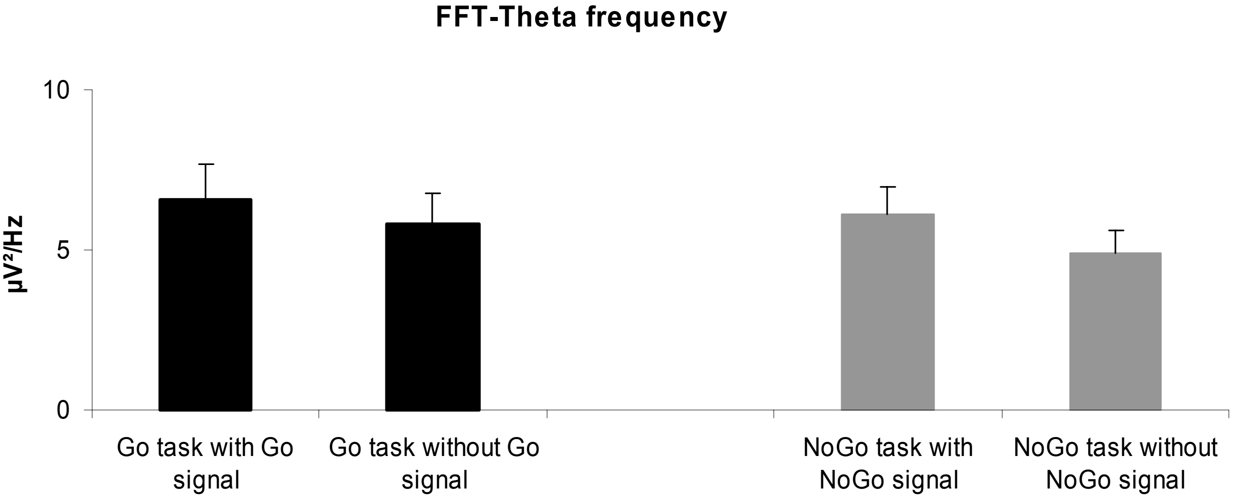

Figure 26. Results of the FFTs in the theta frequency range (3.5-7.5 Hz) for the Go tasks with and without signal and the NoGo tasks with and without signal.

The ANOVA for the amount of alpha frequency (see Figure 27) revealed only a significant main effect of signal, $F(1, 11) = 16.17$, $p < .01$, $\eta^2 = .60$, which resulted from more alpha activity in conditions with signal, all *p*s < .01, while the ANOVA for the amount of beta frequency (Figure 28) revealed neither significant main effects nor a significant interaction between condition and signal.

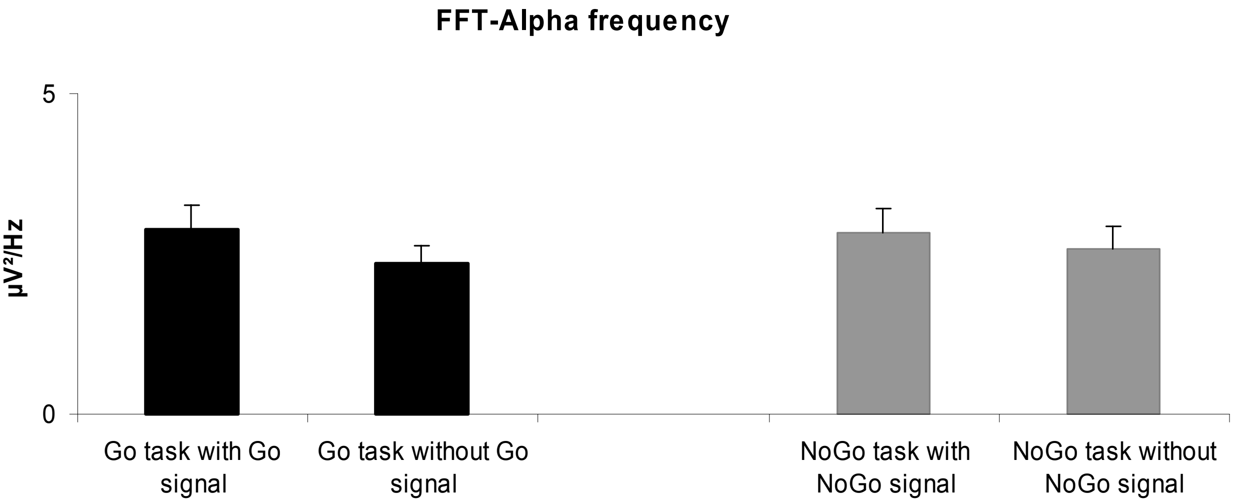

Figure 27. Results of the FFTs in the alpha frequency range (7.5-12.5 Hz) for the Go tasks with and without signal and the NoGo tasks with and without signal.

