Is consciousness required to withhold an impending action? Evidence from event-related brain potentials

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Dedication

In loving memory of

Gareth David Morgan (1961-2002)

Abstract

This thesis explores whether a decision to withhold an impending motor action can be initiated unconsciously. There is much evidence to suggest that both voluntary actions and reactions to external events can be initiated without consciousness. However, there is some debate as to whether inhibition or control of behaviour can occur unconsciously. Libet et al. (1985) proposed that while consciousness is not required to initiate an action, it may be essential in allowing the action to be vetoed. Similarly, evidence from tasks involving response conflict points to a close association between inhibition/control of behaviour and conscious awareness. In particular, both fMRI and EEG correlates of control are seen to be absent when response conflict is unconscious.

The research in this thesis aimed to clarify whether the no-go N2 and P3, ERP correlates of the no-go response, can be modulated by an unconscious prime. In each of five EEG experiments, target-related N2 and P3 components were significantly affected by the nature of the unconscious primes. More specifically, when the unconscious information coded for a no-go response, N2 and P3 amplitude was significantly reduced, suggesting that inhibition of the imminent response was primed by the unconscious stimuli. In addition, there was evidence that the unconscious primes were able to directly engage frontal inhibition/control mechanisms. In experiments 1, 2 and 5 early ERP differences were observed over frontocentral electrodes that were entirely dependent on the nature of the unconscious prime. Furthermore, experiment 5 showed that this early modulation of ERP activity was directly related to the extent to which the participants were influenced by the unconscious primes. These findings suggest that inhibition of an impending motor action can be initiated by an unconscious stimulus. These conclusions are discussed in relation to previous research, and the possible role of consciousness in behaviour. The limitations of the current research and suggestions for future work are also considered.

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List of Abbreviations

ACC	Anterior Cingulate Cortex
ADAN	Anterior Directing Attention Negativity
ANOVA	Analysis of Variance
EDAN	Early Directing Attention Negativity
EEG	Electroencephalography
ERP	Event-Related Potential
fMRI	Functional Magnetic Resonance Imagery
LRP	Lateralised Readiness Potential
LDAP	Late Directing Attention Positivity
MEG	Magnetoencephalography
ms	Milliseconds
μV	Microvolt
NCE	Negative Compatibility Effect
PCE	Positive Compatibility Effect
PFC	Pre-frontal Cortex
RCE	Response Congruency Effect
RP	Readiness Potential
RT	Reaction Time
S-R	Stimulus-Response
SMA	Supplementary Motor Area

Chapter 1

General Introduction

Outline

These first three chapters will give an introduction to the research question addressed in this thesis. The first chapter provides a theoretical background for the research presented in this thesis, focusing on the question of the role of consciousness in control of behaviour. The second chapter will provide a more detailed introduction to EEG methodology and in particular provide a background to ERP correlates of motor preparation and inhibition. The final chapter of this introduction will review experimental work on the ability of unconscious stimuli to influence preparation and inhibition of a motor response.

Conscious Free Will

The experience that our conscious mind influences our actions through the expression of free will was most famously put forward by Rene Descartes (1596-1650; cf. Velmans 2000). He proposed a separation between substances extended in space (*res extensa*) and a fundamentally different substance, which thinks (*res cogitans*). He suggested that sensory stimulation leads to perceptions in the soul by way of movements in the pineal gland. Conversely, free will is exercised by the soul through nerve cells from the pineal gland to the muscles. In this explanation the non-physical mind and the physical brain were assumed to be different substances that interacted via the pineal gland in the brain. Eccles (1980) led a group of modern philosophers who share this view that the non-physical mind influences the physical world through the expression of conscious will. Despite the many philosophical problems with such a perspective (see Velmans, 2000), the folk psychological notion of free will is perhaps still largely informed by the notion that our conscious minds directly controls our behaviour in the world. As Velmans (2002) suggests, in our everyday life we take it for granted that we have control of our actions and

that it is the conscious mind that exercises this control. However a series of experiments conducted by Libet and colleagues began to question the notion that the conscious decisions are the causes of one's actions.

Libet's initial experiments focused on the neural conditions necessary to produce a conscious experience (see Libet 2003b). Libet, Alberts, Wright, Delattre, Levin and Feinstein (1964) showed that a minimum of 500ms of direct electrical stimulation of the somatosensory cortex (at a liminal intensity) was required to produce a conscious experience. They showed that when the later part of this processing is disturbed either by anaesthetics or presentation of a second stimulus (retroactive masking), a participant is no longer able to consciously report the sensation (Libet, Alberts, Wright and Feinstein, 1967). Retroactive masking consists of presenting a second slightly stronger stimulus in order to mask the experience of the first stimulus. Libet et al. (1967) were able to show that using an inter stimulus interval (ISI) of up to 100ms was successful in masking the conscious experience of the initial stimulus. These findings together suggest that incoming sensory information requires a period of unconscious brain processing (between 100 and 500 ms) before it reaches consciousness.

Libet et al. (1983) then investigated whether the same period of "neural adequacy" was required to experience a decision to initiate a motor act. Based on Kornhuber and Decke's (1965) observation that a 'readiness potential' (RP) is recordable on the scalp prior to voluntary action, Libet et al. (1983) were interested in where, in relation to RP onset, one becomes aware of the urge to move. This would allow comparison of the time at which individuals were conscious of preparing to move in comparison to when the brain begins preparation to move. The RP was recorded with an electrode placed on the vertex (over the motor cortex) on the scalp (see Chapter 2 for further information regarding the RP). Participants were asked to flex their wrist spontaneously (without pre-planning), whilst at the same time watching a clock face revolve on a ray oscilloscope at the rate of one revolution per 2.56 seconds. After their action they were then asked to retrospectively report where the clock hand was at the time that they felt the wish to perform

the action. Libet termed this the W judgement for want, the time of the appearance of conscious awareness of wanting to flex one's wrist. This judgement was also described as an 'intention' to act, a 'decision' to move or an 'urge' by some participants. In addition to the W judgements, Libet asked the same participants to report the time at which they performed the action (M judgements) as well as the time of a near threshold stimulus to the skin (S judgements). These three different types of judgements were performed in separate blocks, with practice trials on the particular judgements preceeding each block. The S judgement was to be used as a control task to check the accuracy of the clock reporting method and was found to produce accurate reported times of the stimulation with around 50ms of error.

When comparing the average time of W judgements to the onset of the RP, Libet et al. (1983) found that for unplanned acts the RP preceded the wish to act by 350ms. The remarkable conclusion from such an investigation was that even so called freely initiated acts are not actually instigated by consciousness, but rather are initiated unconsciously. This supports and extends Libet's previous findings that a period of neural processing in the brain is required for the appearance of the conscious experience associated with that processing. There is also some independent evidence that activity in the supplementary motor area (SMA), from where the RP is thought to originate, is causally involved in the experience of the urge to move. Fried, Katz, McCarthy, Sass Williamson, Spencer and Spencer (1991) inserted electrodes on to the SMA during evaluation for surgical treatment of patients with intractable epilepsy. Their primary interest was to explore the somatotopic organisation of this area. However, they also noted that direct stimulation some parts of the SMA produced both experiences of movements in the absence of actual movements and experiences of the urge to move. In addition, at some of these sites where an "urge" was reported in response to light stimulation, further electrical stimulation of the same site was sufficient in producing a movement (although often in a slightly different location to where the urge had been reported). This evidence supports Libet's conclusion that the activity recorded by the RP is the unconscious antecedent of the subjects conscious experience of wanting to move.

In one of only two replications of Libet's experiment Haggard and Eimer (1999) used an identical methodology to that employed by Libet et al. (1983), with the exception that they varied the response hand in order to allow calculation of the lateralised readiness potential (LRP). The LRP is calculated by measuring the activity over the primary motor cortex (1 cm anterior to electrode points C3 and C4), and subtracting activity from the hemisphere ipsilateral to the response from activity of the hemisphere contralateral to the response hand (see chapter 2 for more details). This will result in a marker of motor preparation of movement of a particular side of the body and is thought to better reflect the onset of specific motor programming. Haggard and Eimer (1999) found that the onset of the LRP occurred approximately 300 ms prior to electromyogram (EMG) onset, with the wish to act occurring around 200ms prior to action. Furthermore, they found concomitant variation between the LRP and the judgement of the time of the wish, such that early LRP onset was associated with an earlier wish judgement (and vice versa). Such concomitant variation, that was not evident with RP onset, strongly suggests a causal relationship between the LRP and the wish to perform an act. This provides strong support for Libet's (1982) conclusion that motor acts are initiated unconsciously and suggests that the LRP is causally involved in producing the conscious wish to act.

Trevena and Miller (2002) have argued that the calculation of the RP and LRP is subject to a smoothing bias in which the onset is disproportionately shifted towards the onset in the earliest trials. This would result in an LRP onset that reflects the earliest onset time for all subjects, rather than the average onset time. They collected new data using the same methodology as that employed by Libet and examined the effect of this calculation bias on Libet's findings. To counteract the bias in LRP and RP calculation they compared the onset of the two components in relation to the earliest reported wishes. They found that none of the wishes occurred prior to RP onset and thus concluded that RP onset consistently occurred prior to the conscious wish to act. However, when examining LRP onset in relation to the W judgement they found that in 20% of trials the wish to act occurred prior to

the LRP onset. Since the LRP onset is smoothed towards the earliest LRP onsets Trevena and Miller conclude that "the LRP always started after the conscious decision to move". Regardless of this, the finding that RP onset still occurred significantly earlier that the W judgement provides further evidence that non hand specific motor preparation is initiated prior to the conscious desire to act now.

There have been countless commentaries and reanalyses of Libet's data, including a special edition of consciousness and cognition (2002; issue 11) and commentaries to his 1985 behavioural and brain sciences target article (Libet, 1985). A number of authors have questioned whether it is possible to introspect on ones internal mental states in the manner described by Libet (e.g. Rugg, 1985; Vanderwolf, 1985) and even that the instructions to participants may have resulted in the experience of an intention immediately prior to action that does not occur in natural voluntary movements (Ringo, 1985). There has also been much criticism of the clock method used to measure the time of conscious awareness of wanting to act, suggesting that its use in retrospectively reporting the conscious decision to act is subject to a number of different biases (Breitmeyer, 1985; Rollman, 1985).

Pockett (2002) provides a complete reinterpretation of Libets data, suggesting that rather than taking 500ms for a conscious experience to be generated in the brain, it takes around 80ms. She suggests that the reason for Libet's conclusion is that he used electrical stimulation at liminal intensity and that facilitation of the signal was in fact responsible for his findings. Pockett also applies this logic to Libet's findings that when asked to report their S and M judgements, there appears to be an error of around 50 – 85ms. She suggests that if it takes 80ms for stimuli to reach awareness then their experience of the clock hand at position *x* would actually correspond to *x* minus 80ms. Therefore they experienced moving when they did move, but because of the delay of experiencing the clock, they reported moving at *x* minus 80ms. This reinterpretation thus not only supports the shorter period needed for conscious awareness, but also suggests that reporting of experience using the clock method is subject to error of around 80ms, such

that events would be judged as occurring 80 ms before they actually occurred. This would imply that the time of the decision to perform an act was also judged to have occurred earlier than it really did, thus giving even more weight to the claim that the RP precedes the conscious wish.

Van De Grind (2002) suggests that in fact this error may be the opposite way round. He reviews evidence of the flash lag effect (MacKay, 1958) in which a switch of attention results in a delay of around 80–100ms in reporting the location of a moving object at a specific point in time. He suggests that in Libet's experiment, since initial attention is focused on the clock and participants are told to look out for conscious awareness of the decision, there will be a delay in the reporting of the conscious decision to act. Thus he suggests the bias is actually likely to shift the conscious decision 80 ms later than it truly occured. Gomes (2002) also points out that the use of feedback to participants regarding their S judgements during training makes any interpretation of the findings very difficult, since the effect of any attempted correction of the bias is difficult to discern. He also suggests that when attributing awareness to act we usually only have a unitary experience of wanting to act, and do not judge the wish and the actual time of action separately. Therefore the instructions to participants to distinguish between these two events, and the suggested order of them, will also influence their relative latencies. Instead he suggests that the occurrence of the RP so far in advance of action (-500ms) contrasts sharply with the fact that we feel a wish occurring *immediately* prior to an action, and that this is adequate to conclude that motor acts are initiated unconsciously.

Conscious control of action

Despite some severe methodological problems with the original Libet experiments, the general conclusion that voluntary acts appear to be initiated before the conscious urge to act appears, is strongly supported by the research discussed above. More recent research combining masked priming with EEG has also reached a similar conclusion, that a stimulus that remains unconscious directly initiates preparation for action (Dehaene et al., 1998;

Eimer & Schlaghecken, 1998; Leuthold & Kopp, 1998). These experiments will be discussed in more detail in chapter 3. Despite these conclusions, rather than concluding that consciousness is not the cause of behaviour, Libet has suggested that consciousness is able to veto an unconsciously initiated action (Libet et al. 1983, Libet 1985, Libet, 2003). Libet et al. (1983) conclude by suggesting that "there could be a conscious 'veto' that aborts the performance of 'spontaneous' self-initiated acts" (page 641). In support of this claim, they point out that some subjects reported having felt an urge to act but that they were inhibited before the action could occur.

There have been a number of objections to the idea of conscious veto on theoretical grounds. Danto (1985) for example says Libet's suggestion that we can consciously veto our unconsciously initiated action is "incompatible with everything he had up to that point been at pains to show" (Danto, 1985, page 541). He goes on to argue that this claim seems remarkable in light of his earlier work which showed that conscious awareness of both internally generated (e.g. wish to act; Libet et al., 1982) and externally generated (e.g. tactile stimuli; Libet et al., 1967) events are subject to a minimum of around 100 - 500 ms of neuronal adequacy. The natural question then to ask is does a veto decision have its own unconscious brain correlate? A number of commentators (e.g. Danto, 1985; Merickle & Cheesman, 1985) in Libet's (1985) target article suggest precisely this; that any conscious veto is likely to have its own unconscious physiological correlate, that begins well in advance of the actual experience of the veto. Libet (1985) responds by simply suggesting that there is presently no evidence against such a control function acting consciously. In response to a similar criticism from Velmans (2002), Libet (2003a) suggests that unconscious processes leading to the conscious urge to act (the RP) may include content of the factors that affect a veto decision and that no new unconscious processing is required for the conscious veto to be applied. However, Libet (1985) also describes this conscious control function as "evaluating" and "deciding" on whether to veto that action as well as suggesting that vetoes often occur when the initial urge to act involves some socially unacceptable consequence (Libet, 2003b). As Velmans (2003) points out this leaves something of a strange asymmetry

between a "conscious" decision to act and a "conscious" veto, such that the relatively simple decision to perform a motor action requires some 350ms of unconscious neural activity, a conscious veto occurs in just 150ms. This despite the fact that the veto would likely involve access to long term memory for assessing possible consequences of action, comparison with the need to perform the current action in reference to ones current goals, which is possibly a more complex decision than the initiation of the act itself.

Libet often states that the "existence of a veto possibility is not in doubt" (Libet, 2003b, page 141). He suggests that we often feel urges to do something and then for various reasons consciously decide not to do it. However, simply because we *feel* that we consciously veto a decision this does not mean that consciousness has initiated the veto (Velmans, 2003). As discussed above, Libet's own research has shown that "voluntary" movements are initiated unconsciously despite the fact that people feel the acts to be consciously initiated, so to take these experiences as fact in the case of the veto seems to go against the main implications of his research.

Libet, Wright and Gleason (1983) attempted to explore the possibility of the conscious veto in more detail by asking subjects to veto a voluntarily initiated act. However, due to technical restraints when averaging trials together for EEG exploring the veto is rather difficult. Due to the poor signal to noise ratio of the RP, this EEG component is not visible on single trials. It is therefore necessary to average together a large number of trials for the RP to become visible. In Libet et al. (1983) trials were averaged together with respect to the onset of the muscle movement. However, in the absence of a muscle movement, or any other cue as to when the action would have been performed (in a veto condition) it is impossible to know when the RP should occur and thus form an appropriate average. Libet et al, (1983) were therefore forced to create a rather unusual situation where participants were instructed to pre-prepare a movement when the clock reached a particular point, and then to veto this action 150-200ms before its execution. In some blocks (M series), subjects were instructed to perform the action at the pre-specified time, whilst in other blocks (M-veto series) subjects were to veto

the action at the final moment. The use of this method allowed averaging of the EEG with respect to the time at which the action had been pre-specified and thus allowed calculation of RPs in the absence of overt motor responses.

Libet et al (1983) found that the RPs under these two conditions were remarkably similar until around 150ms prior to the point at which the action was to be performed. Libet concludes that this RP might reflect general "non consummated urges to act" (Libet et al. 1983, page 371) and that the final decision to act now would be controlled by the conscious veto. Libet (1985) admits that this is a limited test of the veto; however it is worth noting some recent research which might provide an alternative explanation to his findings. It has become increasingly evident that activation in the motor system occurs not only when performing an action but also when observing actions (Gallese, Fadiga, Fogassi & Rizzolatti, 1996; Fadiga, Craighero & Olivier, 2005) and imagining actions (Galdo-Alvarez & Carrillo-de-la-Pena, 2004). When observing or imagining an action our "mirror-neuron" system stimulates the exact movements below the threshold which would cause us to perform the action (Fadiga et al., 2005). Of particular note to Libet's findings, Galdo-Alvarez and Carrillo-de-la-Pena (2004) showed that imagining hand movements produced LRPs of a similar onset to actual movements but with lower amplitude. This further confirms that as well as controlling actions, the motor system represents imagined and observed actions, and that these representations are reflected in LRPs. Under such an explanation the RP observed for M-veto series in Libet et al.'s (1983) experiment would simply reflect a representation of the action which they were instructed to prepare for but not actually perform. It is difficult to tell from Libet's data whether the amplitude of the RPs was smaller for M-veto compared to M series trials but it would appear to be, at least in some of the subjects (he provides no statistical analysis of this). Furthermore, the failure to find a difference between RP's recorded at the vertex does not rule out the possibility that a veto may have unconscious antecedents that occur elsewhere in the brain.

Unconscious control triggered by an external stimulus

The research presented in this thesis will attempt to further explore the question of whether a decision to inhibit (veto) an impending action requires consciousness. Given the problems with examining the conscious veto using Libet's paradigm a different experimental approach will be employed. The relation between conscious awareness and preparation or inhibition of action will be explored using a combination of go/no-go tasks and masked priming. The onset of electrical activity associated with preparation/inhibition of action will be established by instructing participants to proceed or withhold an action upon presentation of different types of stimuli. The use of masked priming will allow exploration of the effects of an unconscious stimulus on participants' responses to the relevant target stimulus. These techniques and the ERP components associated with them will be the focus of chapters 2 and 3.

It is important to note that some might argue that such responses to external stimuli can not be relevant to free will, since they do not reflect voluntary processes. Patrick Haggard (2001) for example, suggests that when studying the psychology of action one should only use action initiated internally since action prompted by external events lack the psychological components of generation of an action. Haggard and Clark (2003) point to evidence that the brain networks involved in performing a self-initiated act only partially overlap with those areas involved in responding to an external stimulus. More specifically, while self-generated action shows activation in the basal ganglia and SMA, externally triggered actions are associated with activation of the cerebellum and premotor cortex before the two types of action converge in the primary motor cortex. Whilst it seems clear that there are differences between actions triggered by external stimuli and internally generated actions, it is not clear how exactly this related to the question of intention and free will. For example, Jahanshahi, Jenkins, Brown, Marsden, Passingham and Brooks (1995) compared movements where subjects raised their right index finger whenever they wished to do so, with trials where subjects moved the same finger in response to a tone. While the authors

themselves do not make any claims with regard to the intention of the subjects during these movements, Haggard and Clark (2003) make it clear that whilst the self-initiated movements are intentional and voluntary, the reactions to the stimuli are unintentional and involuntary. Mele (2004) argues that both of these acts are clearly intentional. He suggests that the confusion stems from the fact that Haggard and Clark believe that in *deciding* to perform the act at a specific time, this makes their action intended, and without such a decision the act can not be intentional. However, as Mele (2004) argues, we often act intentionally without having explicitly made a decision to act, such as when I unlock my office door in the morning; I clearly intend to open my door and enter my office but I did not explicitly form a decision to do so.

Furthermore, Bennett and Hacker (2003) point out that Libet's conception of free will is somewhat confused. They suggest that in reality not only are voluntary acts not preceded by an urge or intention to act, but also that when interacting with the outside world we commonly act in reaction to events around us, but this does not make those actions any more 'automatic' or less under the influence of free will than internally initiated actions. They suggest (as others have, see above) that the instructions to introspect on an intention prior to an action may have caused such an experience to have occurred when it otherwise would have no role in the everyday sequence of events leading to action. They argue that "the most interesting results of these experiments is that people, when asked to report such bizarre things as 'a feeling of intention to move one's hand', one will find such a feeling to report, even though it is more than a little doubtful whether there is any such thing" (Page 230).

Keller and Heckhausen (1990) present some data to support the suggestion that the instruction to introspect on an action was responsible for making a normally unconscious process appear in consciousness. They recorded RPs in response to both conscious and unconscious movements. Unconscious movements were those in which subjects were not asked to introspect on their urge to move and reported no such urge prior to movement. Keller and

Heckhausen (1990) found that unconscious movements produced RPs with a similar latency to conscious movements. While RP amplitude was greater for movements where subjects were asked to look out for the urge to move, the authors suggest that this increased activity reflects utilisation of the supplementary motor area following conscious detection of an unconsciously initiated movement. They suggest that movements were initially unconsciously initiated in response to muscular tension and then only entered consciousness when participants were looking out for the urge to move. In this sense they argue that these movements are internally triggered making them rather similar to externally triggered actions observed in reaction time experiments.

In addition to these criticisms of the purported presence of an intention or a decision prior to a freely initiated act, it is fair to say that the experimental paradigm designed by Libet et al. (1983), Haggard and Eimer (1999) and Trevena and Miller (2002) actually consists of very little free choice. The specific action is fixed and the hand of action is fixed, the only thing left open to choice is when to act. Perhaps the most parsimonious strategy would be to forget talk of 'willed action' and 'intention' and simply to focus on how an individual acts in a given situation, in response to either internal or external cues, and whether it is necessary that these cues are conscious, or if they may be influenced without consciousness. However, it is still important to note that the actions performed in the current series of experiments are very different to those explored by Libet et al. (1983), and the conclusions and generalisations of these results should be limited to only these types of scenarios.

Limitations to the current research program

In addition to the limits of the generalisability of the results to externally triggered actions discussed above, it is also important to note that the research in this thesis does not provide evidence on a number of other suggestions that have been made in relation to the role of consciousness in behaviour. For example, Gomes (1998) suggests that although the veto may

also have preconscious neural processing of its own, it is in a sense controlled by consciousness since even any preconscious processing of the veto can only be initiated after conscious awareness of the decision. Under such an interpretation a 'veto' of an action could not occur unless the initial intention had become conscious.

Similarly, a number of authors (Mele, 2006; Gallagher, 2006; Pacherie, 2006; Searle, 1997) have made a distinction between future directed intentions (distal intentions in Mele's or prior intentions in Searle's terminology) and proximal intentions (or intentions in action). Future directed might be something like "I intend to go to the beach this afternoon", while a proximal intention is involved with the actual movements I produce when the time comes for me to go to the beach. In a similar framework to this, Zhu (2003) claims that all of Libet's participants had formed a prior conscious intention to perform the specific actions at the beginning of the experiment. Libet (1985) is sensitive to this point, but suggests that "without an overt motor performance any volitional deliberation....does not constitute *voluntary action*" (page 530). Gallagher (2006) argues, however, that such an argument misses the point of the larger framework in which the action takes place. Whilst he agrees that the movement and the control of the movement is intimately connected with action, actions themselves are specified in relation to the goals of those actions and not in the precise movements one has to make in order to achieve those goals. The current research will not address this debate directly, but rather, in a similar sense to Libet's research will focus on the process of performing (or vetoing) an action, and the role of consciousness in that process.

Summary

Research by Benjamin Libet and Colleagues has found that an internally triggered action is initiated in the brain 350 ms before we become conscious of the urge to perform this action. Despite a number of criticisms of the methodology employed in these experiments, the conclusion that a motor action is initiated unconsciously appears to be supported by the vast majority

of evidence. However, Libet attempts to salvage a causal role for consciousness by suggesting that we might still be able to consciously veto the unconsciously initiated action. Whilst there are a number of theoretical problems with this position, there is no evidence to bear on the issue of whether a veto is always conscious. The research presented here will attempt to provide evidence on the question of whether consciousness is required to inhibit an impending action. This will be assessed using responses to external stimuli, whilst manipulating conscious awareness of these stimuli. Whilst this approach is rather different to that employed by Libet and others, it will provide useful insight into the possible functional role of consciousness in control of behaviour.

Chapter 2

Introduction to Event-Related Brain Potentials

Outline

This chapter will provide a more detailed introduction to Electroencephalography (EEG) and Event-Related Potentials (ERPs) with particular focus on ERP correlates of motor preparation and inhibition. This will include a short introduction to the source of the EEG signal and derivation of ERPs and a literature review of studies exploring ERP correlates of motor behaviour, as well as other ERP components of interest for the current thesis.

Electroencephalography and Event-Related Potentials

Hans Berger (1929) was the first to report that electrodes placed on the scalp were able to pick up electrical activity from the human brain (cf. Luck, 2005). Although this discovery was initially rejected as an artefact from muscle movements, it soon became increasingly clear that EEG could provide a "window on the mind" (Nunez & Srinivasan, 2006, page v). EEG signals recorded at the scalp are a direct measure of activity of a large number of cortical neurons unlike hemodynamic measures such as fMRI which indirectly measure brain activity through measuring levels of oxygenated blood in the brain (Otten & Rugg, 2004). In order for electrical activity of neurons to be detected at the scalp a large population of neurons must be synchronously activated (Ward, 2006). In addition this population of neurons must be aligned so that they summate rather than simply cancelling each other out (Ward, 2006). However, due to volume conductance of the signal from many different cortical sources to the scalp the resulting EEG signal is a conglomeration of many different neuronal sources of activity. The signal recorded from a single scalp electrodes contains synaptic activation from between 100 million and 1 billion neurons (Nunez & Srinivasan, 2006). Despite this it is possible to extract activity associated with cognitive, sensory

and motor events from this signal by means of simple averaging of the signal (Luck, 2005).

Event-related Potentials (ERPs) are fluctuations in EEG voltage associated with some physical or mental event (Picton, Bentin, Berg, Donchin, Hillyard, Johnson, Miller, Ritter, Ruchkin, Rugg and Taylor, 2000). ERPs are generated by averaging a large number of trials under a particular condition with reference to the onset of a stimulus or a response. The logic for this method is that whilst some of the activity in the EEG is associated with the current task (for example reading, memorising, preparing a movement), a large amount of the signal will reflect spontaneous activity of neurons that do not relate to the task (Ward, 2006). By averaging together many trials where the participant is performing a specific task it is possible to isolate the activity that is related only to that task, under the assumptions that spontaneous EEG changes will vary from one trial to the next and therefore cancel out. ERP waveforms are always presented relative to a baseline period. Normally an ERP is averaged relative to the onset of a particular stimulus (stimulus-locked ERPs). In this case the period immediately prior to the stimulus onset is usually used as a baseline. This baseline correction is applied to remove any artifactual differences (such as slow shift in the EEG) between conditions so that at the period that the stimulus appears, the ERP amplitude is the same for all conditions. ERP waveforms will therefore normally be at around zero amplitude in the baseline period and will then show a response to the stimulus. It is important to note that baseline correction procedures may actually introduce artefacts to the data if pre stimulus differences reflect differences due to the experimental manipulation. A simple way of overcoming such a problem is to have different conditions appear in a random order such that the participant will not know the condition until the stimulus has appeared. However, this is not possible when exploring ERP responses based in subsequent self grouping of trials, for example when asking participants to rate their ability to perceive a particular stimulus. In such a situation pre-stimulus factors, such as attention and arousal may predict subsequent perception of the stimulus (Hanslmayr et

al., 2007) and therefore the pre-stimulus baseline EEG will likely be influenced by such processes.

In addition, EEG is always recorded relative to a particular reference point to give a relative value of the electrical signal. EEG is typically recorded relative to the mastoids (the bone behind the ear), the earlobes or the tip of the nose. If a large number of electrodes are used (typically at least 64) EEG may also be referenced to an average of all the scalp electrodes. It is possible to re-reference EEG data to a different location off line once the recording is complete. If comparing results from one experiment to another it is normally advised to use the same reference where possible (Picton et al., 2000) since the choice of reference will dramatically influence the topography of the ERP effect.

In the 1970s and 80s a great number of ERP papers were published which outlined 'components' associated with different cognitive functions (Luck, 2005). While there is no universally agreed definition of a component, they are often defined both with reference to their functional significance and their proposed underlying neural sources (Otten & Rugg, 2004). Components are normally described in terms of their specific scalp distribution and in terms of their relationship to experimental variables. However the ERP waveform represents the sum of a number of underlying components indexing different cognitive processes involved in completing a particular task, therefore a particular peak or trough in the raw ERP waveform is not a pure measure of a particular component (Luck, 2005). For this reason ERP components are often defined as differences between particular experimental conditions rather than in terms of particular parts of the raw ERP waveform (Luck, 2005; Otten and Rugg, 2004). The no-go N2, for example, discussed in more detail below, is defined as a negative deflection for no-go trials in comparison to go trials occurring around 200ms after stimulus onset. By carefully designing an ERP experiment such that only the process that you are interested in exploring differs between conditions it is possible to isolate components associated with particular cognitive processes despite the fact

that the raw ERP contains a combination of peaks and troughs which do not normally relate to a particular cognitive event.

Perhaps the earliest component to be defined was the contingent negative variation (CNV), a negative shift observed prior to a stimulus to which a subject must respond which was absent when passively viewing the stimuli (Walter et al., 1964; cf. Luck, 2005). The presence of this component only when a response was required led to the suggestion that the CNV was associated with preparation or anticipation for the need to make a response. A number of other components have since been identified associated with many cognitive and motor functions, some of which will be discussed in more detail in the following sections of this chapter.

A major strength of EEG and ERPs is that they provide information on cognitive processing with a temporal resolution of milliseconds (Nunez & Srinivasan, 2006; Luck, 2005; Otten & Rugg, 2004). This allows for the measurement of continuous changes over time as opposed to just a single reaction time measure (Ward, 2006), as well as for determining the locus of an effect. For example, by determining which ERP component is affected by a particular manipulation you can determine *how* it is influencing the participants' performance of the task. Similarly, by manipulating the response hand dependent on a particular stimulus dimension you can determine the order in which different aspects of a stimulus is processed by monitoring how it affects the latency of ERP components associated with preparation for a motor response. This will be discussed in more detail later with particular reference to the lateralised readiness potential.

However, perhaps the greatest limitation of EEG is the poor spatial resolution which it affords. Due to the spatial averaging of signals from volume conductance of activity from many different areas of the brain, it is not easy to localise the source of an ERP component (Otten & Rugg, 2004). While methods exist for estimating the sources of particular ERP signals these remain simply estimates. Although a specific source configuration produces a particular scalp topography, the converse is not the case. There are almost

an infinite number of possible combinations of generators that could lead to a particular effect observed at the scalp (Slotnick, 2004). It is possible to constrain the solution in ways that depend on prior empirical knowledge, for example knowledge gained from more spatially accurate techniques such as fMRI and single unit recordings can guide predictions of possible sources of ERP signals (cf. Slotnick, 2004). However it remains the case that the strength of ERPs is the temporal resolution they afford and not their spatial resolution.

An ERP waveform at a single electrode is normally represented graphically, with the x axis usually signifying time in milliseconds (ms) and the y axis reflecting amplitude in microvolts (μV). A vertical line intersecting the x axis is normally presented to show the time of stimulus onset. Waveforms can be plotted with either positive or negative potentials pointing upwards, and there is no general consensus as to which is preferable (Picton et al., 2000). In addition to the single waveforms, a topographic distribution is often presented to show how the activity presented in the waveforms is distributed across the scalp. This is important as it enables one to demonstrate that a component has a similar distribution to that which has previously been described in the literature (Picton et al., 2000). Electrodes are normally fixed and specified in terms of the international 10-20 electrode system where central electrodes are prefixed with the letter C, parietal with P, frontal with F etc. and all left hemisphere electrodes are designated odd numbers (with 1 closest to the centre) and right hemisphere electrodes designated even numbers (cf. Picton et al., 2000). For example an electrode placed over a left central site would be named C1 or C3, depending on its distance to the centre. As well as single electrode waveforms, it is also common for difference waveforms to be presented, which can either represent a difference between conditions, or a difference between electrodes (see LRP and N2 below). Difference waveforms can be a good way of isolating components associated with particular cognitive processes (Luck, 2005) by showing which ERP measures are sensitive to particular changes in the experimental design.

ERP components

The following section will provide a literature review of the ERP components of interest for this thesis. After a brief overview of the early visual components and the P300 component, the main focus of this section will be the ERP correlates of motor preparation and inhibition which will be used to asses the ability of unconscious information to initiate inhibition of a motor response.

Visual and attention related ERP components

The early ERP components measured over posterior electrodes are normally considered to reflect the primary visual response to the presentation a stimulus (L. Wang, Kuroiwa, Li, Wang, & Kamitani, 2001). This visual evoked response normally includes an early negative deflection (termed C1), followed by a positive deflection (P1) and a further negative deflection (N1). The C1 component occurs around 50 to 80ms after stimulus onset and appears to be sensitive to whether a stimulus is presented in the upper or lower visual field (Clark, Fan, & Hillyard, 1995). Di Russo, Martinez, Sereno, Pitzalis and Hillyard (2002) showed using both EEG and fMRI that this component is generated in primary visual cortex. The P1 component partially overlaps the C1 component, occurring around 70 to 120ms after stimulus presentation (Clark et al., 1995), and is followed by the N1 component (120 to 190ms). These later visual ERP components are thought to be generated in extrastriate cortical areas (Di Russo et al., 2002). The amplitude of P1 and N1 components at lateralised visual electrodes is modulated by the side of stimulus presentation, such that a greater P1 and N1 will be observed over electrodes contralateral to the side of stimulus presentation (Clark & Hillyard, 1996). Moreover, unlike the C1, the P1 and N1 are sensitive to attention amplification (Clark & Hillyard, 1996; Hillyard, Teder-Salejarvi, & Munte, 1998). More specifically, when a cue directs attention to a lateral visual target, the N1 and P1 response is increased over the contralateral hemisphere, in comparison to when a cue directs attention to the opposite visual field (Clark & Hillyard, 1996). Thus the N1 and P1

reflect visual responses to stimuli that can be modulated by the degree to which they are attended (Eimer, van Velzen, & Driver, 2002).

In addition to these increases in visual ERPs when a stimulus is attended, a number of later components have also been recently identified which are thought to reflect control of spatial attention. These components were first described by Harter, Miller, Price, LaLonde and Keyes (1989). They recorded ERPs in response to a cue which reliably (on 75% of trials) predicted the subsequent location of the stimulus. They found an early lateralised effect over occipital electrodes from 200 to 400ms after stimulus onset, with increased negativity contralateral to the side to which attention was cued. This effect was termed the early directing attention negativity (EDAN). A second effect was observed over posterior electrodes from around 500 to 700 ms after stimulus onset, with increased amplitude contralateral to the attended direction (the late directing attention positivity; LDAP). A number of later studies have also shown an increased frontal negativity contralateral to the direction of the attentional shift (anterior directing attention negativity; ADAN; Nobre, Sebestyen, & Miniussi, 2000). These components are thought to reflect successive steps in the control of covert spatial orienting.

Another lateralised posterior component, the N2pc has also been strongly linked with spatially selective visual processing. This component is characterised by an increased negativity over occipito-parietal electrodes contralateral to a target stimulus. Unlike the EDAN and LDAP, the N2pc responds selectively to target-related visual information (Eimer, 1996). In the visual search task, this component is exhibited contralateral to the side of the target in an array of distractors (Woodman & Luck, 2003). In a recent attempt to determine the exact nature of this component, Kiss, van Velzen and Eimer (2007) recorded ERPs in response to targets and non-targets following a cue which informed participants as to the location of the upcoming target. As expected they found ADAN and LDAP components in response to the cue stimulus, suggesting that the cue was successful in directing attention toward the cued location. Crucially, they found that the

N2pc was present even in situations where participants' attentional focus was cued to the location of the target. Therefore, they suggest that rather than reflecting shifts in spatial attention, the N2pc reflects spatially specific processing of task relevant features of the stimulus.

The P300 component

The P300 (also known as P3 or P3b) component is a large positive ERP component measured maximally over posterior electrodes. Coles et al. (1995), suggest that it is the most commonly reported of all ERP components, perhaps because it is large and is often evident even on single trials. Despite this popularity there remains a great deal of debate as to the exact functional significance of this component (Verleger, Jaskowski, & Wascher, 2005). The most common way to elicit a P300 component is in the oddball paradigm (Coles et al., 1995). In this task, participants are asked to look out for infrequent target stimuli presented in a sequence of non-targets. The P300 component is typically larger for the infrequent target stimuli than the frequent non-target stimuli. This has led to the interpretation that the P300 reflects the updating of a stimulus representation. Under this interpretation, each stimulus is compared to the previous stimulus and only when the stimulus is different and requires some mental or physical response is there a need to update the memory representation of the stimulus context. The context updating theory of P300 suggests that this component reflects this updating process (Polich & Criado, 2006).

In addition, the latency of the P300 is often used as a marker of stimulus evaluation time in part due to its purported role in context updating, since in order for context updating to occur the stimulus must have been fully evaluated (Coles et al. 1995). A number of studies have attempted to show that P3 latency is a marker of stimulus evaluation that is independent of response processing (see Verleger, 1997 for a review). Central to this claim is the suggestion that while P300 latency is sensitive to manipulations of availability of stimulus information, it is insensitive to manipulations of stimulus-response compatibility (Coles et al., 1995). However, in a thorough review of the literature Verleger (1997) finds that while with extreme cases of

stimulus evaluation and response selection processes this claim holds – i.e. P300 amplitude is only modulated by manipulations of stimulus evaluation – for intermediate stages P300 latency does not appear to be a reliable measure of stimulus evaluation time. In addition, Verleger et al. (2005) showed that P300 amplitude was as large in response locked ERPs as in stimulus-locked ERPs, suggesting that it is equally time locked to both stimulus and response processes. They suggest that rather than being a pure measure of stimulus evaluation, P300 reflects the transition from stimulus processing to response processing, perhaps monitoring whether the decision to classify a stimulus is successfully translated into an action.

ERP correlates of Motor Preparation

Kornhuber and Deecke (1965) were the first to show a movement related EEG potential occurring prior to voluntary movements. They found a central negativity beginning several hundred milliseconds before a voluntarily initiated movement, which they called the Bereitshaftspotential or Readiness Potential (RP). However, it is not entirely clear whether this RP reflects the execution of a specific act, or a more general readiness or preparatory state (Kutas & Donchin, 1980). Deecke, Scheid, and Kornhuber (1969) outlined a number of different sub-components of RP; they reported that when performing voluntary finger movements the onset of the RP was around 850ms prior to movement onset. They also reported two separate pre-movement components, a pre-movement positivity around 90ms before movement onset, and a surface negative motor potential around 55ms prior to movement. In addition to these extra components, Shibasaki and Hallet (2006) point out that the RP clearly divides into late and early components, with a sharp increase in the negativity occurring around 400ms prior to movement.

Kutas and Donchin (1980) also point out that the RP can overlap with the CNV, which has been found to be associated with general readiness or anticipation and not a specific motor program. However, they also show that the late section of the RP, which is lateralised to the contralateral hemisphere to the response hand, appears to be motor specific. They asked

participants to squeeze a dynamometer either at their own pace or in response to a stimulus, which was sometimes preceded by a warning signal and at other times occurred unpredictably. The response hand was varied from one block to the next so that following the warning cue participants could begin to prepare a hand-specific response. They found that the asymmetry in the RP began significantly earlier in the conditions where the response hand could be prepared further in advance of the movement (the self-paced and warned conditions) in comparison to the unwarned condition. This strongly suggested that the later part of the RP, which shows increased contralateral amplitude, reflects preparation to respond with one hand or another.

Coles (1989) was the first to formalise the calculation of this lateralised motor related activity and named it the lateralised readiness potential (LRP). Coles derived a method to isolate the activity in the EEG solely related to the lateralised motor preparation. Rather than simply exploring raw EEG activity over the contralateral hemisphere (following Kutas and Donchin, 1980), Coles suggests averaging the increased activity recorded over the right motor cortex during a left hand response with the increase in activity in the left hemisphere during a right hand response. The formula for this calculation is shown below, where C'_4 and C'_3 are 1 cm anterior to electrodes C3 and C4. The critical aspect of this calculation is that any non motor related asymmetries will sum to zero, since the measure shows the average difference between the two hemispheres for responses with both the left and right hand (Coles, 1989).

$$LRP = [Mean(C'_4-C'_3)_{\text{left-hand movement}} + Mean(C'_3-C'_4)_{\text{right-hand movement}}]/2$$

Since the RP is a negative going waveform and a greater negativity is observed contralateral to the response hand, the resulting LRP will be negative going for activation of the correct hand and positive going for activation of the incorrect hand. Gratton, Bosco, Kramer, Wickens, Coles and Donchin (1990) validated this measure by showing that activation of the correct and incorrect hand was dependent on the validity of a pre-cue which

informed the subject which hand would be required for the target stimulus. When a valid cue was presented, the LRP began to exhibit a readiness to act with the correct hand, while when the cue was invalid the LRP showed an opposite deflection, suggesting activation of the hand cued by the invalid pre-cue. No LRP activation was observed in response to a neutral pre-cue. In another experiment, Gratton, Coles, Sirevaag, Eriksen and Donchin (1988) showed that the LRP was directly related to the readiness to act by separating trials with fast response latencies from those with longer response latencies. They found that the LRP onset was earlier for the fast responses than the slower responses. In addition they found that a response appeared to be executed when the LRP amplitude reached a particular amplitude. This suggests that when the LRP reaches a response threshold, an overt response will be executed. A number of experiments with intracranial recordings in animals (see Coles, 1989; Coles, Smid, Scheffers and Otten, 1995) and magnetoencephalography (MEG; see Coles et al., 1995) have shown that the LRP is generated in the motor cortex.

ERP Correlates of Motor Inhibition

ERP correlates of inhibition are usually explored using variations of a go/no-go task. The simplest form of the go/no-go task involves participants responding to one stimulus, whilst withholding a response to another stimulus (Pfefferbaum, Ford, Weller, & Kopell, 1985). Sometimes the go/no-go stimulus is preceded by a warning signal, which in some variations of the task also contains certain information about the impending response (Eimer, 1993). A further variation on this task is the stop signal paradigm (Logan, 1994). This procedure involves inserting a stop signal on a small percentage of trials, a short time after the primary task signal (in a speeded choice reaction time task). This allows calculation of the so called stop-signal reaction time, i.e. the amount of time before the response the stop signal must appear to win the race and prevent the response.

Falkenstein, Hoornmann and Hohnsbein (1999) suggest that the no-go N2 and no-go P3 are the most widely reported correlates of no-go trials in the go/no-go task. They report that the no-go N2 is measured maximally at

frontal electrodes and that it may reflect a frontal lobe inhibition/control mechanism (Jodo & Kayama, 1992). A number of animal studies have suggested that the origin of the no-go N2 may be the caudal-dorsal principal sulcus. Sasaki, Gemba and Tsujimoto (1989) found that direct stimulation of this area 150 ms after a go stimulus caused inhibition of the response. Falkenstein et al. (1999) cite a number of studies in which the no-go N2 has been identified using visual go/no-go tasks, but suggest that it appears somewhat less reliably for auditory go/no-go tasks. The absence of the no-go N2 for auditory no-go trials is evidence against its role in inhibition. Falkenstein et al. (1999) conducted a series of go/no-go trials using both auditory and visual stimuli. They instructed participants to attend to one of the two sensory domains for different experimental blocks, so that the focused attention to the auditory domain in some blocks should increase the likelihood of obtaining a reliable no-go N2. In order to assess the interpretation of the no-go N2 and P3 components as inhibitory processes they divided participants into those with high false alarm rate and low false alarm rate. If either of these components reflect inhibition they should be greater in the subjects with the low false alarm rate, since they would be more likely to successfully inhibit an inappropriate response.