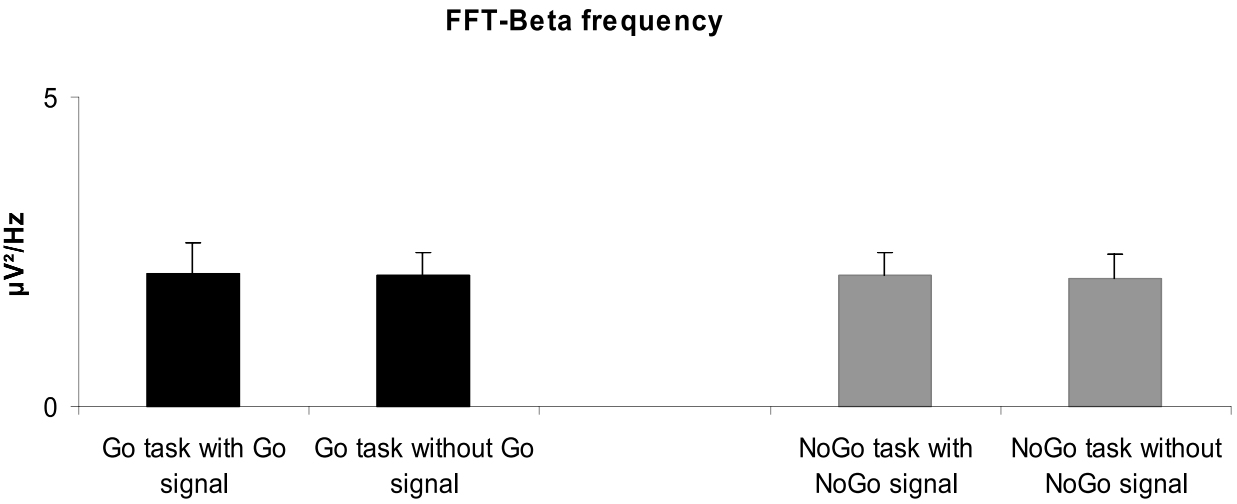

Figure 28. Results of the FFTs in the beta frequency range (12.5-30 Hz) for the Go tasks with and without signal and the NoGo tasks with and without signal.

8 Zusammenfassung

Die Hemmung von bereits geplanten oder initiierten Aktionen ist eine Alltagsleistung. Trotzdem ist es schwierig Hemmung, zu untersuchen, da die der Hemmung zugrunde liegenden Mechanismen nicht direkt beobachtbar sind. Um trotzdem Hemmung untersuchen zu können, wurden Aufgaben entwickelt, von denen angenommen wurde, dass in ihnen Hemmung stattfindet. Eine der bekanntesten Aufgabenarten ist das Stopp-Signal Paradigma. In Aufgaben, die diesem Paradigma entsprechen, sollen Versuchspersonen auf einen bestimmten Reiz hin (dem Go-Signal) eine Taste drücken. In manchen Durchgängen folgt nach dem Go-Signal noch ein weiteres Signal (Stopp-Signal), das anzeigt, dass der bereits initiierte Tastendruck gehemmt werden soll. Logan und Cowan (1984) versuchten, die mit diesem Paradigma gefundenen Befunde mithilfe ihres Horse-Race Modells zu erklären. Das Modell nimmt an, dass sich Antwortprozess und Hemmprozess eine Art Wettstreit liefern. Der Prozess, der zuerst abgeschlossen werden kann, legt die Aktion fest, die ausgeführt wird. Während Logan und Cowan dabei an die Existenz eines einzelnen zentralen Hemmprozesses dachten, vermuteten De Jong, Coles, Logan und Gratton (1990), dass sich die Datenlage nur mithilfe eines zweiten peripheren Hemmmechanismusses erklären ließe. Jedoch wurde die Existenz dieses zweiten Mechanismusses von einigen Forschern bezweifelt (Band & van Boxtel, 1999; van Boxtel, van der Molen, Jennings, & Brunia, 2001). Kapitel 1 der vorliegenden Arbeit berichtet die Befunde des Stopp-Signal-Paradigmas, beschreibt das Horse-Race Modell, diskutiert vermutete Hemmmechanismen und gibt einen Überblick über Hirnregionen, die als Quellen von Hemmmechanismen angenommen werden.