As predicted, in both auditory and visual domains participants with fewer false alarms exhibited a greater difference between go and no-go N2 components than participants with a greater number of false alarms. However, these differences were considerably smaller in the auditory than the visual sensory domain. The no-go P3 appeared to be insensitive to the same performance differences. They also found that focusing attention on auditory signals and ignoring the visual information increased no-go N2 amplitude, providing support for their assumption that visual attention bias may account for the lack of reported auditory no-go N2s in previous experiments. Falkenstein et al. (1999) suggest that the difference in amplitude between the auditory no-go N2 and the visual no-go N2 may imply that although the N2 appears to reflect inhibition in both modalities it may stem from generators specific to each modality. Support for this conclusion is offered by the finding that in monkeys, while the visual N2 appears to stem

from the cordal principal sulcus, the auditory N2 originates in the dorsal bank of the principal sulcus (Gamba & Sasaki, 1990, in Falkenstein et al., 1999). The authors thus suggest that the no-go N2 reflects modality specific inhibition and therefore is likely to occur prior to specific motor programming.

However, the functional significance of the no-go N2 and P3 has been the subject of intense debate in recent years. Niewenhuis, Yeung, Wildenberg and Ridderinkhof (2003) showed that a no-go N2 was observed on go trials when they were less frequent than no-go trials. The presence of the no-go N2 on go trials rather than no-go trials is clearly a problem for the hypothesis that the N2 reflects inhibition, since inhibition would not be present on a go trial. They suggest that the N2 on these trials reflects triggering of conflict monitoring when one is required to overcome a predominant response. They source localised this component to the anterior cingulate cortex (ACC), an area that is known to be activated during monitoring for conflicts during response selection (Botvinick, Cohen, & Carter, 2004). Similarly, Donkers and Van Boxtel (2004) showed that a similar N2 was observed in a go/GO task to that observed in a go/no-go task. In the go/GO task subjects were required to make a normal response on go trials and a larger amplitude response on GO trials. When a go stimulus was presented on 80% of trials an N2 was observed for GO trials. The authors argue that such a finding is very difficult to explain in terms of inhibition since inhibition should not be present when participants were required to make a stronger response than usual. In contrast, this result would be predicted by the conflict monitoring hypothesis, since subjects were required to overcome the predominant response force for that block of trials.

However, both Donkers and Van Boxtel (2004) and Botnovik et al. (2004) found a slight difference in the latency of the N2 for situations involving conflict monitoring, such that the N2 associated with conflict situations appears to occur around 50ms earlier than the N2 associated with motor inhibition. Falkenstein (2006) also suggests that while it is clear that the N2 does reflect conflict monitoring in the ACC, there is also much evidence to suggest that at least part of the component is generated in prefrontal cortex

and is associated with inhibition. Support for this claim comes from Lavric, Pizzagalli and Forstmeier (2004) who found that when go and no-go are equally probable the N2 is localised to ventral and dorsolateral prefrontal cortex, concluding that the N2 does indeed partly reflect inhibition. Further support for the inhibition hypothesis comes from the finding that inhibitory strength is also correlated with the no-go N2, such that when one is required to inhibit a high amplitude response the N2 is greater than when one is required to inhibit a smaller response (Nakata, Inui, Wasaka, Tamura, Akatsuka, Kida & Kakigi, 2006). Falkenstein (2006) suggests that whilst the debate about the N2 is not yet finished, it seems likely that the N2 reflects overlapping activity from the ACC (conflict monitoring) as well as another frontal source associated with inhibition.

In addition to the N2, the P3 has also been presented as a candidate for a correlate of response inhibition (Falkenstein et al., 1999). Like the N2, the no-go P3 is normally observed maximally over frontocentral electrodes. Donkers and Van Boxtel (2004) showed that, unlike the N2, the no-go P3 was only present for no-go versus go trials, and not GO versus go trials, suggesting that the no-go P3 rather than the N2 reflects inhibition of the response. Similarly, Smith, Johnstone and Barry (2007) showed that the amplitude of the no-go N2 was smaller following a no-go cue than a go cue despite the fact that the go cue was successful in increasing participants' readiness to respond to the upcoming target. This finding provides evidence against both the inhibition and conflict monitoring hypothesis of the N2, since both would predict a greater N2 in a situation where a response was expected but then withheld. Smith et al. (2007) did however show that the P3 component was of greater amplitude for no-go trials following go cues than those following no-go cues. Thus, the authors suggest that the no-go P3 reflects inhibition and/or conflict monitoring.

However, Falkenstein et al. (1999) point out that although the reliable presence of the no-go P3 is not in doubt, even its onset is too late to reflect inhibition mechanisms as it often occurs later than the response itself in go trials. They suggest that rather than reflecting the process of inhibition itself,

the no-go P3 may reflect the reset or closure of a preceding inhibition process. Similarly, Dimoska, Johnstone and Barry (2006) suggest that the P3 on successfully stopped trials (in the stop-signal paradigm) reflects the outcome of inhibition of the response in the primary motor cortex. However, Falkenstein et al. (1999) point out that interpretation of the P3 is confounded by its overlap with motor-related activity, and it might therefore simply reflect the fact that in one condition a motor response is programmed while in the other it is not (see also Verleger, Paehge, Kolev, Yordanova, & Jaskowski, 2006). The latter problem is often overcome by exploring the P3 only on trials where no response is made, dependent on the information in a cue prior to the target stimulus (Eimer, 1993; Smith et al., 2007). By comparing the P3 in situations where a response is present in both conditions, the resulting difference can not simply be due to motor-related activity.

Fallgatter and Strik (1999) developed an ERP index of motor inhibition which utilises this increased frontal P3 for no-go trials. They calculated the no-go anteriorisation as a measure of the degree to which the P3 component becomes more anteriorly distributed for no-go trials. Participants completed a number of blocks of the continuous performance task, which involves presenting a continuous string of stimuli and asking participants to make a response following a certain stimulus sequence. Fallgatter and Strik (1999) asked participants to respond when the letter O was directly followed by an X. In this way, the O acts as a warning stimulus which is then followed by either a go stimulus (an X) or a no-go stimulus (any one of 10 letters). They found that the normally parietal P3 (discussed earlier) becomes more frontally distributed on no-go trials, where it shows more positive amplitude than go trials. They termed this change in topography of P3 for no-go trials the no-go anteriorisation.

In summary, both the no-go N2 and P3 components, which are maximal over frontocentral electrode locations have been strongly linked to frontal inhibition/control mechanisms. While the exact functional significance of these components still requires some clarification, it is clear that the N2/P3 complex reflects activity in the pre-frontal cortex related to conflict monitoring

or cognitive control/inhibition mechanisms. Evidence from fMRI has points to a role for dorsolateral and ventrolateral pre-forntal cortex, ACC and pre-SMA in inhibitory control (Garavan, Hester, Murphy, Fassbender, & Kelly, 2006). In addition, a number of studies have reported a right lateralization of pre-frontal activity in the go/no-go task (cf. Garavan et al., 2006). However, EEG source analysis has also revealed a left frontal source for activity related to no-go trials (Verleger et al., 2006), while all the studies cited above show the no-go N2 and P3 to be maximal at midline electrodes.

Motor Cortex Inhibition

De Jong, Coles and Gordon (1995) investigated motor inhibition in three different situations to test the hypothesis that there are two different systems responsible for inhibition of movement. In a previous experiment (De Jong, Coles, Logan & Gratton, 1990) they had explored ERP's using the stop-signal paradigm in a speeded reaction task and found that on a large proportion of trials where the response was successfully inhibited, activity in cortical motor structures exceeded the threshold normally associated with movement onset. They suggested that such a finding is consistent with the idea that a peripheral non-motor system is able to successfully inhibit a movement even if it has been fully programmed by motor structures. A distinction between central and peripheral motor inhibition structures has also been proposed by Bullock & Grossberg (1988) and has some empirical support (Jennings, van der Molen, Brock, & Somsen, 1992).

De Jong et al. (1995) explored situations in which complete inhibition of a pre-prepared response was required, with situations in which participants were asked to selectively inhibit a particular response, whilst continuing with another response. They suggest that only a central (motor cortex) motor inhibition process would allow such selective inhibition, since the peripheral inhibitory system (prefrontal cortex) is assumed to work in a largely non-specific manner (Bullock and Grossberg, 1988). De Jong et al. (1995) hypothesised that the peripheral inhibition system is faster and all other things being equal successful inhibition is most likely to occur via this system. A second prediction was that in situations where central motor

inhibition processes successfully inhibited a response, the LRP would fail to reach a level associated with action, while for a peripheral control mechanism successful inhibition may still occur downstream from the motor activation commands.

De Jong et al. (1995) used the stop-signal procedure to explore these predictions. This procedure involves inserting a stop signal a short time after the primary task signal (a speeded choice reaction time stimulus). The shorter the stop-signal delay, the more likely successful inhibition will be achieved. De Jong et al. (1995) employed this paradigm in four different experimental conditions: stop-all; stop-change; selective-left hand; and selective-right hand. Participants were required to make speeded responses with the left and right hand to the letters M, N, V and W, with each response hand specified by two of the target letters. In the stop all condition participants were instructed to abort any response on trials with a stop-signal (an auditory stimulus). In the stop change condition participants were required to abort the hand movements and make an alternative movement with their foot. In the selective-left hand and selective-right hand the stop signal required participants to abort the responses only with the specific hand. These four conditions were administered in separate blocks, with a stop-signal presented on 50% of trials. The delay between the warning signal and the stop signal, known as the stop signal delay, was initially set to 250 ms, but was then adjusted individually for each participant to result in inhibition success rate of around 50% in each condition.

De Jong et al. (1995) found that in all three conditions (stop-all, stop-change, and selective-stop) the LRP appeared significantly diminished on the trials where stop signals were present. This suggests that central inhibition mechanisms were operating in all three conditions. Further analysis showed that there were no significant differences in LRP for go trials in the different experimental conditions, providing support for the notion that a response is activated when the LRP reaches a certain threshold. This LRP amplitude at movement onset (LRP threshold) for go trials was then compared to the LRP's associated with successfully inhibited trials in each condition. For the

stop-all and selective-stop conditions the maximum LRP exceeded this threshold value, whilst in the stop-change condition the LRP failed to reach the amplitude required to initiate movement. This suggests that whilst a central motor inhibition was responsible for successful inhibition in stop-change blocks, peripheral inhibition was more likely associated with inhibition in the other two conditions. A possible electrophysiological correlate of such a peripheral inhibitory mechanism is the N2 negativity reviewed above. De Jong et al. (1995) suggest that this mechanism operates downstream from the motor cortex and can inhibit motor responses that are above the threshold of motor activation in the motor cortex, such as was observed in the stop-all and selective-stop conditions.

This description of a frontal peripheral control system operating downstream from the motor cortex is contrary to the interpretation provided by Falkenstein et al. (1999), who suggest that N2 related inhibition measured over frontal electrodes operates upstream from motor systems. Band and Van Boxtel (1999) also discuss the difference between central and peripheral mechanisms and come to similar conclusions to Falkenstein et al. (1999). They suggest that De Jong et al.'s (1995) conclusion rest too much on the assumption that movement onset is subject to a threshold in the LRP, and that a peripheral system of motor control operating upstream (prior) to motor programming (in the motor cortex) equally well describes their data. Whichever interpretation is correct it seems evident that there is an important interplay between motor cortex activation and frontal cortex inhibition (such as indexed by the N2), and that future research should focus on recording and interpreting both components in parallel.

Summary

The current chapter outlined the background to the use of EEG in psychology and cognition and presented evidence regarding ERP components associated with preparation and inhibition of a motor response. Preparation for a response has been strongly associated with the readiness potential (RP) measured over the motor cortex. Whilst this component is

thought to reflect a general readiness to respond, a more specific component has also been identified which measures specific activity related to the readiness to respond with one hand. This lateralised readiness potential (LRP) has been identified using choice reaction time tasks in which one hand is cued in advance of the target stimulus. Components associated with inhibition of a motor response have been identified using various forms of the go/no-go task. Two components seem reliably associated with response inhibition in these tasks, the N2 and P3. While there is still some debate as to whether these components reflect inhibition per se, or conflict monitoring processes, it seems likely that at least part of the no-go N2 P3 complex reflects activity associated with inhibiting a motor response.

Chapter 3

Introduction to Subliminal Priming

Outline

This chapter will provide a more detailed introduction to subliminal priming and its use as a method for studying unconscious processing. This initial part of the chapter will focus on the methodology and the theoretical considerations concerning unconscious perception. The latter part will provide a literature review of research exploring unconscious processing in motor preparation and inhibition. This will lead directly on to the hypotheses for the research in the current thesis.

The origins of subliminal priming

The word subliminal means below the threshold (of consciousness) and comes from the word sub meaning below and limen meaning threshold. Subliminal perception first burst onto the scene in 1957 when James Vicary called a press conference to announce that he had successfully influenced cinema goers in New Jersey to buy popcorn and cola by briefly flashing "Drink Coca-Cola" and "Hungry? Eat popcorn" during the film (cf. Brannon and Brock, 1994 and Karremans, Stroebe and Claus, 2006). Despite the fact that the audience were unaware of these messages, Vicary claimed a substantial increase (of up to 60%) in sales of popcorn and cola when the messages were presented. Although Vicary later admitted to having falsified the results in order to promote his advertising business the myth of subliminal advertising is still present in public perception and many countries have banned its use (Karremans et al., 2006).

Although some recent evidence from Karremans et al. (2006) suggests that subliminal stimuli may influence choice of a drink when participants are thirsty, the majority of evidence suggests that, in the form often used in self-help audio tapes and in advertising, subliminal messages are largely

ineffective (Brannon and Brock, 1994). In addition to their use in subliminal messaging and advertising, subliminal stimuli have been utilised in the scientific study of unconscious perception. However, this area of research also been the subject of much controversy (cf. Erdelyi, 2004; Reingold & Merikle, 1988).

The earliest reports of perception below the threshold of awareness compared the ability of subjects to make some kind of (unconscious) discrimination, with their subjective report of the stimulus. Perhaps the earliest of all of these was an experiment described by Peirce and Jastrow (1885) where they showed that subjects were able to make fairly accurate judgements of relative brightness of stimuli, despite the fact that they reported no confidence in these judgements. In this experiment the subjective report is proposed to be the measure of conscious awareness, while the results from the discrimination task are meant to show that despite being unaware of the differences between different stimuli the subjects were able to reliably judge their relative brightness. In an early review, Adams (1957) suggested that the ability to make some discrimination between stimuli in the absence of awareness was a highly replicable effect.

However, despite this early optimism a number of important theoretical issues still required clarification. Most importantly, Eriksen (1960) has questioned the assumption that a lack of subjective confidence equates to a lack of consciousness of a stimulus. Similarly, Snodgrass and Shevrin (2005) have argued that denials of awareness may simply reflect a lack of confidence rather than indexing a boundary between conscious and unconscious. An alternative approach to the subjective measure of conscious awareness has been to assess consciousness with objective measures. This typically involves asking participants to perform a two alternative forced-choice task either requiring them to detect the presence or absence of a stimulus (detection task) or to determine whether a stimulus belongs to one group of stimuli or another (recognition task or identification task). Participants' performance on such a task is then compared to chance, either by comparing their percentage of correct responses with the number

that would be predicted by chance, or by calculating a measure derived from signal detection theory (*d'*), which gives a measure of the ability to discriminate the possible alternatives that is independent of response bias (cf. Stanislaw & Todorov, 1999).

Reingold and Merikle (1988) suggest that any measure of conscious awareness should be both exhaustive and exclusive for it to be an appropriate index of consciousness. The exhaustiveness criterion states that the measure must be sensitive to *all* the information in consciousness. The exclusiveness criterion states that the measure must be a pure measure of conscious information and must therefore not be influenced by unconscious information. A subjective measure of consciousness likely violates the exhaustiveness criterion, since it only appears to be sensitive to conscious information of which the participant is highly confident. In contrast, an objective measure may violate the exclusiveness criterion since performance on a forced-choice task may be influenced by unconscious processes. This has led Reingold and Merikle (1988) to argue, along with others (e.g. Erdelyi, 2004), that since it is impossible to know whether a measure of consciousness meets both of these criteria it is inappropriate to use a single index of whether a stimulus reached consciousness. This problem is highlighted by the fact that while early studies (such as Peirce & Jastrow, 1985) used a discrimination task to index unconscious processes, the very same task is now often used to measure whether participants are conscious of a stimulus (Dehaene, Naccache, Le Clec, Koechlin, Mueller, Dehaene-Lambertz, Van de Moortele & Le Bihan, 1998; Leuthold & Kopp, 1998). Reingold and Merikle (1988) suggest combining a subjective measure of conscious awareness with an objective task that shows a qualitative difference between conscious and unconscious stimuli, such that whilst a conscious stimulus will have an effect in one direction an unconscious stimulus will produce the opposite effect (Merikle & Cheesman, 1985).

While a number of important theoretical and methodological obstacles remain, the use of objective measures of awareness is now widely regarded as an appropriate tool for exploring unconscious cognition (Snodgrass &

Shevrin, 2005). These measures are normally utilised in combination with a priming task in a procedure known as masked priming. This involves the brief presentation of a prime followed by a mask, which further reduces visibility of the prime (Dehaene & Naccache, 2001). Immediately after the mask, the target stimulus is presented. Participants are required to make a judgement or response to the target stimulus, with the influence of the subliminal prime assessed by means of how it affects participants' response to the target. Typically, conscious awareness of a stimulus is assessed by a two alternative forced-choice task that presents the same sequence as that used in the masked priming task, but participants are now asked to respond to the prime rather than the target. This methodology has also been called subliminal priming and unconscious priming as well as "indirect without direct effect". The first two names reflect the fact that the unconscious influence is observed through the presence of an unconscious prime, and is measured by its effect on a target stimulus. Similarly, the label of direct without indirect effect reflects the fact that whilst the prime was able to influence participants' responses when they were responding to the target (indirect effect) it disappears on the control task where subjects are required to directly respond to the briefly presented prime. One of the earliest examples of unconscious perception using this method was reported by Marcel (1983), who showed that masked words (primes) were able to influence participants preference for a subsequent word (targets), despite the fact that they performed at chance level when asked to discriminate the different primes.

However, a further problem with this methodology is that in order to show that a priming effect is unconscious it is necessary to accept the null hypothesis. For example, when conducting a control task to assess participants' awareness of the stimulus, performance at chance level is deemed to be sufficient to show that subjects were not conscious of the stimulus. The problem with such a position is that it is not possible to conclusively know that the null hypothesis (i.e. that subjects did not perform significantly better than chance) has been supported, since the failure to reject the null hypothesis may reflect measurement error with the subjects true performance exceeding chance (cf. Erdelyi, 2004; Greenwald Klinger

and Schuh, 1995; Reingold and Merikle, 1988; Snodgrass, 2004). Greenwald et al. (1995) propose a method to overcome this problem. Their approach is to calculate the regression between the direct and indirect measure and to determine the point at which the regression line crosses the y axis. They suggest that this gives a measure of the indirect without direct pattern, i.e. the amount of priming that would be present when there is zero delectability of the stimulus. This allows a statistical test of this value, which will then provide a rejection of the null hypothesis if priming is observed in the absence of awareness. However, as Snodgrass and Shevrin (2005) point out, since the direct performance still contains measurement error which will be reflected in the regression slope, this does not entirely solve the problem. The significant advantage of this procedure though is that when prime visibility is above chance, it is possible to assess to what extent the observed priming effect is due to the visibility of the prime (Kouider & Dehaene, 2007).

While Snodgrass and Shevrin (2005) accept that the null sensitivity problem remains difficult to overcome, they propose that if the priming effects observed on the direct task reflect residual awareness of the prime, then participants performance on the task should be correlated with performance in the visibility task. They suggest that showing a negative correlation between performance on one task, sensitive to unconscious information, and a visibility task would provide a qualitative difference between conscious and unconscious processing. The two most common tasks used to assess the visibility of a stimulus are stimulus detection and stimulus identification. Since stimulus identification requires first detection, and then categorisation of a stimulus, the threshold for detection of a stimulus is lower than it is for identifying it. Therefore, if priming is caused by residual awareness of the prime, one would expect priming to be greater at the identification threshold than the detection threshold. Snodgrass (2004) reviews the literature and find in fact that priming appears greater at the detection threshold than at the identification threshold. They suggest that this negative relationship between priming and prime visibility constitutes a qualitative difference between conscious and unconscious processing. Snodgrass, Bernat and Shevrin

(2004) argue that when conscious influences are completely absent (at the objective detection threshold) the unconscious information is free to influence behaviour without contamination from conscious access to the prime. In contrast when conscious information is relatively weak, but nonetheless present (between the detection and the identification thresholds), the availability of the information to consciousness reduces its ability to influence the prime. In this window the conscious effect is only large enough to produce very small priming effects, with the priming effects from the unconscious perception completely abolished. Once the primes become fully conscious they are able to exert a greater influence on behaviour and thus priming increases again when signal strength is above the identification threshold.

Holender and Duscherer (2004) have challenged the data on which Snodgrass et al. (2004) base their recommendations. They suggest that many of the studies quoted by Snodgrass as showing the necessary negative relationship between priming and prime visibility are unlikely to be replicable under more stringent conditions. They also question the general approach to the study of unconscious perception and suggest that no evidence exist of truly unconscious effects on behaviour. Despite their pessimism, others are more optimistic that the wealth of research into unconscious cognition has not been in vain. Merikle, Smilek and Eastwood (2001) review over one hundred years of research on perception without awareness and conclude that regardless of which particular method was used for determining consciousness of the stimuli, there is overwhelming evidence in support of unconscious cognition. With the increase in interest in consciousness in recent years the study of unconscious perception is currently enjoying a boom period, where the majority of people believe that the appropriate methods exist to allow successful investigation of the effects of unconscious events on individuals' behaviour (cf. Koudier & Dehaene, 2007). While some (e.g. Merikle et al., 2001) still prefer to assess consciousness via subjective measures, the vast majority of recent research (e.g. Neumann and Klotz, 1994; Leuthold and Kopp, 1998; Eimer and Schlaghecken, 1998; Dehaene et al., 1998) compares performance on an

indirect task with that on a forced-choice task where subjects are asked to respond directly to the unconscious stimulus (prime). Since objective measures are thought to be more conservative than subjective measures they are likely to persuade more sceptics that participants in such experiments were unaware of the subliminal prime (Merikle et al., 2001).

The exact forced-choice task used in the literature varies. While some research has reported priming in the absence of detection of the stimulus (e.g. Dehaene et al. 1998) many assess awareness with identification tasks (e.g. Leuthold and Kopp, 1998; Eimer and Schlaghecken 1998). Since the indirect task normally involves classifying the stimulus as one type or another it seems appropriate to use a similar classification task (identification task) to assess consciousness, since one is interested in whether participants were able to extract the appropriate information from the prime consciously, not simply whether they were able to see the prime (detection task). In the experiments described in this thesis awareness of the primes was assessed using objective measures in addition to subjective reports from subjects. In most experiments participants are asked to identify the prime, but in some experiments they also performed a detection task.

Masked priming and motor preparation

Neumann and Klotz (1994) were the first to show that completely masked stimuli are able to exert some influence over motor preparation. Fehrer & Raab (1962) showed that whilst subjective reports of the visibility of a masked stimulus change in accordance with the stimulus onset asynchrony (SOA) between prime and mask, reaction times were unaffected. Neumann and Klotz (1994) were interested in exploring whether this observed dissociation between awareness and motor priming was observed when prime visibility was below the objective threshold of identification. These experiments were designed to asses their hypothesis of direct parameter specification, inspired by the work of Wilhelm Wundt and his student Hugo Münsterberg who had suggested that our motor apparatus does not wait for

consciousness before starting to prepare a response (Neumann & Klotz, 1994).

Neumann and Klotz (1994) conducted five experiments to explore the limits of direct parameter specification, in other words to explore the situations in which one can observe a dissociation between conscious awareness of a stimulus and a motor response to the stimulus. All experiments utilised a metacontrast masking sequence. Metacontrast masking involves the presentation of the prime followed by a target, which also acts as a mask. In this procedure the prime stimulus normally fits into a space left in the centre of the target stimulus (the mask). In the first experiment the target stimuli consisted of one diamond and one square. The participants were asked to respond with the left key when the target (diamond or square, counterbalanced across subjects) was on the left and to respond with the right hand for a right sided target. Unknown to the participants smaller replicas of the stimuli (the primes) were presented in advance of the target. On a congruent trial the primes were the same as the target stimuli, on incongruent trials the side of the diamond and square were reversed and on neutral trials two of the non-target stimuli were presented. Following the reaction time (RT) part of the experiment participants were asked to determine whether the target contained a small replica of the target. This amounted to an identification task, since rather than detecting if a prime was present, participants were asked to determine if the mask was congruent with the target stimulus. The sequence of stimuli for this part was identical to the RT part of the experiment. The results of this first experiment showed that despite showing a d' that was not significantly different from zero, participants were on average 50ms quicker to respond to a stimulus with a congruent prime than a stimulus with an incongruent prime.

A second experiment replicated this basic effect of the first with slightly different stimuli. In this experiment subjects were asked to respond with the appropriate hand when one of two lateralised stimuli were flanked by horizontal bars. Once again a congruent prime was found to improve RT on this task despite zero d' in the identification task. Two further experiments

extended these findings to incompatible response mappings and to situations where response mappings could vary from one trial to the next. Finally, experiment 5 showed that when a prime was located in a third location its effect on behaviour was dependent on the response mapping of the three stimuli such that when this middle location acted to guide a right hand response, facilitation for a subsequent right sided stimulus was observed. The authors argued that this shows that the priming of the responses is dependent on the specification of the motor response and not simply from visual effects of the prime. They conclude from these experiments that direct parameter specification from an unconscious prime is a robust and replicable effect and thus that a stimulus can guide a motor response independent of a participants awareness of the stimulus. This finding is in line with the data discussed in chapter 1 using a very different paradigm, and suggests a similar conclusion; that motor preparation can begin in advance of conscious awareness of an intention to act or conscious discrimination of a priming stimulus.

Leuthold and Kopp (1998) have since examined the theory of direct parameter specification in more detail combining behavioural and electrophysiological measures. They asked subjects to respond with one hand when a stimulus above fixation was flanked by vertical bars and with another hand when the stimulus below fixation was flanked by vertical bars. When the unconscious primes were congruent with the target, reaction times were faster than on incongruent trials. Critical to the hypothesis that the unconscious primes were able to *directly* initiate motor preparation was the evidence provided by the lateralised readiness potential. Leuthold and Kopp (1998) reasoned that if the unconscious prime was able to directly initiate motor processes then early LRP activity should be determined by the subliminal prime. If the prime is able to directly program the motor response then hand specific motor preparation, as indexed by the LRP should begin in response to the prime. Leuthold and Kopp's (1998) result was entirely consistent with this hypothesis. They found that following an incongruent prime the LRP showed initial activation of the incorrect hand. For congruent trials the LRP began its prime-related activation in the correct direction,

before continuing to increase in response to the conscious target stimulus. This difference in early partial activation of the LRP then led to an earlier onsetting LRP for congruent trials, which in turn was likely responsible for the behavioural priming effect. Despite this difference in motor related electrical activity, early visual ERP components did not appear to be modulated by the unconscious prime, supporting Neumann and Klotz's (1994) suggestion that the unconscious primes are able to directly specify the motor codes without modulation by perception.

Dehaene et al. (1998) have also shown that pattern masked number words (e.g. ONE, FOUR) can influence activity in the motor cortex recorded by both EEG and fMRI. In their experiments they asked subjects to respond to numerals above five with one hand and below five with the other hand. Unknown to the participants number words were presented prior to the target in between forward and backward masks consisting of random letter strings. Despite performance on detection and identification tasks not differing from chance modulation of reaction times and lateralised motor activation was seen in response to the prime. This further supports the assumption that motor activation can be initiated unconsciously, and in this example that it may be initiated by primes that are semantically related to targets and not visually related (e.g. ONE primes a response to 1).

Masked priming and motor inhibition

Eimer & Schlaghecken (1998) have reported evidence that unconscious primes can drive exogenous motor inhibition. In a typical subliminal priming experiment, an unconscious prime normally produces a positive compatibility effect, such that reaction times are reduced when the unconscious prime is congruent with the target, and increased when an incongruent prime is presented (Leuthold & Kopp, 1998; Neumann & Klotz, 1994) . However, Eimer and Schlaghecken (1998) found that masked primes in such a task produced a negative compatibility effect (NCE), such that congruent masked primes *impeded* responses. In this experiment, primes were presented for 16ms, immediately followed by the mask for 100ms and then the target for

100ms. Primes and targets were typically double left and double right pointing arrows (<<, >>; or >< and <> for neutral primes), and masks were the two stimuli overlapped (⋘⋙). Participants were asked to respond to left pointing arrows with a left hand key press and right pointing arrows with a right hand press. Reaction times were fastest when an incongruent prime was presented prior to the target (e.g. a left pointing prime presented prior to a right pointing target). Similarly, RTs were greatest following a congruent prime. This surprising result of increased RTs on congruent trials was interpreted as being caused by automatic inhibition of the unconsciously activated response. In support of this assumption, they reported that ERP components associated with preparation to respond with either the left or right hand (LRP) showed an initial activation on congruent trials, followed by a reversal of this activation. This reversal was interpreted as reflecting a temporary, automatic inhibition of the unconsciously initiated response. They suggest that such a mechanism may prevent us from responding automatically to small insignificant changes in our environment.

Eimer and Schlaghecken have replicated this NCE using a number of different stimulus parameters, and other research groups (Klapp & Hinkley, 2002) have also published similar findings. In one experiment, Eimer (1999) manipulated the stimulus onset asynchrony (SOA) between the masked primes and the target stimulus and measure participants' reaction times. When the SOA was short (0ms and 32ms) a positive compatibility effect was present such that congruent primes improved reaction times, but when SOA was longer (96ms and above) a negative compatibility effect was evident. Eimer (1999) suggests that this finding provides further evidence of the "activation followed by inhibition" hypothesis since with a low SOA the stimulus would come while the masked prime is still partially activated, but for a long SOA the target stimulus would begin to be processed while the initial primed response was being automatically inhibited. They suggest that such an automatic inhibition of unconsciously initiated responses that are no longer online is evolutionarily advantageous as it prevents us responding automatically to every small insignificant change in our environment. When the primes are not masked (and thus become available to conscious

introspection) the negative compatibility effect disappears and only endogenous inhibition can then prevent movement. Klapp and Hinkley (2002) claim that this shows a qualitative difference between conscious and unconscious processing.

Aron et al. (2003) showed using a similar paradigm in an fMRI scanner that initial unconscious response activation initiated by the masked primes is associated with increased activity in the hand area of the primary motor cortex contralateral to the direction of the prime. This provides further evidence of initial unconscious motor activation, and highlights a likely neural basis for this effect. When exploring the subsequent inhibition of this unconsciously initiated act they found significant increases in activity in the posterior parietal cortex and in several sub-cortical areas. Notably they did not find any activation of prefrontal areas thought to be responsible for the type of endogenous inhibition associated with the N2 ERP component as described in chapter 2.

The inhibition hypothesis of the NCE has been widely disputed in recent years. Lleras and Enns (2004) suggest that it might be due to perception of the difference between the prime and the compound mask rather than motor inhibition. They suggest that updating of the visual scene leads to processing of the change between the prime and the mask (the prime-mask effect). Since Eimer and Schlaghecken (1998) used a mask that was formed from a compound of the two possible stimuli, the presentation of the mask effectively involved the addition of two arrows in the opposite direction, which cued a response opposite to that cued by the actual prime. Lleras and Enns (2004) suggest that it is this change between the prime presentation and the mask presentation that is responsible for the NCE. Similarly, Verleger, Jaskowski, Aydemir, van der Lubbe and Groen (2004) conducted a series of experiments to explore the possibility that the NCE is caused by a specific interaction between the prime and the mask. They showed that when using a checked mask rather than the compound of the two possible targets, a positive compatibility effect was observed, such that congruent primes facilitate motor preparation. They also found that no LRP reversal was

observed on trials with the checked mask. These findings suggest that the NCE observed by Eimer and Schlaghecken may be the result of the particular prime-mask combination.

While Schlaghecken and Eimer (2006) have conceded that with related masks, the NCE is most likely caused by object updating, they present new data to show that it is still possible to obtain an NCE even when using unrelated masks. Jaskowski (2007) and Lleras and Enns (2006) suggest that rather than reflecting automatic inhibition caused by the prime, the NCE in these studies reflect what they call mask induced inhibition. They show that a combination of physical, spatial and temporal similarity combine to turn a positive compatibility effect into an NCE. Each of these factors appear to be additive in reducing the positive priming effect, eventually resulting in a strong NCE when a central, related mask is presented immediately after the prime. Jaskowski (2007) suggests that the mask acts as a distracter, that inhibits the response associated with the prime. He shows that even when a related stimulus is used as a flanker rather than a mask; it still produces an NCE, even though there is no spatial overlap with the prime. This provides evidence against a simple perceptual object updating mechanism.

While the exact mechanism that causes the NCE is still under dispute it seems likely that some kind of inhibition of the primed response is involved in reversing the priming effect. Eimer and Schlaghecken (2003) suggest that whilst this exogenous inhibition is initiated unconsciously as defence mechanism against automatic response activation, endogenous inhibition (such as in a go/no-go task) can only be initiated with conscious awareness:

"This *endogenous* inhibition is voluntary, optional, and is presumably mediated in prefrontal cortex. Since endogenous inhibition depends on the conscious detection of task-relevant signals, it is not available when stimuli are presented subliminally". (Eimer & Schlaghecken, 2003; page 8).

However, they only point to indirect evidence in support of this assumption. They suggest that evidence for this hypothesis comes from negative priming,

the Stroop effect and shifts of spatial attention. However, the inhibition involved in each of these processes is very different to inhibition of a motor act currently under preparation. Negative priming for example assesses the influence of a stimulus that was previously to be ignored and now acts as the target. Participants typically respond more slowly to such a stimulus, than to a stimulus that had not previously been ignored. This effect disappears when the stimulus is masked during its original presentation as a to-be-ignored stimulus (Lalchandani, Loula, & Carrasco, 2003). However such inhibition of a response in negative priming is in fact more like an automatic process to inhibit information that was recently deemed irrelevant, than voluntary and conscious inhibition. Similarly, success in the Stroop task relies on the ability to inhibit an automatically initiated interference, a very different process to inhibition of a motor action.

Eimer and Shlaghecken (1998) also point out that the N2 ERP component appears somewhat atypical when found in unconscious priming paradigms. The N2 component is a frontal negativity around 200 ms after stimulus onset typically found in conflict tasks such as the flanker task (Kopp, Rist, & Mattler, 1996) and in go/no-go tasks (Falkenstein et al., 1999) and is thought to reflect inhibition (Falkenstein et al., 1999) or cognitive control (Nieuwenhuis et al., 2003). Importantly, when conflict between two responses is induced by an unconscious stimulus, this negativity appears to have a parietal, rather than frontal topography (Leuthold & Kopp, 1998). Eimer & Schlaghecken (2003) suggest that this topographic difference may reflect the fact that only exogenous inhibition can be initiated unconsciously. They suggest that whilst a parietal N2 may be observed in response to unconscious conflict, a true frontal no-go N2 indexing inhibition can not be modulated by unconscious primes.

The suggestion that frontal control mechanisms require conscious awareness is supported by research using a number of different paradigms. Dehaene et al. (2003) explored activation of the anterior cingulate cortex (ACC) in patients with schizophrenia and normal participants. They presented participants with subliminal primes that were either congruent or

incongruent with a target stimulus. Primes consisted of the words ONE, FOUR, SIX and NINE. Targets consisted of the numbers 2, 4, 6 and 9. Participants were asked to press one hand in response to targets above five and another for targets below 5. Both the masked and unmasked primes produced behavioural conflicts, manifested in increased reaction times to incongruent trials. However, ACC activation (measured using fMRI) was recorded in response to conscious response conflicts, but not in the subliminal conflict condition. Dehaene et al. (2003) argue that this shows that the ACC is activated exclusively for resolving conscious conflicts. Similarly, Praamstra and Seiss (2005) showed that no genuine frontocentral N2 was elicited by conflicts induced in the NCE, suggesting that unconscious response conflicts were regulated from within the motor system.

Praamstra, Turgeon, Hesse, Wing, and Perryer (2003) explored the error related negativity (ERN) in response to errors that were consciously detected and those that were not detected. Like the N2, the ERN is thought to reflect activity in the ACC related to the detection of response conflict on trials where participants make the incorrect response (Yeung, Botvinick, & Cohen, 2004). Praamstra et al. (2003) showed that while conscious errors were associated with an ERN, unconscious errors did not seem to engage frontal conflict detection processes. Similarly, Niewenhaus, Ridderinkhof, Blom, Band and Kok (2001), showed that the error positivity (analogous to the no-go P3) was only present when participants were aware of having made an error. Mayr (2004) reviews a number of studies that explore fMRI or EEG correlates of ACC activity in response to conscious and unconscious conflict and conclude that conscious awareness seems to be crucial for many kinds of ACC-related activity.

In a prominent recent theory of consciousness, Dehaene, Changeux, Naccache, Sackur and Sergent (2006) outline a neuronal workspace model in which sensory inputs compete for access to a neuronal workspace. They suggest that sensory information only enters the neuronal workspace, and therefore becomes conscious, when it is sufficiently strong and when it received top-down attention. They claim that subliminal stimuli fail to reach

consciousness because they do not have sufficient bottom-up strength but may still produce feedforward activation leading to unconscious priming of behaviour. Crucially, they suggest that subliminal activation does not lead to durable activation of fronto-parietal brain circuits, once again highlighting the association between consciousness and engagement of processing in frontal brain regions.

Summary

This chapter introduced the technique of masked priming as a way of studying unconscious perception. Despite the difficulty in measuring whether a participant is conscious of a stimulus, the research described in this chapter convincingly shows that a number of processes can occur in the absence of consciousness. In support of the research outlined in Chapter 1, evidence from subliminal priming studies suggests that motor preparation can be initiated unconsciously. Furthermore, the research supporting a link between inhibition and control mechanisms and consciousness provides some support for Libet et al.'s (1985) claim that consciousness may have a role in vetoing unconsciously initiated acts.

General Summary and Aims of Current Research

The research outlined in these three introductory chapters highlights an important association between consciousness and inhibition/control of behaviour. Libet et al. (1985) suggested that while consciousness does not initiate a voluntary action it may be required if one decides to veto that action. The research presented in this thesis aims to directly explore the association between frontal inhibition/control mechanisms and consciousness. While previous research in masked priming has focused on activity associated with response conflict or error processing, no research to date has explored possible modulation of ERP components associated with inhibition of a response in the go/no-go task. Since this task is known to produce a no-go N2/P3 complex, combining this task with a masked priming paradigm will allow exploration of possible modulation of these components

dependent on the unconscious information. Therefore, this task will allow direct exploration of Libet et al.'s (1985) suggestion that consciousness is required to inhibited an imminent action. Due to the excellent temporal resolution of EEG the current research will also attempt to determine if these frontal inhibiton/control mechanisms can be *directly* elicited by the unconscious primes. Leuthold and Kopp (1998) showed that LRP activity shows initial modulation that is entirely dependent on the unconscious primes. In a similar way, the current research will assess if subliminal primes are able to directly engage frontal inhibition/control mechanisms indexed by the no-go N2. If the N2 shows an early modulation dependent on the prime type, then this would support the hypothesis that inhibition of an impending action can be initiated unconsciously.

Chapter 4

General Methods

Outline

This chapter will provide a more in-depth account of the experimental methods used in this thesis. In particular it will focus on the precise behavioural tasks employed as well as the way in which the behavioural data was analysed. In addition it will outline the basic EEG recording parameters for all the experiments as well as details of how this data was processed and analysed.

General procedure

In each experiment participants were recruited by means of poster advertisement. This consisted either of posters around the university advertising for volunteers, or posters targeted at first year undergraduate students aiming to collect course credits for a participation recruitment scheme. Participants received either course credits or £15 compensation for each experimental session. Only right-handed individuals between the ages of 18 and 40 were eligible to participate in the experiments. Participants were not informed of the exact nature of the study in which they were participating. They were simply informed that the experiment was exploring EEG correlates of motor preparation and inhibition. This was so that they would not guess about the presence of the unconscious stimuli.

When participants expressed an interest in participating, they were sent an information sheet describing the EEG procedure and any associated risks. They were given the opportunity to ask questions about the procedure before confirming that they would like to participate. Participants were also asked to complete a consent form at the beginning of the session which confirmed that they had fully understood the description of the procedure, as well as being asked a number of medical questions. Any individuals with a history of epilepsy or currently taking any psychoactive drugs (such as anti-

depressants) were excluded from the study. Participants with corrected vision were asked to wear glasses or contact lenses during the experiment.

After completing the consent forms the participants were prepared for the EEG recording. Further details of the EEG recording procedure are described in a later section of this chapter. Participants were then seated in an electrically shielded, dimly lit room for the duration of the experimental session. In each experiment the participants were seated 100cm from the stimulus presentation screen. At the beginning of the session the distance from the participant's head to the computer monitor was measured and the seat moved so that they were the correct distance. Participants were asked to move as little as possible during the experiment, so as to maintain the correct distance from the screen (as well as to avoid movement related EEG artefacts). The behavioural tasks were presented using E-prime version 1.1. Screen refresh rate was set to 60Hz. Stimulus presentation was synchronised with this refresh rate such that stimulus presentation was always in multiples of 16ms (one screen refresh). The timing of the sequence was verified by an external light meter connected to the EEG recording system. Additional timing data was also obtained from E-prime.

In each of the five experiments participants completed a go/no-go and a two or three alternative forced-choice task. Experiment 1 was conducted over two sessions, with the sessions separated by exactly 24 hours. Experiments two to five were conducted in a single experimental session. At the beginning of the experiment participants received instructions regarding the go/no-go task and were given the opportunity to ask any questions. The go/no-go task was combined with a masked priming paradigm such that unconscious masked primes were presented prior to the target stimulus. In addition to the go/no-go task a forced-choice task was used to assess visibility of the primes. In the first experiment the participants completed blocks of the forced-choice tasks during each of the two sessions of the experiment. This meant that they were aware that the primes were being presented when they were completing the go/no-go task. In each of the

other four experiments participants were blind to the presence of the primes until all go/no-go trials had been completed.

The Go/No-Go Task

In this task participants were asked to simply press a button in response to one stimulus and refrain from responding to another stimulus. In each experiment participants were required to respond to go trials as quickly as possible without sacrificing accuracy. In addition, to ensure that they were actively preparing to make a response, all go responses were required to be within 500ms. This was to ensure that participants would begin readying themselves for a response in advance of stimulus identification, and would then need to inhibit this imminent response on identification of a no-go target. In experiments one to four, participants were required to respond within 500ms of a go stimulus. In experiment 5 this was reduced to 450ms. In each experiment response hand varied on a block by block basis such that in one block participants were required to make a left hand button press to a go stimulus and in the next block they were asked to make a right hand response to a go stimulus. The starting hand was counterbalanced across participants. In experiment 3, left and right hand responses were included in each block by asking participants to respond with a different hand for two of the stimulus mappings and to make no response to a third mapping (no-go trials). The inclusion of trials with both left and right hand responses was required for calculation of the lateralised readiness potential (LRP; see later section on EEG analysis). For each experiment the stimuli requiring go and no-go responses were counterbalanced from one participant to the next (or one session to the next). For example in the final experiment half the participants were required to make a go response to left pointing arrows, the other half made a go response to right pointing arrows.

Masked primes were presented prior to the target stimuli on the go no-go task. These prime were congruent, incongruent or neutral with respect to the target stimulus. For example a no-go prime followed by a no-go target would be classed as a congruent no-go condition. Table 4.1 shows the

combinations of different prime and target combinations used for the experiments. This basic structure was similar for all five experiments with the exception that in experiment 3, additional conditions were present due to the manipulation of response hand. Experiment 1 also included the addition of no prime trials which were excluded from future experiments in order to maintain sufficient trial numbers in the other conditions.

Table 4.1: Trial types for experiments 1,2,4 and 5

Target Type	Prime Type			
	Go	No-Go	Neutral	No Prime*
Go	Congruent Go	Incongruent Go	Neutral Go	Go*
No-Go	Incongruent No-go	Congruent No-go	Neutral No-go	No-go*

*No prime trials were only presented in experiment 1

The go/no-go task was largely the same in each of the five experiments. In each experiment the sequence always began with a fixation cross (+), presented in the centre of the screen for 700-800milliseconds (ms). Primes were always presented for 16ms (one screen refresh) and the target stimulus was presented for 100ms. The exact sequence between these two events varied from one experiment to the next depending on the particular masking stimuli that were used and will be outlined in more detail in the experimental chapters.