Der zentrale Hemmmechanismus wurde mit zwei Ereigniskorrelierten Potentialen (EKPs) in Zusammenhang gebracht, der N200 und der NoGo-P300. Die N200 ist eine negative Welle, die 200-300 ms nach der Präsentation eines Signals im EEG erscheint (z.B. Kok, 1986). Die Amplitude der N200 ist geringer, wenn eine Antwort ausgeführt wird, als wenn diese Antwort gehemmt wird. Hingegen ist die NoGo-P300 eine positive Welle im EEG, die 300-600 ms nach Darbietung eines Stopp-Signals auftritt (z.B. Pfefferbaum, Ford, Weller & Koppell, 1985). Das Maximum des hirnelektrischen Feldes liegt an der Cz-Elektrode und unterscheidet sich damit von der Topographie, die bei der P300 nach Darbietung eines Go-Reizes gefunden wird (Go-P300). Fallgatter, Brandeis und Strik (1997) bezeichneten diesen topographischen Unterschied als NoGo-Anteriorisierung (NGA), da das Maximum der NoGo-P300 des hirnelektrischen Feldes weiter vorne lokalisiert ist als das der Go-P300. Kapitel 2 gibt einen Überblick über Studien, die die N200 und die NGA untersuchen. Dabei wird das Pro und Kontra bezüglich einer Verbindung zwischen diesen Potentialen und einem Hemmmechanismus diskutiert.

Während für die N200 kürzlich Konfliktüberwachung und / oder mentale Anstrengung als Grund beschrieben wurde (z.B. Botvinick, Cohen & Carter, 2004), gibt es noch immer rege Diskussionen darüber, was die NGA bedingt. Der empirische Teil dieser Arbeit berichtet vier Experimente, die Einwände gegen einen Zusammenhang zwischen NGA und einem Hemmmechanismus ausschließen.

Experiment 1 und Experiment 2 benutzen die Methode des subliminalen Primings, um zu überprüfen, ob negative motorische Potentiale während Go-Aufgaben den vorderen positiven Anteil der P300 auslöschen und so die NGA bedingen. Die Ergebnisse schließen eine solche Möglichkeit aus und zeigen, dass die NGA durch Stimuli ausgelöst wird, die anzeigen, dass eine Hemmung erfolgen soll (NoGo-Stimuli).

Experiment 3 vergleicht die P300s von Go- und NoGo-Aufgaben, die dieselbe Art von Konflikt enthalten. Die Ergebnisse zeigen, dass die NGA nicht durch Konflikt auf der Handlungsebene bedingt ist.

Experiment 4 belegt, dass die NGA nicht daher resultiert, dass die NoGo-P300 stärker durch Prozesse wie Stimulus Identifikation aufgrund von geringerer kognitiver Antwortauswahl in NoGo-Aufgaben beeinflusst ist. Zu diesem Zweck werden die P300s von Go- und NoGo-Aufgaben verglichen, die keinen Stimulus enthalten, der identifiziert werden müsste. Mithilfe der Frequenzanalyse von EEG-Aktivität in Go- und NoGo-Aufgaben kann gezeigt werden, dass in NoGo-Aufgaben verglichen, mit Go-Aufgaben, eine größere Aktivität über der Cz-Elektrode im P300-Frequenzbereich (0.5-3.5 Hz) besteht.

Zusammenfassend lässt sich sagen, dass weder motorische Potentiale in Go-Aufgaben, noch stärkere Antwortkonflikte oder weniger kognitive Antwortauswahl in NoGo-Aufgaben die NGA erklären können. Damit kann die Annahme, dass es einen Zusammenhang zwischen NGA und Hemmmechanismus gibt, nicht falsifiziert werden. Die Arbeit schließt mit einer Zusammenfassung der Ergebnisse.

Lebenslauf

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