Visual feedback was presented on each trial for correct responses (hits) and incorrect non-responses (misses) to go trials, as well as incorrect responses (false alarms) and correct non-responses (correct rejections) to no-go trials. After an incorrect response participants were presented with MISS for misses or INCORRECT for false alarms; after all correct responses CORRECT was presented in the centre of the screen. Following the visual feedback a blink pause was presented before the commencement of the next trial. This blink pause varied in length from 800ms to 1200ms for the five experiments. Participants were informed that they should avoid blinking

during the trial and that they should try to blink only in the blink pause. This was to ensure that the EEG was not contaminated by blink artefacts.

Assessing Prime visibility

In experiment 1, participants were informed prior to the experimental sessions that a masked prime was presented on some trials. Participants were asked to report whether they could see the masked primes. In experiments two to five, participants were not informed of the presence of the masked primes until after all go/no-go trials had been completed. Immediately following the go/no-go task participants were asked the following questions about the primes: (1) did you notice that there were stimuli presented prior to the target stimuli? (2) Could you tell what they were? (3) Did you notice anything flicker on the screen? The exact sequence of stimuli was then presented to the participants in slow motion with at least one of each prime condition presented. Following this slow motion sequence participants were again asked if they had seen these primes during the go/no-go task.

It was then explained to the participants that in the subsequent task they would be presented with this same sequence (except in some cases with the target omitted) and that their task was now to respond to the prime. In all five experiments participants were asked to complete a prime identification task, where they had to choose which prime had been presented on each trial. In addition, in experiment 1, participants were asked to complete a prime detection task, where they had to detect whether or not a prime of any sort had been presented on each trial, or if no prime was presented. In each of these tasks participants were able to respond without time restriction and had to make a choice on each trial. Participants were informed before the task that although it may seem very difficult, many people are able to successfully identify the prime despite believing that they had not seen it. Participants were told to keep focused on the task and that if they were unsure about how to respond, to let their instincts guide them into making a response. Feedback was presented on each trial to help keep participants

interested in the task and to ensure that they were able to use any visual cues from the primes to help them reliably identify the correct response. The precise details regarding the number of trials in each block and the number of blocks varied from one experiment to the next and will be specified in the relevant experimental chapters. Following the prime identification task participants were asked to report whether they had been able to see the primes during this part of the experiment.

Stimuli

The precise stimuli varied from one experiment to the next. This was the major difference between each of the experiments. Full justification for the use of the different stimuli will be given in the experimental chapters. However, this section will provide a brief introduction to the different classes of stimuli used in the different experiments. Experiment 1 and 5 utilised pattern masking stimuli. With these stimuli the mask is presented overlapping the prime. The mask normally consists of a complex pattern that makes the features of the prime more difficult to extract. The first experiment used a mask that shared all physical characteristics with the prime, so that all of the lines present in the prime were also present in the mask. The use of this type of mask is particularly effective in reducing visibility of the prime (Breitmeyer & Ogmen, 2000; Lleras and Enns , 2004) In experiment 5, two different pattern masks were generated that consisted of random chequerboard type patterns made up of two shades of grey as well as black and white. These patterns formed a rectangle which covered the area in which the primes were presented. These masks were presented both before and after the prime.

In experiments two, three and four, metacontrast masking stimuli were used. The mask in metacontrast masking differs somewhat from that used in pattern masking. In metacontrast masking the mask does not actually cover any part of the prime; rather the internal contours of the mask just touch the external contours of the prime. The simplest example of such a stimulus set up is a prime consisting of a small circle, with the mask consisting of a

doughnut shape (a larger circle with a small circle cut out). The prime would then fit exactly into the shape in the centre of the mask. In experiments two and three, two stimuli were presented on each trial. These stimuli were either diamond shapes or square shapes. Unbeknownst to the participants primes were presented in advance of these stimuli such that they filled the spaces inside the contours of the target shapes. One major difference between this procedure and the procedure employed with pattern masking is that no additional mask is presented – the target stimulus acts as both the mask and the target. In experiment 4 a metacontrast paradigm was also employed but on this occasion a single stimulus was presented in the centre of the screen. In addition, in this experiment a separate target stimulus was presented which did not act as a mask, making the sequence more similar to experiments one and five.

Another difference between metacontrast masking and pattern masking is that the former usually produces U-shaped masking functions while the latter normally produces monotonic masking functions (cf. Breitmeyer & Ogmen, 2000). This means that for metacontrast masking procedures, optimal masking is normally achieved at non-zero inter-stimulus intervals between the prime and the mask. There is some debate as to the exact cause of this U-shaped masking function (Breitmeyer & Ogmen, 2000; Francis, 2000; Herzog, 2007), which can also be observed in certain situations using pattern masks. In this thesis, experiments two and three included a 49ms inter-stimulus interval between the prime and the mask and experiment 4 used a 16ms interval. In each experiment this inter-stimulus interval was found to be sufficient in eliminating awareness of the mask both in pilot testing and in the experiments themselves.

Behavioural analysis

Initial behavioural analysis in each experiment focused on the visibility of the primes. Subjective measures of awareness are presented in tables outlining the number of participants responding yes and no to the questions described

above. Objective measures of prime visibility were analysed using participants raw scores on the forced-choice task, which were compared to chance performance using single sample t-tests. In addition, d' values were calculated in excel using the formula found in Stanislaw & Todorov (1999). These values give an estimate of discrimination performance independent of response-bias. For example if a participant performs at 51% accuracy but responds with a left button on 90% of trials, this calculation will account for the response bias by weighting the d' value dependent on the asymmetry of the response distribution. Thus in most cases d' will give a similar measure of discrimination performance, but it will be more sensitive to occasional correct discrimination when a participant responds predominantly with one hand. A d' of zero indicates that performance on the discrimination task was at chance, thus d' scores were compared to zero using single sample t-tests.

Analysis of performance on the go/no-go task includes tables showing mean reaction times for go trials as well as error rates for both go and no-go trials. One-way ANOVA was conducted on reaction times for go trials and error rates for go and no-go trials separately. Initial analysis focused on the results obtained with all participants before further analysis excluding those people who were deemed to have some residual awareness of the primes (where appropriate). Finally in order to show that any effects were independent of consciousness of the stimulus, correlations were computed between performance on the prime identification task, and the amount of priming observed in the go/no-go task. Prime identification performance was measured using the raw accuracy scores and d' scores, as well as absolute measures of both these variables. The absolute values were calculated to provide a measure of the difference from chance regardless of whether the participant was slightly above or below chance. For d' values this simply involved taking the absolute values of d', while for the raw accuracy scores, each participant's score was compared to chance (50%), with the absolute value of this difference then calculated. Priming measures were calculated as the pair-wise differences between each of the conditions (e.g. congruent go vs. incongruent go, congruent go vs. neutral go and neutral go vs. incongruent go) for both reaction times and error rates. In addition, the

results between 'aware' and 'unaware' participants were compared checking whether priming effects were maintained even when excluding those participants who may have had some residual awareness of the masked primes.

EEG recording and processing

EEG was recorded from 64 scalp electrodes and a further six external electrodes using the BioSemi Active Two system. External electrodes were attached to the left and right mastoids, with the other four electrodes attached to the left, right, above and below the right eye for measuring eye movement and blinks. Data was recorded unreferenced and unfiltered at a digitisation rate of 1024 Hz. All data was filtered offline with a 0.3 Hz high pass filter and a 30Hz low pass filter. Data was segmented relative to the onset of the target stimulus, with the 100ms preceding prime onset used as a baseline. All data was re-referenced to the average of the two mastoid electrodes prior to analysis. All segments containing blink or other artefacts were removed prior to averaging. This artefact rejection was completed in a semi-automated manner such that an initial amplitude criterion of +/- 80 μV in the vertical electro-oculogram identified possible blinks. After visual inspection this criterion was adjusted independently for each participant and trials on which EEG amplitude exceeded this criterion were removed. A second semi-automatic rejection criterion of +/- 50 μV maximum amplitude and a maximum gradient of two μV per sampling point was applied to the horizontal electro-oculogram to detect horizontal eye movements. Finally all scalp electrodes were checked against a criterion +/- 120 μV as well as a maximum difference within a segment of 150 μV and a gradient of three μV per sampling point. This final check was to remove any trials containing large hardware artefacts such as slow drifts or spikes in the EEG. In addition, all go trials with no response within 500ms of stimulus onset and all no-go trials containing a response within 600ms of stimulus onset were excluded from analysis. This was so that only correct go and no-go trials would be included. EEG pre-processing was completed using Brain Vision Analyzer, with later grand averaging and data handling completed using

custom built Matlab scripts. Statistical analysis was conducted using Statistical Package for the Social Sciences (SPSS).

ERP Figures

In each experiment ERP waveforms are presented from 400ms prior to stimulus onset until 600ms after stimulus onset. The values on the y-axis represent the amplitude of the ERP measured in μV with positive values plotted upwards. The values on the x-axis represent time relative to stimulus onset measure in milliseconds. The x-axis and y-axis intersect at target onset (0ms) and at 0 μV. Additionally, a dashed line is presented intersecting the x-axis representing the time of the onset of the prime. Topographic maps are presented showing average amplitudes (in μV) for the time window specified below the map. Specific effects in subsequent analyses are often supplemented by bar graphs showing average amplitude in a particular time window. In these figures the average amplitude is plotted on the y-axis (in μV), with the different electrodes on the x-axis. Different conditions are signified by bars shaded in different tones.

EEG Analysis

LRP analysis

LRP was calculated by averaging the difference in activity between electrode C3 and C4 for left and right hand responses using the equation below.

$$LRP = [\text{Mean } (C_4\text{-}C_3)_{\text{left-hand movement}} + \text{Mean } (C_3\text{-}C_4)_{\text{right-hand movement}}]/2$$

LRP onset was determined using a 50% relative criterion method in combination with the jackknife procedure. The 50% relative criterion method takes the point at which the LRP reaches 50% of its maximum, as the onset. The jackknife procedure involves applying this method to an average of all participants minus one participant at a time, rather than taking the onset from each individual participant (Miller, Patterson and Ulrich, 1998; Ulrich and Miller, 2001). These values are then used in the statistics, with the final F value corrected for the decreased error term produced by the procedure. It

is necessary to adjust the F value, since applying the jackknifed onset values to the factorial analysis will result in much smaller estimates of the standard error, and thus a much larger F value. This adjustment simply involves dividing the observed F value by $(n-1)^2$, where n is the number of participants (see Ulrich & Miller, 2001 for a proof). Similarly, for subsequent t-tests the error term was adjusted according to the formula presented in Miller et al. (1999). This procedure is used due to the particularly low signal to noise ratio of the LRP, since attempting to calculate measures of onset for individual participants may result in criterion being falsely met due to noise, or alternatively not being met at all in some participants (Miller et al., 1998). Morkadoff and Gianaros (2000) have run a number of simulations for different methods of determining the onset of an LRP and found that the 50% relative criterion method combined with the jackknife procedure gave the most accurate measure of significant differences between LRP onsets. It is important to note that since the onset is given as 50% of the amplitude it will give a rather late estimate of the onset of the LRP. However, while this measure provides a somewhat inaccurate estimate of the actual onset, it is effective at determining the difference in onset between conditions (Miller et al, 1999). In addition to onset analysis, amplitude analysis was also conducted for the LRP to explore any early separations in the LRP that were not picked up by the onset analysis.

Frontal no-go N2 and P3 analysis

Initial analysis in each experiment focused on the specific hypothesis that the no-go N2/P3 complex would be influenced by the unconscious primes. N2 difference waveforms are presented at electrode FZ as the difference between each condition and the neutral go condition. Since the neutral go condition always contained a prime that was neither congruent nor incongruent to the target, it should not influence the onset of activity associated with target processing. Thus computing the difference waveform between all other conditions and the neutral go condition should reveal any early differences related to the go and no-go primes alongside the normal no-go N2. The neutral no-go difference ERP provides a baseline, since it is the comparison of neutral no-go and neutral go trials and thus will reflect

go/no-go differences where no relevant unconscious information was presented. The congruent and incongruent difference waveforms should reveal any early differences associated with the processing of the prime, as well as any modulation of the target-related ERP components. These no-go difference waveforms were subjected to amplitude analysis focused around the peak of the N2 and P3 components to determine if the average amplitudes varied as a function of the unconscious prime.

N2 and P3 topographies were initially explored by computing scalp maps for the time windows where these components were evident, with further analysis conducted as part of the more comprehensive analysis across the scalp (see below). N2 onset was determined by a segmented regression technique (see Falkenstein et al., 1999). This technique involves fitting two regression lines to the N2 waveforms. The start of the first regression line is fixed to the onset of the prime. The end of the second regression line is fixed to the time at which the N2 reaches its maximum amplitude. The remaining four parameters (the time at which the two lines intersect and the amplitude at this point, as well as the amplitude at the beginning of the first line and the end of the second) are allowed to vary until the two regression lines have the minimum residual sums of squares. The time at which the two lines intersect is then given as the onset of the component. This procedure was conducted on each individual participant for every condition to determine the onset of the N2. This procedure was conducted using custom built scripts in Matlab.

To determine if any observed modulation of the no-go N2 and P3 was associated with conscious awareness of the prime, correlations were calculated between performance on the prime identification task and the amount of priming observed in the ERP components. As with the behavioural analysis, prime identification performance was defined both in terms of raw scores and absolute scores for d' and accuracy. Similarly, priming measures included all pair-wise comparisons for N2 onset and latency and P3 latency. Furthermore, participants with higher identification performance were compared with those with lower performance to confirm

the absence or presence of priming effects using only those participants who showed no discrimination in the identification task.

Early visual ERP effects

Early ERP effects were analysed at electrode Oz as well as lateral electrodes O1/O2 and PO7/P08. Initial analysis focused on the early P1 and N1 peaks observed at Oz to determine if any differences were evident dependent on the congruency of the prime with respect to the target stimulus. Since in each experiment (except Experiment 3), the target that was allocated to go or no-go was counterbalanced across participants the grand average waveforms would not show any effects of specific target type or prime type. However, if there is an effect of prime congruency on either the N1 or P1 then this should show up in the grand average ERP waveforms.

To explore specific effects of the physical prime and target stimuli, grand average ERPs were calculated reflecting the physical stimuli presented on each trial. For example, in Experiment 1 congruent go trials for participants instructed to respond to a right pointing arrow consisted of a right arrow prime followed by a right arrow target. For those participants who were instructed to respond to a left pointing arrow, this same stimulus configuration (right arrow prime followed by right arrow target) was present for congruent no-go trials. Therefore, ERPs that reflected right-right conditions where formed from congruent go trials for some participants and congruent no-go trials for others. Similarly left-left trials were computed from these same two conditions but this time using the participants from the alternative response mapping. Right-left and left-right ERPs were formed in a similar way using incongruent go and incongruent no-go conditions. This analysis would allow disentangling of ERP effects associated with left and right pointing primes and targets.

For each condition, difference waveforms were calculated between electrode 01 and 02 as well as electrode PO7 and PO8. These difference waveforms thus reflect the difference in activity between the left and right hemisphere over occipital and parietal electrodes. This is particularly important since in

many of the experiments participants were responding to stimuli presented in one visual field or the other. Since these difference waveforms were computed for each condition, this analysis comprised a repeated measures ANOVA with prime type (left, right, neutral) and target type (left, right) as repeated measures factors. However, in order to explore whether response mapping also influenced the relationship between the physical stimuli a between participants factor of response mapping was also included. A significant interaction between prime type, target type and response mapping would signify that any posterior asymmetries were dependent on the meaning of the prime and not simply its physical attributes. For example, a left prime may produce a significant right sided component only when the right prime acts as a go stimulus and not when it acts as a no-go stimulus. Such an observation would suggest that prime-related asymmetries were dependent on whether the stimulus was a target or not and therefore might reflect an N2pc component, exhibited contralateral to target stimuli (see Chapter 2). Similarly, a target by response mapping interaction would reflect an N2pc to the target.

Go/no-go differences

Go/no-go differences were explored in more detail in each experiment by presenting ERP waveforms at a number of electrodes across the scalp to show the topographic distribution of the effects. Statistical analysis was conducted by means of repeated measures ANOVA with five factors. The first two factors were typically prime type (go, no-go, neutral), target type (go, no-go). Two further factors: anterior-posterior (Frontal polar, Frontal, Fronto-Central, Central, Parietal and Occipital), and hemisphere (left, right) explored the spatial distribution of the effects. The fifth factor in the analysis was time, where the data entered into the ANOVA was the average amplitude at each electrode site for the specified time window. In experiment 1 there were four time windows which were explored to capture effects of the prime, prime-mask effects, and N2 and P3 time windows. In experiments two to five, three time windows explored early prime-related effects, the N2 and P3 respectively. If the initial ANOVA showed no effects involving hemisphere then further analysis was conducted at midline electrodes only. All ANOVA

effects are reported with a Greenhouse-Geisser correction applied. As Dien and Santuzzi (2004) point out, ERP factorial analysis often violates the sphericity assumption for repeated measures ANOVA. By correcting the degrees of freedom using the Greenhouse-Geisser correction for all ERP comparisons, this should reduce the effect of this possible violation. Where effects of hemisphere were observed, follow up analysis was conducted to determine their precise nature. Where a significant four way interaction was observed at midline electrodes this was followed up with separate three-way ANOVA for each time window. Significant effects in these time windows were then explored using contrasts and t-tests with uncorrected p values greater than 0.001 accepted as significant.

LRP and go/no-go differences

Finally, since in each experiment the major go/no-go differences were explored collapsed across right and left hands it is possible that go/no-go differences could be an artefact from lateralised movement-related activity over the motor cortex (see Praamstra and Seiss, 2005); consequently LRPs were calculated separately for each response hand to show that the effects persist over central and lateral electrode sites for both response hands.

Chapter 5

Experiment 1 – An EEG investigation of go/no-go inhibition in the negative compatibility effect.

Introduction

The aim of this experiment was to adapt Eimer and Schlaghecken's (1998) masked priming paradigm to a go/no-go task to explore possible unconscious modulation of the no-go N2 and P3. Eimer and Shlaghecken (1998) presented participants with left or right pointing double arrows and asked them to make a right hand response to right pointing arrows and a left hand response to left pointing arrows. Unconscious masked primes were presented 116ms in advance of the target stimulus. Surprisingly they found a negative compatibility effect (NCE); such that when the prime was congruent with the target (i.e. pointed in the same direction), reaction times were *slower* than when the primes were incongruent with the target. They suggested that this reversal of priming is accounted for by exogenous inhibition of the unconscious prime. They present evidence from the lateralised readiness potential in support of their claim. Initial activation of the LRP for the primed hand was quickly replaced by an opposite going LRP. Since the LRP measures hand specific response preparation, they argue that this reversal shows inhibition of the primed response. Such a mechanism could work to prevent automatic responses to insignificant changes in the environment by inhibiting partial response activation that is no longer on line.

However, the exact mechanism behind this NCE has been questioned in recent years (see also chapter 3). Lleras and Enns (2004) suggest that the reversal of the priming effect is caused by an interaction between the prime and the mask, whereby the physical characteristics of the mask reverse the effects of the prime. Since the mask is a compound of left and right pointing arrows (see figure 5.1), the onset of the mask effectively involves the addition of the arrows pointing in the opposite direction to those presented in the prime. Lleras and Enns (2004) argue that it is the updating of the visual

scene from the prime presentation to the mask presentation which causes the reversal of priming effect. Similarly, Verleger et al. (2004) showed that the reversal of the LRP was only evident when using related masks and not when using unrelated masks. In addition, they show that for related masks the LRP reversal appears to be accounted for by an increased activation over the motor cortex contralateral to the un-primed hand following the onset of the mask. They claim that this shows that rather than reflecting inhibition, this LRP reversal indexes preparation of the un-primed hand caused by the interaction between the prime and the mask.

Pramstraa and Seiss (2005) also measured EEG during a similar task to that employed by Eimer and Schlaghecken (1998). They explored whether an N2 was observed for congruent go trials. An N2 here would reflect the conflict induced by the reversal of the initial prime effect. They found no genuine N2 on these trials and thus concluded that the inhibition involved in producing the NCE is not mediated by frontal control mechanisms. They suggest that the NCE is caused by reciprocal inhibition between the response alternatives and that it is regulated from within the motor system. It is also noteworthy that they did find a pseudo N2 over central electrodes which appeared to be caused by averaging together right and left hand responses. Since in the final stages of motor activation an asymmetry is observed in the negative readiness potential (indexed by the LRP), an increased negativity would be expected over the left hemisphere for trials with a right response, and over the right hemisphere for trials with a left response. When averaging together these two responses this produces an impression of negativity over central electrodes.

The experiment described in this chapter explored behavioural and ERP priming effects in a go/no-go task to allow more direct examination of unconscious modulation of frontal inhibition/control mechanisms (see chapter 2 for more details). Participants were instructed to respond as quickly as possible to arrows in one direction, and refrain from responding to arrows pointing in another direction. The stimuli (see figure 5.1) were identical to those originally employed by Eimer and Schlaghecken (1998). As discussed

above, this particular prime-mask combination is known to produce a negative compatibility effect where the mask causes the reversal of the activation induced by the prime. In this chapter the initial effect of the prime will be described as the prime effect and the reversal of this effect will be termed the prime-mask effect.

Table 5.1: Prime/prime-mask effects and hypotheses for experiment 1.

Target Type	Prime/Prime-Mask Effect (*Hypotheses*)			
	Go/No-go	No-Go/Go	Neutral	No Prime
Go	Congruent Go/ Incongruent Go *B1. Slower reaction times.* *E1. Early N2 associated with no-go prime-mask effect.*	Incongruent Go/ Congruent Go *B2. Faster reaction times.* *E2. Early N2 associated with no-go prime.*	Neutral Go	Go
No-Go	Incongruent No-go/ Congruent No-go *B3. Fewer false alarms.* *E1. Early N2 associated with no-go prime-mask effect.* *E3. Reduced target-related N2.*	Congruent No-go/ Incongruent No-go *B4. More false alarms.* *E2. Early N2 associated with no-go prime.* *E4. Increased target-related N2.*	Neutral No-go	No-go

Table 5.1 shows the prime effect and the prime-mask effect for the different conditions in this experiment. The prime type and prime-mask congruency are written in black with the prime-mask effect in red. Since neutral and no prime trials have no initial prime effect, they also have no prime-mask effect. However, for the two conditions with active primes the initial effects of the prime are reversed. In all analysis and figures in this chapter, conditions are labelled relative to the prime congruency not the prime-mask effect. Where conditions are described and interpreted in terms the prime-mask effect, they

will be labelled as such (for example no-go prime-mask effect). Each cell also contains the main hypotheses for that condition with respect to the neutral prime condition. Hypotheses B1 to B4 outline the predicted behavioural priming effects, while hypotheses E1 to E4 specify the predicted ERP effects.

Hypotheses

Reaction times and error rates are predicted to follow the NCE with fastest reaction times to incongruent go trials and slowest reaction times to congruent go trials (Hypotheses B1 and B2 in figure 5.1). Similarly false alarm rates should be reduced for incongruent no-go trials and increased for congruent no-go trials (Hypotheses B3 and B4 in figure 5.1). In addition, if the unconscious primes are able to facilitate inhibition or control functions then they should affect the no-go N2/P3 complex. If this effect is related to unconscious modulation of the frontocentral no-go N2/P3 complex, then these components should be maximally distributed over frontocentral electrodes. This modulation could occur either in terms of latency of these components, or in terms of amplitude. For example, if the unconscious information (the prime-mask effect) codes for a go response while the target codes for a no-go response (congruent no-go condition) one might expect greater N2 amplitude, reflecting the fact that inhibition was more difficult to achieve, since the response was already partially activated (Hypothesis E4 in table 5.1). Conversely, N2/P3 amplitude should be reduced for incongruent no-go (congruent no-go prime-mask effect) trials in comparison to neutral no-go trials, if the prime-mask effect was successful in initiating the processes associated with these components (Hypothesis E3 in table 5.1). Similarly, if the unconscious prime in combination with the mask codes for a no-go response then this might shift the N2 or P3 earlier in time (Hypothesis E1 in table 5.1). Importantly, if the unconscious information in the prime is able to *directly* initiate these components, rather than facilitating performance through priming of processing of the target stimulus then there should be some modulation of the ERP waveforms at frontocentral electrodes which is entirely determined by the nature of the prime, regardless of the target (Hypotheses E1 and E2 in table 5.1).

Method

Participants

Sixteen paid volunteers (eight male and eight female) were recruited by means of poster advertisement. Participants received £15 in compensation for their time. All participants were right handed and had normal or corrected to normal vision. The mean age of participants was 24 years and nine months, with a range of 18 to 32 years.

Experimental Procedure

All participants completed 16 blocks of the go/no-go task and two blocks of the prime detection and prime identification trials in each of the two experimental sessions. The two sessions were always separated by exactly 24 hours to ensure that they were both conducted at the same time of day, and not too far apart. There were four practice blocks at the beginning of each session; two go/no-go, one prime identification and one prime detection.

The go and no-go stimuli were left and right pointing double arrows (>> and <<). These were presented in black on a white screen positioned 100cm from the participants, and measured 3.5cm across and 1.9cm from top to bottom. Each stimulus was used as go in one session and no-go in the other session, with the order counterbalanced across participants. The participants were informed that that they had a time limit of 500 milliseconds (ms) to respond to the go stimuli and that they should react as quickly as possible without sacrificing accuracy. Masked primes were presented prior to some of the target stimuli. These were congruent with, incongruent with, or neutral (<> or ><) to the target stimulus. There were an equal number of each of these trial types and trials with no prime presented. The primes were masked by the two stimuli superimposed over one another (⋙⋘). The precise sequence of stimuli is presented in figure 5.1. Each go/no-go block contained 64 trials (16 from each condition) presented in a random order. The response hand was varied from block to block, with the starting hand counterbalanced across participants.

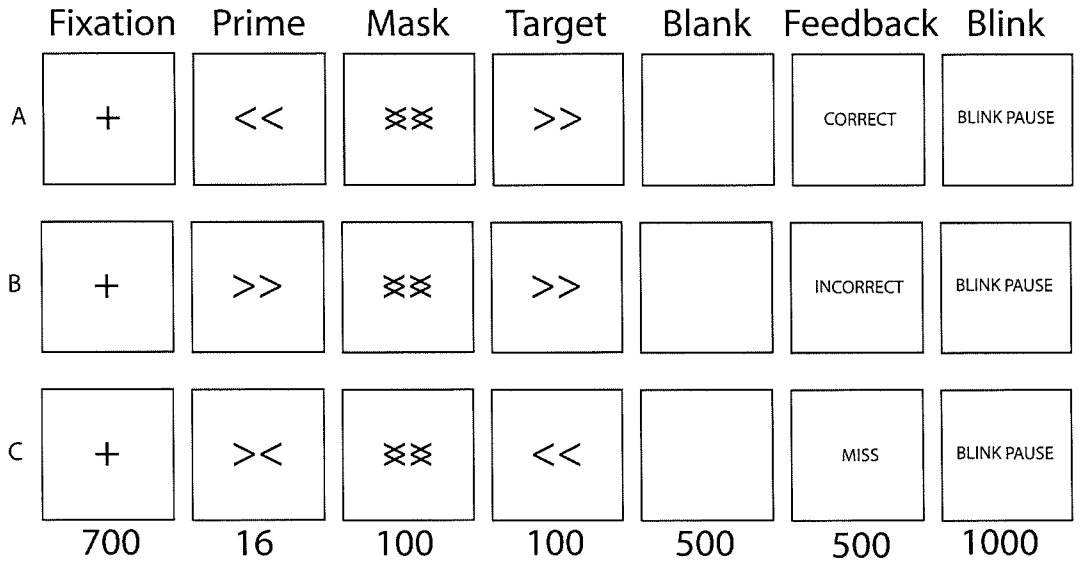

Figure 5.1: Stimuli for experiment 1. (a) an incongruent trial (b) a congruent trial and (c) a neutral trial. The feedback screens signify the three different types of feedback received during the task.

In the prime detection task one of the primes (<<, >>, >< or <>) was presented on half the trials and no prime was presented on the other trials. Participants were required to indicate, without time restriction, whether the prime was present or absent. The timing of the stimuli was identical to that used in the go/no-go task, but no target stimulus was presented (in accordance with the procedure employed by Eimer & Schlaghecken, 1998). In the prime identification task participants were required to choose whether the prime was left pointing or right pointing. Half the trials contained the left pointing arrow prime and half contained the right pointing arrow prime. As well as the trials where the prime was presented for 16ms, some longer prime presentation trials (48ms) were included. In addition to recording the responses on these tasks the participants were asked at the end of the block whether they felt they could see the masked primes.

Behavioural Results

Awareness of Primes

Of the sixteen participants only two reported any ability to detect or recognise the masked primes when presented for 16ms. All other participants

indicated that they were randomly guessing the answer. T-tests showed that d' values for the prime detection and prime identification tasks using all sixteen subjects did not significantly differ from zero. Prime detection d' values ranged from –0.51 to 1.84 with a mean of 0.219 ($t(15)=1.556$, $p=0.141$). Prime identification d' values ranged from –1.57 to 0.91 with a mean of -0.283 ($t(15)= -2.033$, $p=0.06$). Despite the fact that the overall d-values did not significantly differ from zero, a number of individual scores were high enough to suggest some awareness of the primes. Participants (n=8) with prime identification d-values more than 0.16 away from zero, or prime detection d-values more than 0.51 away from 0 were classed as 'aware' (this corresponded to approximately 47% to 52% correct and 45% to 55% correct for the two tasks respectively). This left a total of eight participants who had no awareness of the primes; these were classed as 'not aware'. T-tests for prime detection ($t(7)=1.107$, $p =0.31$) and prime identification ($t(7)=0.79$, $p =0.45$) showed no significant deviation from zero in these participants. Prime detection rates for the aware participants significantly differed from zero($t(7)=2.683$, $p<0.05$), with prime identification performance approaching significance($t(7)=-2.15$, $p =0.069$).

Performance on the long duration primes (measured by d') did not significantly differ from chance (M=-0.6; $t(15)=-0.7$, $p=0.5$). However on closer examination of participants' scores it appeared that while some subjects were performing significantly above chance, others were performing below chance, with only two subjects performing near to chance (40% to 60% correct). Nine subjects appeared to perform well below chance, with the other seven performing above chance. A further t-test was run on the absolute d' scores to give the difference from chance independent of whether this was above or below chance. This test confirmed that performance on the task was significantly better than would be expected by chance (M=3.83; $t(15)=5.6$, $p<0.001$). Similarly, splitting the group between above and below chance performers and running t-tests on the two groups separately using their forced-choice scores showed that the above chance group performed significantly greater than 50% (M=83%; $t(6)=8.8$, $p<0.001$) and the below chance group performed significantly below 50% (M=13%; $t(8)=-3.2$, $p<0.05$).

It is important to note that dividing the groups based on whether they performed above or below chance is likely to substantially increase the probability that these groups differ from chance regardless of their actual performance. The t values must therefore be treated with some caution, but examination of the mean scores of the two groups at 83% and 13% show that these scores were not simply marginally above or below 50% performance and are therefore unlikely to be merely due to natural variation from chance.

Priming

The behavioral results on the go/no-go task replicated Eimer and Schlaghecken's (1998) NCE, with fastest reaction times (in milliseconds) for incongruent go trials and slowest reaction times for congruent go trials with reaction times to neutral trials and no prime trials in between (see table 5.2.1). A 2x4 mixed ANOVA with prime congruency as a four level repeated measures factor and awareness as an independent measures factor revealed a significant main effect of prime congruency ($F(3,42)=91.1$, $p<0.001$). However, there was no main effect of awareness ($F(1,14)=0.05$, $p=0.84$) and no significant interaction between awareness and prime congruency ($F(3,42)=1.42$, $p=0.25$). Subsequent t-tests (corrected for multiple comparisons) revealed that all four prime congruency conditions were significantly different from the other three conditions with the exception that no prime trials did not significantly differ from neutral prime trials ($t(15) = 2.1$, $p>0.05$). In addition, reaction times were not significantly different for the two different types of neutral trials ($t(15) = 1.1$, $p=0.31$).

Table 5.2.1: Mean Reaction times *(and Standard Deviations)* for go trials

	Congruent	Incongruent	Neutral	No prime
All	395 *(21)*	338 *(25)*	359 *(23)*	362 *(22)*
Aware	401 *(15)*	337 *(22)*	360 *(13)*	360 *(13)*
Not Aware	390 *(25)*	338 *(29)*	358 *(30)*	363 *(29)*

Table 5.2.2: Mean accuracy *(and SD)* for go and no-go trials

		Congruent	Incongruent	Neutral	No prime	Total
All	Go	0.88 *(0.10)*	0.96 *(0.03)*	0.94 *(0.06)*	0.95 *(0.04)*	0.93 *(0.05)*
	No-go	0.75 *(0.15)*	0.94 *(0.06)*	0.89 *(0.07)*	0.92 *(0.06)*	0.87 *(0.08)*
Aware	Go	0.85 *(0.13)*	0.97 *(0.03)*	0.93 *(0.08)*	0.94 *(0.05)*	0.92 *(0.07)*
	No-go	0.74 *(0.16)*	0.95 *(0.04)*	0.90 *(0.06)*	0.92 *(0.05)*	0.86 *(0.07)*
Not Aware	Go	0.90 *(0.06)*	0.95 *(0.03)*	0.95 *(0.03)*	0.95 *(0.03)*	0.94 *(0.03)*
	No-go	0.76 *(0.16)*	0.93 *(0.08)*	0.89 *(0.09)*	0.92 *(0.07)*	0.87 *(0.08)*

Similarly, accuracy was lowest on congruent trials for both go and no-go targets (see table 5.2.2). A three way mixed ANOVA with prime congruency (four levels) and target type (two levels) as repeated measures factors, and awareness as an independent measures factor revealed a significant main effect of prime congruency ($F(3,42)=36.2$, $p<0.001$) a main effect of target ($F(1,14)=8.84$, $p<0.05$) as well as a target by prime congruency interaction ($F(3,42)=12.56$, $p<0.001$). Once again there was no main effect of awareness and no interaction between awareness and any other factors. Inspection of table 5.2.2 also reveals that the main effect of target was due to significantly greater accuracy for go trials than no-go trials. Subsequent one way ANOVAs were conducted to explore the interaction between target type and prime congruency. A significant main effect of congruency was present for both go trials ($F(3,42)=12.56$, $p<0.001$) and no-go trials ($F(3,42)=12.56$, $p<0.001$). Inspection of table 5.2.2 reveals that although this effect was present for both go and no-go trials, a greater cost of prime congruency was observed for no-go than go trials. Subsequent t-tests revealed that

congruent go trial accuracy significantly differed from all other conditions (at $p<0.005$ uncorrected) with other comparisons for go trials not reaching significance. Pair-wise comparisons of no-go accuracy were all significant (at $p<0.005$ uncorrected) with the exception of the comparison between no prime and incongruent trials, which failed to reach the corrected p value ($p=0.015$). Finally, there was no significant correlation between participants' performance on the forced-choice tasks and the amount of priming in the go/no-go task either for reaction times in the go trials or for error rates in the go or the no-go trials, suggesting that the influence of the masked primes in the go/no-go task was independent of their visibility.

EEG Results

ERPs were formed for each condition relative to the onset of the target stimulus for each of the sixteen participants. ERPs were formed from an average of between 160 and 200 trials per condition with a minimum of 60 trials per condition and approximately equal numbers of left (average 94 trials) and right (average 95 trials) hand response trials.

Lateralised Readiness Potential

Figure 5.2 shows grand average LRP for the four go conditions with stimulus onset at time zero (prime onset at -116ms, signified by dashed vertical line). Initial analysis focused on LRP onset for congruent, incongruent and neutral go conditions as well as the no prime go condition. ANOVA showed a significant main effect of prime congruency on LRP onset ($F(1.5,23.3)=8.79$, $p<0.01$), with subsequent t-tests showing that LRP onset was significantly earlier for incongruent trials in comparison to congruent ($t(15)=6.74$, $p<0.001$), and neutral trials ($t(15)=4.75$, $p<0.001$). In addition, congruent go trials showed significantly later LRP onset compared with neutral ($t(15)=2.89$, $p<0.05$) and no prime trials ($t(15)=4.03$, $p<0.005$). Neutral go LRP onset did not significantly differ from incongruent go LRP onset or go LRP onset.

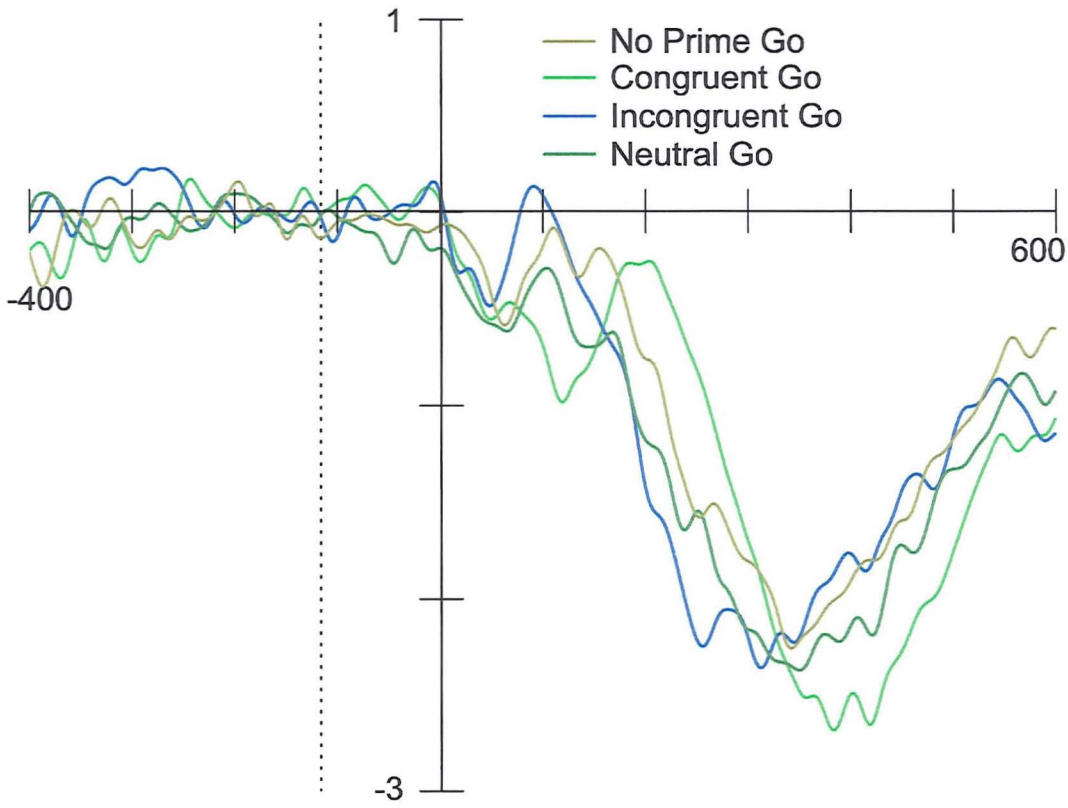

Figure 5.2: LRP for go target trials in experiment 1 relative to stimulus onset. Prime onset at -116ms signified by dashed line.

Amplitude analysis was conducted on the six conditions in which a prime was presented. These six conditions are presented in figure 5.3. ANOVA was conducted with prime type (go, no-go, neutral) and target type (go, no-go) as repeated measures factors. Awareness was included as a between participants factor to check whether any prime-related modulations were related to visibility of the primes. This analysis revealed a significant main effect of prime in the 50 to 150ms time window ($F(1.7,24)=5.9$, $p<0.05$), with a significant linear contrast ($F(1,14)=8.4$, $p<0.01$) revealing that no-go prime trials showed the least negative amplitude and go prime trials showed the most negative amplitude, with neutral trials in between. There was no significant main effect of target or target x prime interaction in this time window. Additionally, there were no significant effects involving awareness. These early prime-related separations suggest that the unconscious primes were directly initiating the motor response. From 150 to 220ms there was a significant main effect of prime ($F(1.5,20)=6.8$, $p<0.01$), with a significant linear contrast ($F(1,14)=9.3$, $p<0.01$) revealing that no-go prime trials were

now more negative in comparison to neutral and go prime trials. This reversal reflects the prime-mask effect induced by the mask. There was no significant main effect of target or target x prime interaction in this time window and no significant effects of awareness. It is important to note that the congruent no-go condition shows a partial onset of the LRP in this period, this once again suggests direct unconscious LRP activation, in this case in response to the prime-mask effect. In a 220ms to 500ms time window there was a significant main effect of target ($F(1,14)=29.8$, $p<0.001$), signifying an increased LRP for go target trials

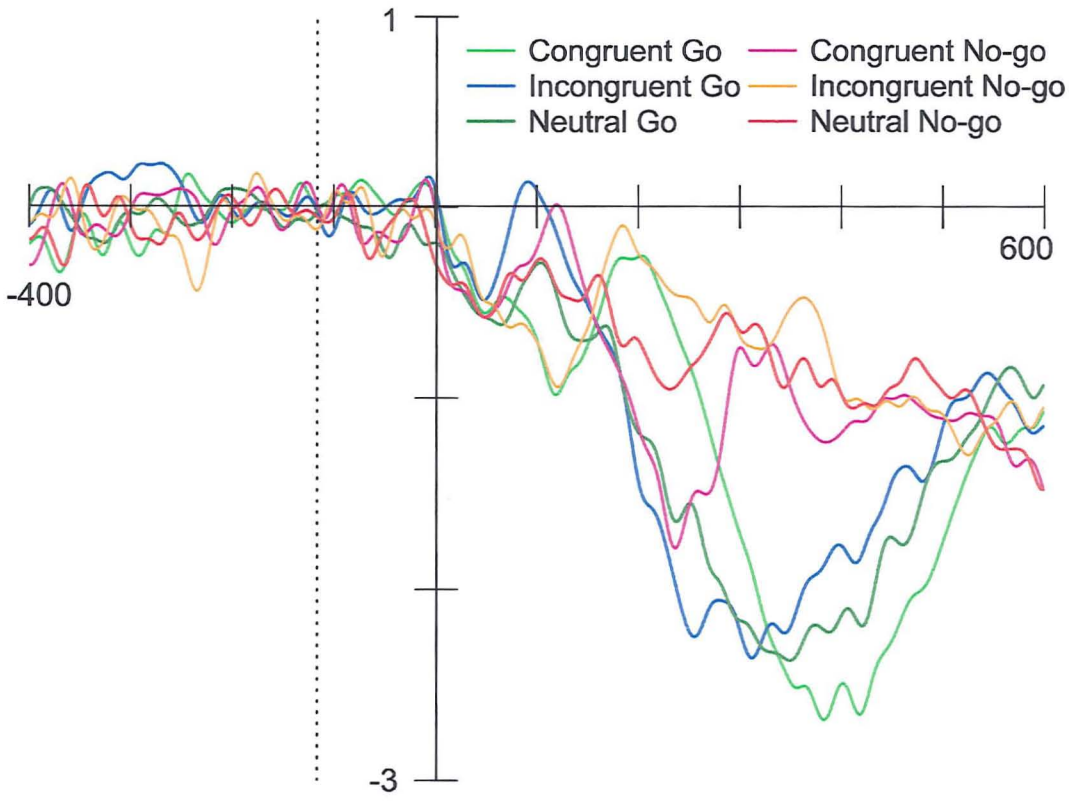

Figure 5.3: LRP waveforms for experiment 1.

Frontal No-go N2 and P3 Analysis

Initial analysis focused on the specific a-priori hypotheses outlined in the introduction, namely that the no-go N2 will vary in amplitude or onset dependent on the nature of the unconscious prime. This specific hypothesis was explored by calculating difference waveforms for each of the three no-go conditions in comparison to the neutral go condition. In addition, the difference between the no-go and go trials where no prime was presented

was included as a further baseline of the normal no-go N2 P3 complex. Analysis of these difference waveforms was conducted at electrode Fz in line with previous research showing a frontocentral maximum for the no-go N2 and P3. This analysis was initially performed with all participants regardless of their performance on the forced-choice tasks. Initial analysis focused on the onset of the no-go N2 for the four no-go conditions only. N2 onset was explored by determining the greatest negative peak in each difference waveform and calculating the onset of that peak using the segmented regression technique described in chapter 4.

Figure 5.4 shows the difference waveforms for the four no-go conditions. N2 onset for each participant was calculated using the segmented regression technique (see chapter 4). Three participants were excluded from this analysis because the N2 was not well defined enough to allow onset calculation in one or more condition. The nature of the prime modulated N2 onset for no-go trials ($F(2.13,25.6)=29$, $p<.001$). Mean onset for the incongruent (no-go prime-mask effect) no-go condition ($M = 137.1$ms, $SD = 25.1$ms) was earlier than the congruent (go prime-mask effect) condition ($M=248$ms, $SD=29.1$ms; $t(12)=10.7$, $p<0.001$;) as well as the neutral condition ($M=216$ms, $SD=38$ms; $t(12)=7.3$, $p<0.001$) and the no prime condition ($M=213$ms, $SD=31.7$ms; $t(12)=7.6$, $p<0.001$). The modulation of N2 onset was consistent with the time between the onset of the mask and the onset of the target (see Figure 1), such that the early N2 in the incongruent no-go condition occurred around 100ms earlier than in the congruent and neutral no-go conditions. There was no main effect of awareness on N2 onset.

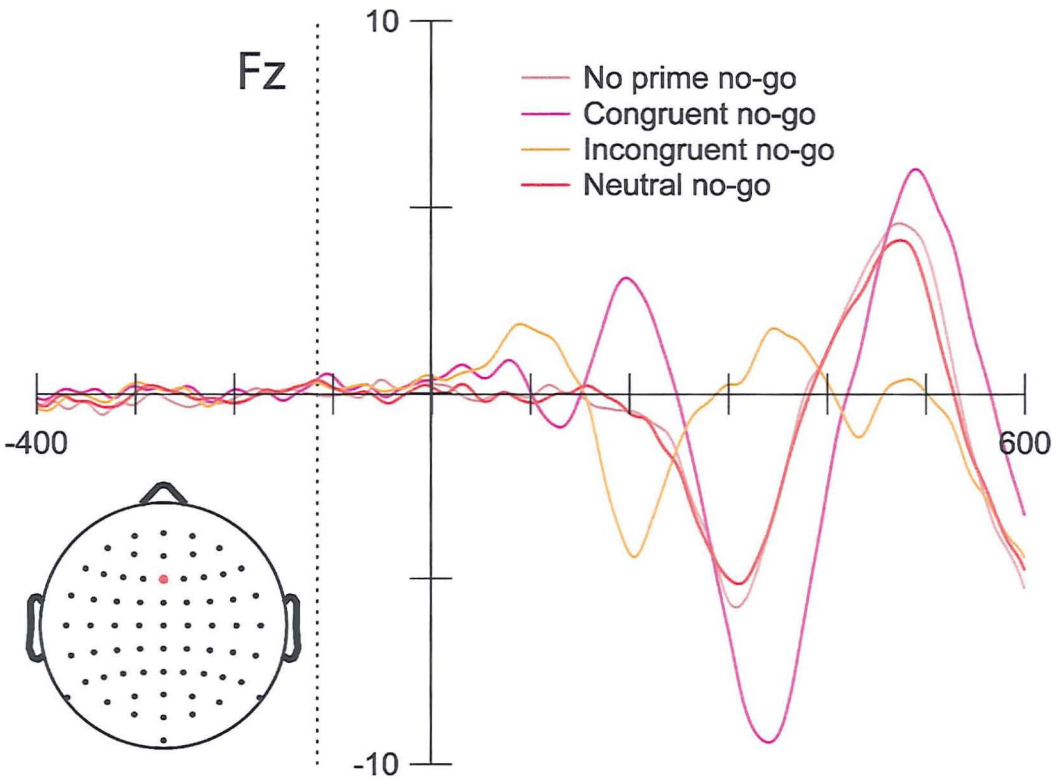

Figure 5.4: Difference ERP waveforms for the four no-go conditions at electrode Fz, with respect to target onset.

Two separate two-way ANOVAs were conducted at electrode Fz for the four no-go difference waveforms to explore the amplitude of the no-go N2 and P3. The N2 time window (250ms to 350ms) was chosen to encompass the period around the peak latency of the typical no-go N2. Similarly the P3 time window (450ms to 550ms) was meant to capture any differences in average amplitude of the no-go P3. Each ANOVA (one for each time window) included prime congruency as a repeated measures factor (congruent, incongruent, neutral and no-prime) and awareness as an independent factor.

In the N2 time window there was a significant main effect of prime congruency ($F(1.8,25.5)=13.01$, $p<0.001$), but no main effect of awareness ($F(1,14)=0.34$, $p=0.57$) and no prime type x awareness interaction ($F(1.8,25.5)=0.6$, $p=0.31$). Follow up t-tests confirmed that congruent no-go trials ($m=-5.9$; $std=4.2$) showed significantly more negative amplitude than both neutral ($m=-4.1$; $std=2.8$; $t(15)=-2.7$, $p<0.05$) and incongruent no-go trials ($m=0.25$; $std=2.4$; $t(15)=-5.9$, $p<0.001$). Similarly, neutral no-go trials showed significantly more negative amplitude than incongruent no-go trials

(t(15)=-6.4, p<0.001). Neutral no-go amplitude did not significantly differ from no-prime no-go trials (m=2.7; std=3.3; t(15)=0.25, p=0.8).

In the P3 time window there was once again a significant main effect of prime congruency (F(1.2,17.2)=16.4, p<0.001) but no significant main effect of awareness (F(1,14)=0.3, p=0.57) and no interaction between awareness and prime type (F(1.2,17.2)=1.54, p=0.24). Follow up t-tests confirmed that no-go P3 average amplitude was significantly greater for congruent no-go trials (m=4.5; std=5.8) in comparison to both neutral (m=2.09; std=3.7; t(15)=3.4, p<0.005) and incongruent no-go trials (m=-0.4; std=3.3; t(15)=-4.2, p<0.001). Similarly, neutral no-go trials showed significantly greater average amplitude than incongruent no-go trials (t(15)=3.9, p<0.001). Neutral no-go P3 amplitude was not significantly different to no-prime no-go P3 amplitude (m=2.7; std=3.3; t(15)=-1.2, p=0.25).

Further analysis was conducted to explore the possibility that the magnitude of the N2 and P3 effects was affected by participants' scores on the forced-choice tasks. Correlations were calculated between the three no-go difference amplitudes in each time window and performance on the two forced-choice tasks measured by both percentages correct and d'. In addition, absolute values of d prime and the absolute difference from 50% performance were also calculated and correlated with the ERP amplitudes. Differences between each of the three no-go difference waveforms were also calculated as a measure of the amount of priming between conditions. Finally, correlations between no-go N2 onset, and forced-choice performance were explored. None of these correlations were found to be significant, suggesting that the modulation of the ERPs in the participants was not due to residual awareness of the primes. Finally, running two one-way ANOVAs using only the eight participants classed as unaware confirmed that the amplitude and onset modulation of the N2 and the amplitude of the no-go P3 were still present for this group.

Finally, to explore whether early frontal ERP activity was modulated by the nature of the primes two-way repeated measures ANOVA was conducted

with prime type (go, no-go, neutral) and target type (go, no-go) as repeated measures factors and awareness as an independent factor. Since the analysis described above found that incongruent no-go N2 showed an earlier onset than the other two conditions, it is important to consider if this early N2 might also be evident for the congruent go condition. Such a finding would imply that the unconscious primes were directly activating the frontal control processes indexed by the no-go N2. Figure 5.5 shows the raw ERPs at electrode Fz for these six conditions. Visual inspection of the ERPs shows the N2 for congruent and neutral no-go trials peaking around 320ms after stimulus onset. The incongruent no-go N2 is also evident, peaking at around 200ms. Importantly, an identical N2 appears to be present in this time window for congruent go trials (no-go prime mask-effect). ANOVA confirmed that there was a significant main effect of prime ($F(1.1,15.6)=17$, $p<0.001$) from 150 to 200ms after stimulus onset, with go prime trials showing significantly increased negative amplitude in comparison to neutral ($F(1,14)=18.8$, $p<0.001$) and no-go prime trials($F(1,14)=35$, $p<0.001$). There were no significant effects involving awareness or target type in this time window and no target x prime interaction. This finding suggests that the early N2 in response to the prime-mask effect was directly initiated by the unconscious prime, since it is present even when the final target stimulus codes for a go response.

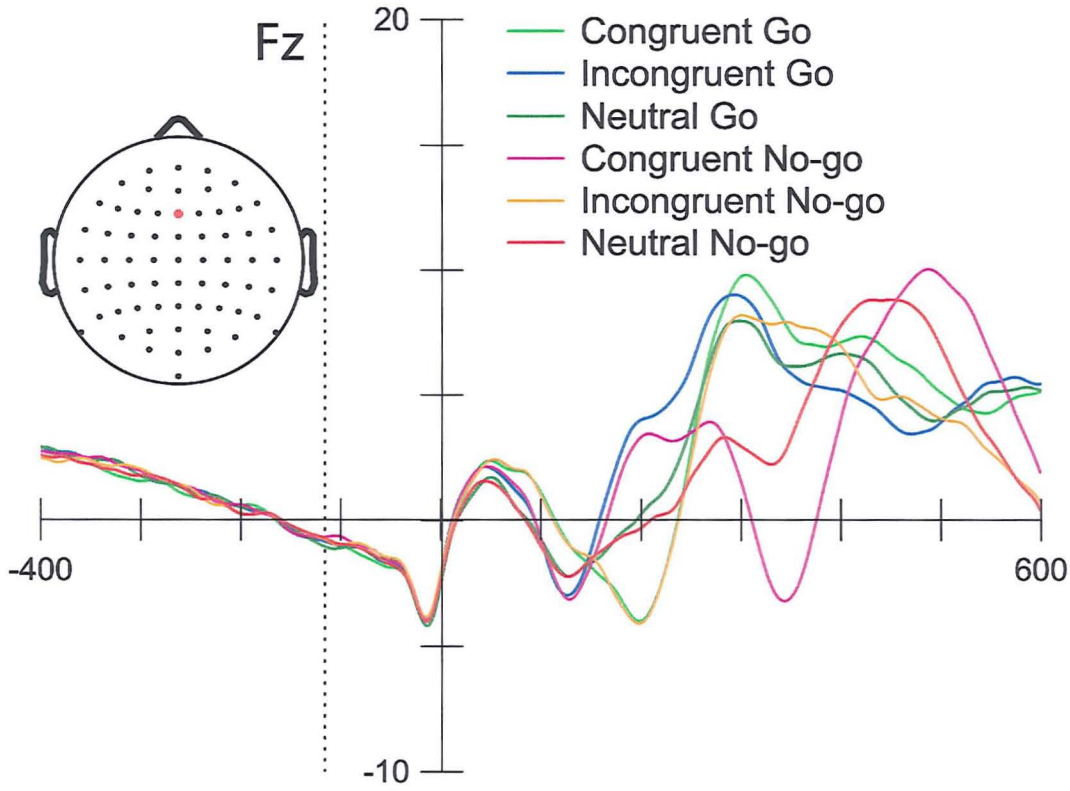

Figure 5.5: Grand average ERP waveforms for six primed conditions at electrode Fz.

N2 and P3 topography was explored by computing scalp maps of the difference waveforms for those conditions which showed a notable N2 or P3. Figure 5.6 shows the scalp maps for the no-go N2 for the neutral prime, no prime and congruent prime conditions. In all three conditions the no-go N2 appears to initially show a frontal maximum, beginning around 260 to 270 ms. This initial frontal maximum then becomes rather more centrally and parietally distributed, which in the neutral and no prime trials ends up as a separate parietal component. The bottom right panel shows the early no-go N2 for incongruent no-go trials. This condition showed a similar frontal component, but not the parietal contribution observed in the other conditions. These apparently separate frontal and parietal contributions to the difference waveforms in this time window will be discussed in more detail in the following sections.

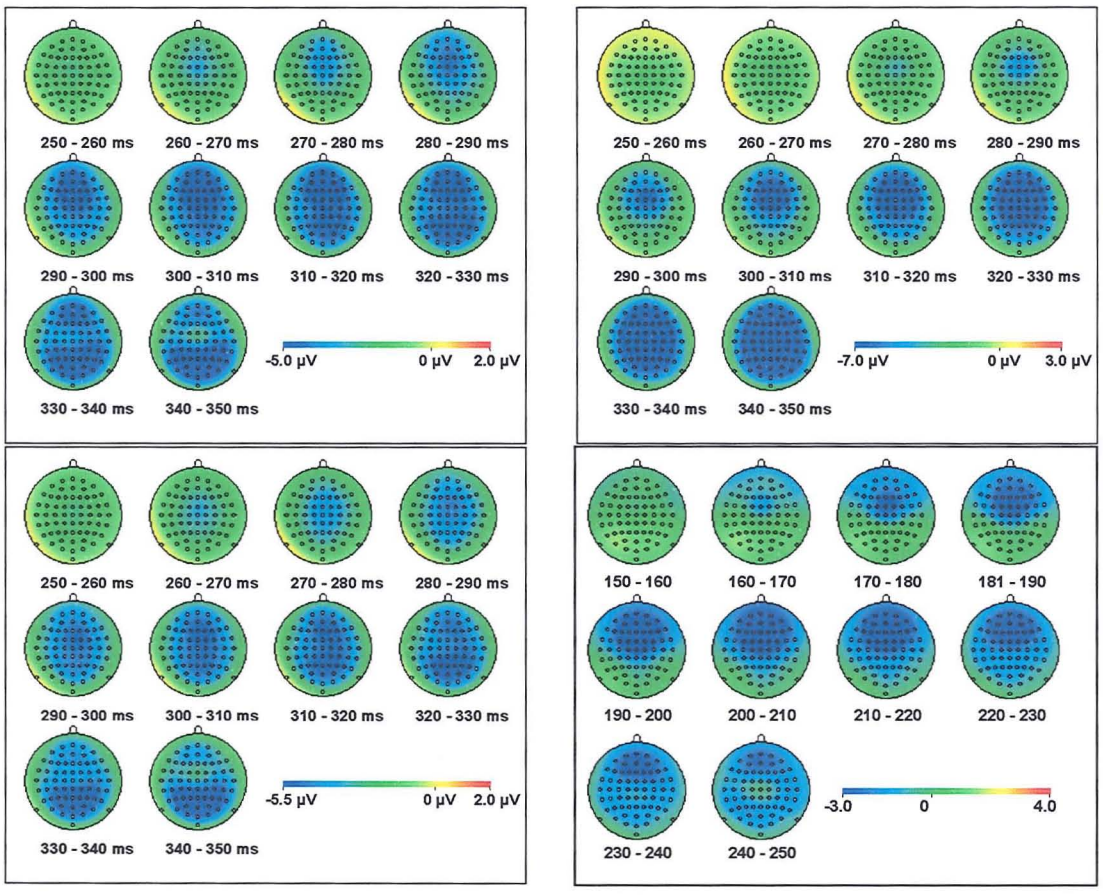

Figure 5.6: Scalp distribution of no-go N2 for no prime (top left), congruent (top right), neutral (bottom left) and incongruent no-go trials (bottom right). Each scalp map represents the average amplitude for the specified 10ms time window.

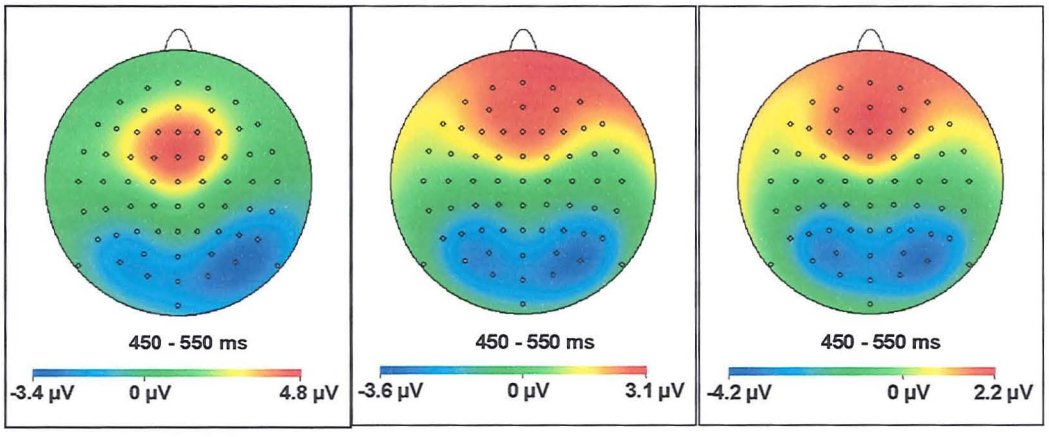

Figure 5.7: Scalp distribution of no-go P3 for congruent (left panel), no prime (central panel) and neutral (right panel) no-go trials.

Figure 5.7 shows the scalp distribution of the no-go P3 which appears to have a frontocentral distribution in all three conditions, but is more focused on Fz FCz and Cz in the congruent no-go condition. A bilateral

parietal/occipital negativity is also present in this time window and will be examined in the later more comprehensive analysis of go/no-go differences.

Early visual ERP effects

To explore whether the observed modulation of the no-go N2 and P3 were mediated by differences in early visual ERP components EEG activity was explored at electrode Oz. Figure 5.8 shows the grand average ERPs at electrode Oz. ANOVA focused on the P1 (-25 to 15ms) found no significant main effect of prime or target and no significant interaction. Similarly, a time window focused on the N1 (25 to 75ms) showed no main effects and no interaction. In a third time window (75ms to 175ms) there was a significant main effect of prime type ($F(2,29.5)=21.5$, $p<0.001$), with go primes showing significantly more positive amplitude than neutral ($F(1,15)=35.2$, $p<0.001$) or no-go primes ($F(1,15)=23.2$, $p<0.001$). Neutral and no-go prime trials did not differ.

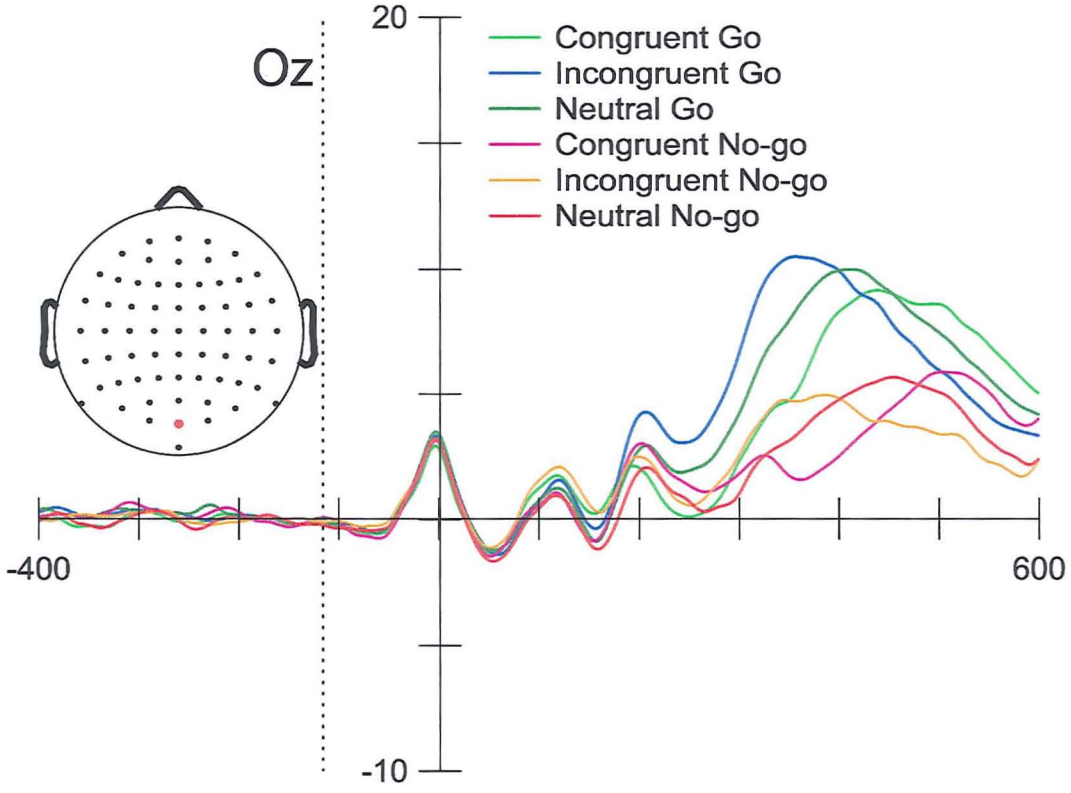

Figure 5.8: Grand average ERPs for six conditions where a prime was presented at electrode Oz.

Visual ERP effects were explored further by computing grand average ERPs dependent on the direction of the prime and target arrows regardless of the experimental condition. Since each participant completed one session where a left arrow signified a go response and another session with the reverse opposite mapping, the grand average presented above (figure 5.8) is collapsed across these two response mappings. Therefore, to explore ERPs for the different visual primes and targets separate grand averages were formed for sessions requiring different response mappings. Analysis at Oz for these two different response mapping revealed essentially the same effects as those observed for all subjects – namely a significant increase in amplitude from 75ms to 175ms after target onset for go trials.

Further analysis explored possible lateralised ERP effects dependent on the type of stimulus presented. O1-O2 and PO7-PO8 difference waveforms were calculated for each different physical prime/target combination, regardless of the response. Figure 5.9 shows these grand average ERP difference waveforms. Visual inspection reveals that the early visual ERP components were slightly right lateralised, with the P1 showing decreased amplitude over electrode O1 in comparison to O2. The N1 component also appears slightly right lateralised showing slightly lower amplitude at electrode PO7 in comparison to PO8. More importantly there appears to be some consistent effects of prime type and target on these difference waveforms, examined in more detail below. ANOVA were conducted in various time windows with prime type (left, right, neutral) and target type (left and right) as repeated measures factors. In addition, response mapping was included as a repeated measures factor to explore whether the differences any differences were related to the meaning of the response rather than simply their physical differences. For example a left arrow might only produce a right hemisphere response when it is designated as the go stimulus. This would reflect an N2pc response to the arrow, which should exhibit a posterior contralateral negativity following a target stimulus but not a non-target distracter (see chapter 2).

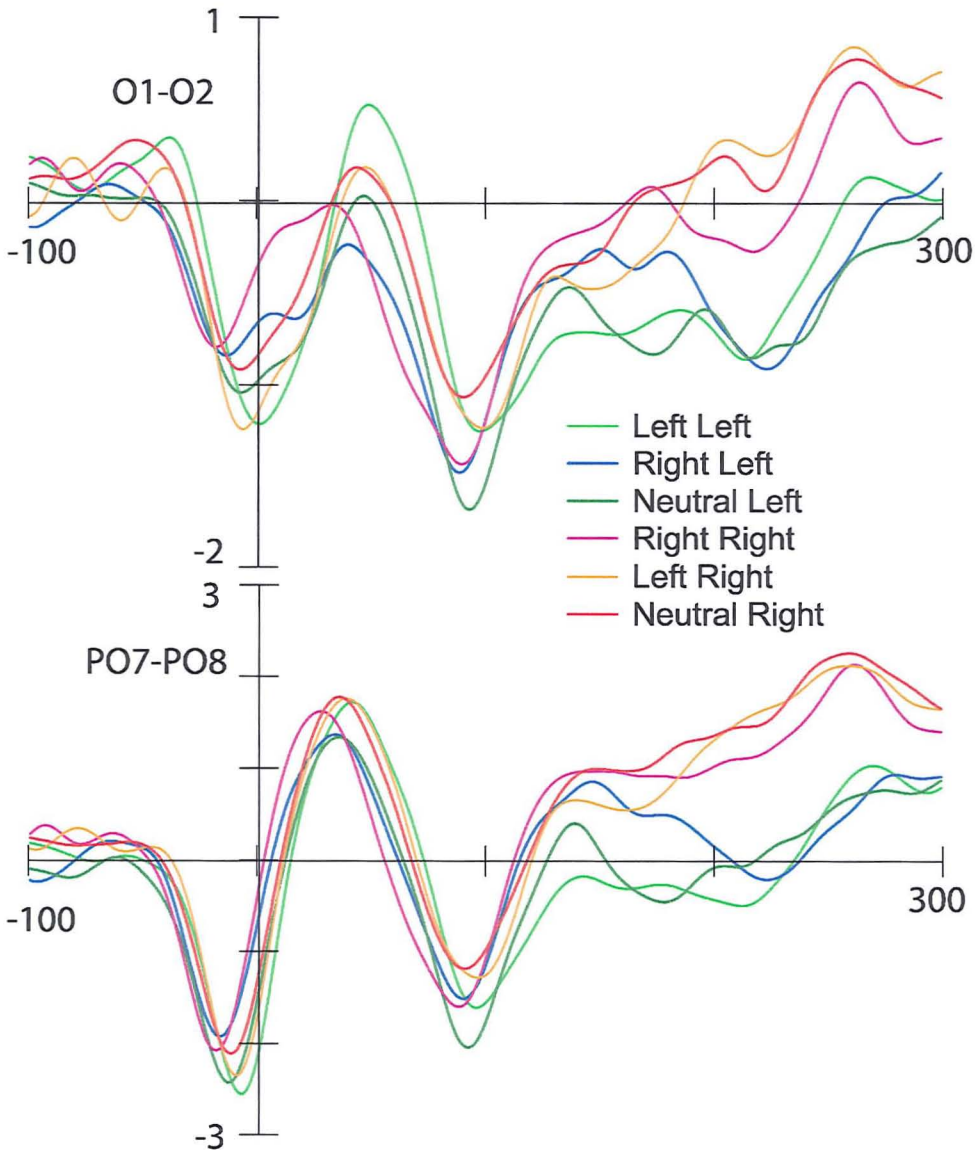

Figure 5.9: Lateralised occipital/parietal effects dependent on the physical stimuli presented in experiment 1.

For the PO7-PO8 difference, ANOVA in the P1 time window (-25 to 15ms) revealed a significant main effect of prime ($F(1.7,25.3)=5.5$, $p<0.05$), but no main effect of target type ($F(1,15)=2$, $p=0.18$) and no significant effects involving response mapping. A significant linear contrast ($F(1,15)=7.3$, $p<0.05$) was evident such that left pointing primes showed greater positivity over the right hemisphere than right pointing arrow primes, with neutral primes in between. Similarly, in the N1 time window (25 to 75ms) there was a significant main effect of prime type ($F(1,15)=3.8$, $p<0.05$) but not target type ($F(1,15)=0.6$, $p=0.44$), and no effects of response mapping. A significant linear contrast ($F(1,15)=6.4$, $p<0.05$) was evident for prime type

with left prime trials showing reduced negativity over the right hemisphere and right prime trials showing the greatest increased N1 amplitude over electrode PO7. Similar results were obtained for the O1-O2 comparison, with the P1 ($F(1.4,20.2)=8.4$, $p<0.01$) and N1 ($F(1.7,25.7)=3.7$, $p<0.05$) showing significant modulation dependent on prime type. From around 150ms after stimulus presentation the difference ERPs begin to separate based on the target, with increased activity contralateral to the direction of the arrows. ANOVA confirmed significant main effects of target for 200 to 300ms post-stimulus for both the O1-O2 difference ($F(1,15)=11.5$, $p<0.01$) and the P07-P08 difference waveform ($F(1,15)=17.6$, $p<0.001$). As in the early time windows, there were no significant effects involving response mapping. These results suggest that the particular physical characteristics of the prime and target stimuli were coded extremely early in the visual system, despite the primes being completely unconscious. These early visual asymmetries were independent of the functional significance of the stimulus and simply reflected the physical characteristics of the stimuli.

Go/no-go Differences

This section provides a more comprehensive analysis of go/no-go differences throughout the epoch and across the scalp. Figure 5.10 shows the raw ERP waveforms for the six conditions where a prime was presented. Visual inspection of the ERPs reveals a clear frontocentral negativity peaking around 350ms after stimulus onset for the congruent no-go condition and for the neutral no-go condition, which likely reflects a no-go N2. An earlier negativity is also evident maximal over frontal and central electrodes peaking around 200ms after stimulus onset in the congruent go condition and the incongruent no-go condition. Interestingly in both these conditions the prime-mask effect codes for a no-go response, and thus this earlier negativity could reflect an early no-go N2 elicited by the unconscious prime-mask effect. There is also frontal positivity in this same time window for the two conditions that contain a go prime-mask effect (incongruent go and congruent no-go), which appear to be more positive than the conditions with a neutral prime. Also evident in the ERP waveforms is a P300 component which is maximal over central/parietal electrodes and is bilaterally distributed. This component

seems larger for the three go conditions and its onset appears to be modulated by prime congruency such that its onset is earliest for incongruent go trials and latest for congruent go trials.

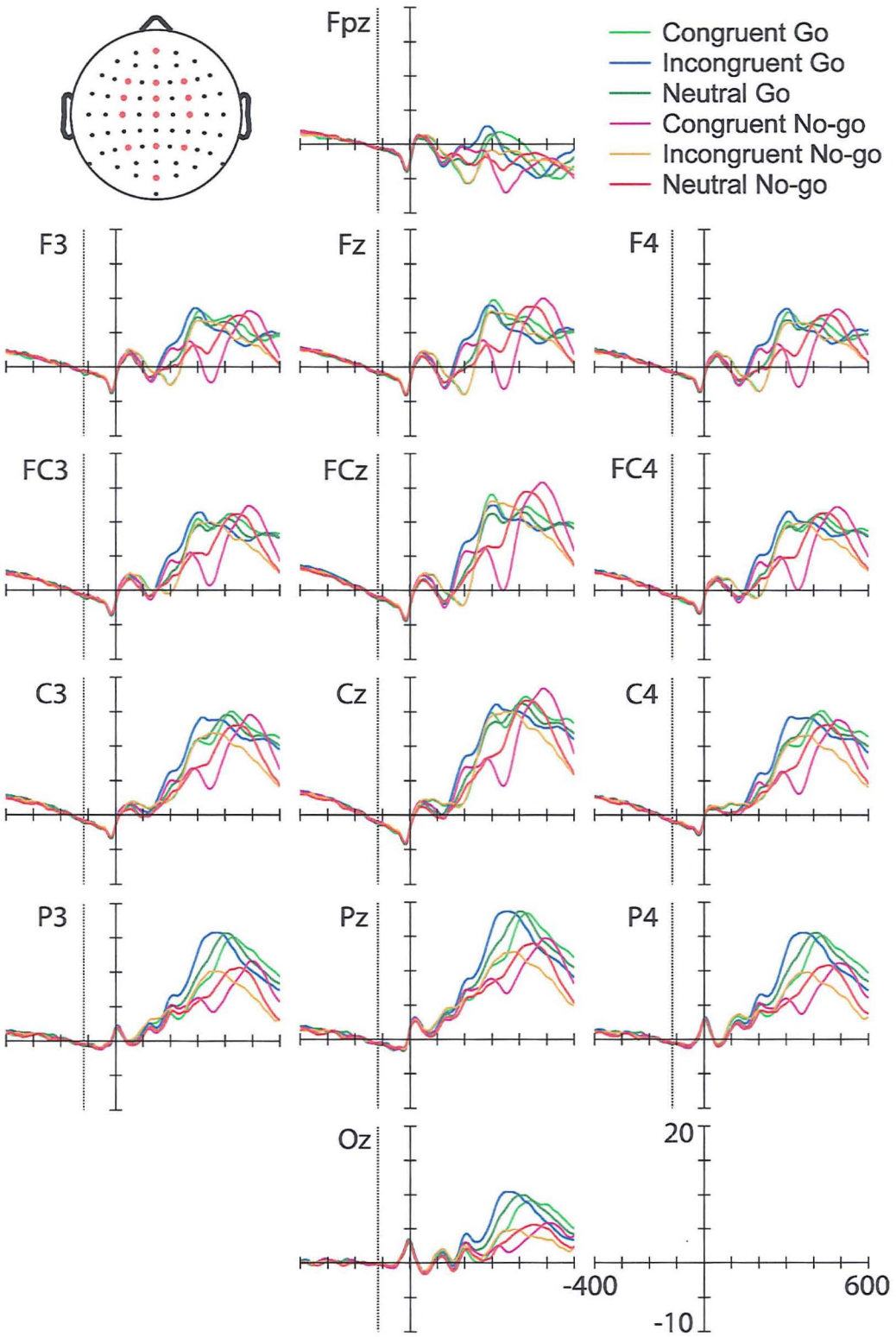

Figure 5.10: Grand average ERPs for six conditions where a prime was presented. Scalp map shows the array of electrodes presented in the figure. Prime onset signified by dotted line.

Statistical analysis of the go/no-go differences was explored further using a six way ANOVA with prime type (go,no-go,neutral), target type (go,no-go), time (85-135, 135-235, 235-385 and 385-585ms), hemisphere (left,right), anterior-posterior (Fp, F, FC, C, P, and O) as within-subjects factors and awareness as a between subjects factor. There was no main effect of awareness nor any significant interaction with awareness and any other variable or combinations of variables. Similarly, there was no main effect of hemisphere and the only interaction to reach significance was a hemisphere x anterior-posterior x time x prime interaction ($F(7.7,108)=2.72$, $p<0.01$). Further exploration revealed that this interaction was caused by larger left sided amplitude for neutral prime conditions in the early time window, which then reversed over frontal electrodes in the second time window before largely disappearing in the final two time windows. Importantly, the four-way interaction prime x target x anterior-posterior x time was highly significant, suggesting that further exploration of the effects of the prime and target stimuli and their interaction with anterior-posterior electrode locations in the four time windows was required.

Four follow up three-way ANOVAs were conducted exploring prime type (go, no-go, neutral), target type (go, no-go) and anterior-posterior electrode location (Fpz, Fz, FCz, Cz, Pz, and Oz) in each of the four time windows. Since awareness and hemisphere appeared to play no role in the relationship between prime and target type, all subjects were analysed in a single group regardless of their awareness of the prime, with analysis conducted on six electrodes along the midline. The time windows were selected to capture the effects of the prime, prime-mask effect and target respectively. The first time window was centred on 201-251ms after prime onset which was equivalent to 85ms to 135ms after target onset. This period was chosen to capture any initial negativity beginning 200ms after prime onset (an N2 to the prime). Since the prime effect would be immediately followed by a prime-mask effect, the second time window (135 to 235ms after target onset) was chosen to reflect the period around any N2 related to the prime-mask effect. Similarly, the third time window (235ms to 385ms post target onset) was chosen to reflect the period around the peak latency

of the N2 related to the target. Finally, the fourth time (385ms to 585ms) window was chosen to capture any no-go P3 activity which is normally seen following the no-go N2.

In the early time window (201ms to 251ms post prime; 85ms to 135ms post target), there was no significant main effect of target ($F(1,15)=1.07$, $p=0.75$) and no target x prime interaction ($F(1.6,23.4)=0.11$, $p=0.7$). There was a main effect of prime ($F(1.9,28.7)=22.12$, $p<0.001$), as well as a prime x anterior-posterior ($F(3.7,55.5)=7.6$, $p<0.001$) and a marginally significant target x anterior-posterior interaction ($F(2.1,32)=3.3$, $p<0.05$), but no three way interaction ($F(1,15)= 0.11$, $p=0.7$).

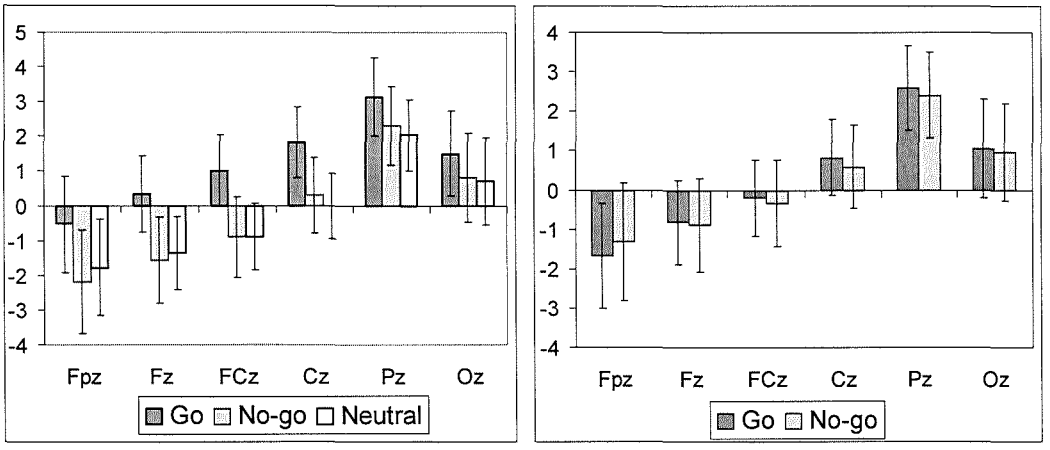

Figure 5.11: Average amplitude for midline electrodes in early time window dependent on prime type (left panel) and target type (right panel). Amplitude in microvolts on y axis and electrodes on x axis.

Figure 5.11 shows a summary of the data for this time window. The left panel shows that the main effect of prime appears to be present across all electrode sites, but maximal at electrodes Fz and FCz, with this decreased difference at posterior sites likely driving the prime x anterior-posterior interaction. Follow up comparisons revealed that there was no significant difference between no-go and neutral prime trials (all $ps>0.1$) at any electrode sites. There were significant differences between go primes and both neutral and no-go primes at all six electrode locations (all p values less than 0.001 uncorrected, with go versus no-go at electrode Pz the only p value not reaching $p<0.001$), with the effect maximal at Fz. The right panel

shows that the go and no-go targets appear to have a similar amplitude at all electrode sites, with only a slight difference evident at electrode Fpz. T-tests confirmed that there was no significant difference between go and no-go targets at any of the six electrode sites (all t values less than 1.3, $p>0.2$). Thus, the significant effects in the early time window are driven by a widespread increase in ERP amplitude in response to go primes in comparison to neutral and no-go primes peaking at electrode Fz.

In the second time window (135 to 235ms after target onset), the initial ANOVA showed a significant main effect of prime ($F(1.2,18.3)=26.37$, $p<0.001$) and significant prime x anterior-posterior ($F(1.9,28.7)=22.12$, $p<0.001$) and target x posterior anterior ($F(2,30.1)=4.35$, $p<0.05$) interactions. There was no main effect of target ($F(1,15)=3.4$, $p=0.08$), no interaction between prime and target ($F(1.35,20.2)=3.78$, $p=0.055$) and no three way interaction between prime, target and anterior-posterior ($F(3.5,51.7)=1.9$, $p=0.13$).

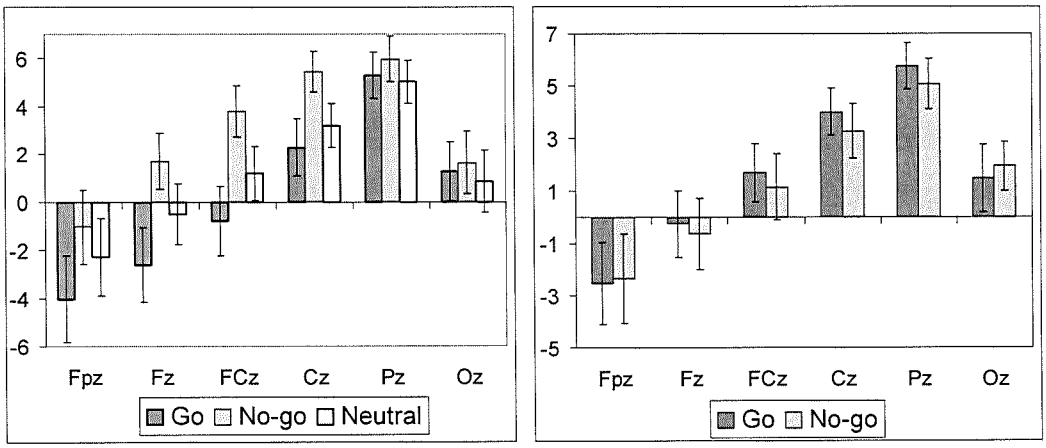

Figure 5.12: Average amplitude for midline electrodes in second time window dependent on prime type (left panel) and target type (right panel).

Figure 5.12 shows the data from the second time window. The left panel shows that trials with a no-go prime appear to be more positive than the neutral trials, with neutral trials in turn more positive than go prime trials. This effect appears to be evident across frontal electrodes and much less so at parietal and occipital sites. T-tests confirmed significant differences (at $p<0.003$ uncorrected, with only two comparisons failing to reach $p<0.001$)

between all prime conditions at Fpz, Fz and FCz. At Cz the difference between no-go prime trials and the other two conditions reached significance (at $p < 0.001$ uncorrected), while the difference between go and neutral trials was not significant ($t(15)=-1.9$, $p=0.08$). At electrode Pz and Oz only the comparisons between go and no-go primes remained significant (at $p<0.001$). The difference between the prime conditions peaked at electrode Fz and FCz.

The right panel of figure 5.12 shows that go target trials appear to be slightly more positive than no-go target trials, especially at central and parietal electrodes. T-tests confirmed that there were marginally significant differences at electrodes Cz, Pz and Oz ($p<0.025$, uncorrected), but not at anterior electrodes. Summarising the results from the second time window, it appears that once again the major contribution to the differences was a frontal difference dependent on prime. This effect was reversed from the effect in the first time window such that go primes trials appeared more negative than neutral and no-go prime trials. This effect likely reflects modulation of the frontal no-go negativity associated with the prime-mask effect. A greater negativity was evident for go prime (no-go prime-mask effect) trials, which likely reflects the early N2 component observed for these trials in the earlier onset analysis. A significant difference was also observed between no-go and neutral prime trials in this time window, with no-go prime trials showing a greater positivity. Finally, a modest effect of target type also appeared in this second time window, which was only present at posterior electrode sites.

In the third time window (235ms to 385ms post target onset) there was a main effect of target ($F(1.2,18.3)=26.37$, $p<0.001$), a significant main effect of prime ($F(1.2,18.3)=26.37$, $p<0.001$) as well as a target x prime interaction ($F(1.2,18.3)=26.37$, $p<0.001$). In addition there was a significant interaction between prime and anterior-posterior ($F(1.2,18.3)=26.37$, $p<0.001$), with the target x prime x anterior-posterior interaction also showing statistical significance ($F(1.2,18.3)=26.37$, $p<0.001$). Figure 5.13 shows the average amplitude for the three prime conditions separately. The figure shows that

while there is no difference between go and no-go target trials following a go prime, there appear to be widespread differences following a neutral and no-go prime. This was confirmed by paired t-tests which showed significant differences (at $p<0.001$) between go and no-go trials at all electrode locations following no-go primes and neutral primes (with the exception of Fpz which was at $p<0.002$ for the comparison between neutral and no-go primes). In contrast the only significant difference following go primes was at electrode Fpz ($t(15)=3.73$, $p<0.002$).

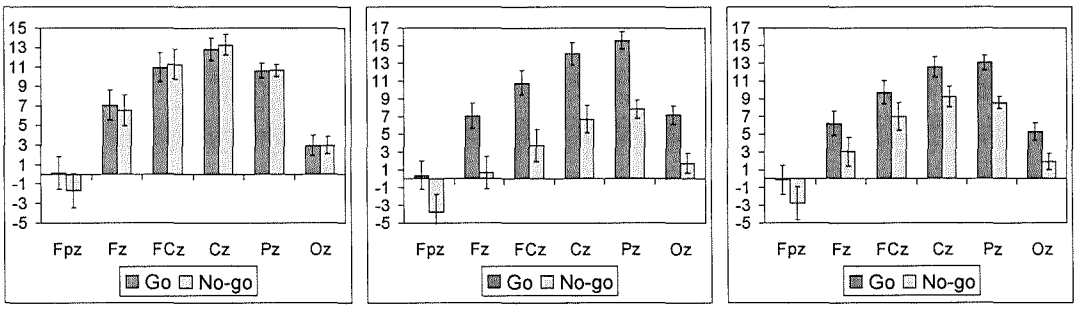

Figure 5.13: Average amplitude for midline electrodes in third time window dependent on target type for go primes (left panel), no-go primes (middle panel) and neutral primes (right panel).

In addition to this differential effect of target type for the three prime type conditions (the target x prime interaction), the relationship between prime and target also appeared to be dependent on the anterior-posterior electrode location (the prime x target x anterior-posterior interaction). Figure 5.14 shows the effect of prime type for go and no-go target trials separately on the six midline electrodes. It is evident from this graph that while a frontal modulation is present for no-go target trials (right panel) a parietal/occipital modulation is driving the differences on go target trials (left panel). This relationship was confirmed with paired sampled t-tests which showed that for go trials the only significant differences (at $p<0.001$ uncorrected) between prime types were at electrode Pz and Oz, with all three conditions differing from one another at these sites. For no-go trials all three conditions significantly differed (at $p<0.001$) from one another at electrodes Fz, FCz and Cz only with the differences peaking at FCz. Go and no-go prime trials differed at electrodes Oz and Pz and Fpz (at $p<0.002$).

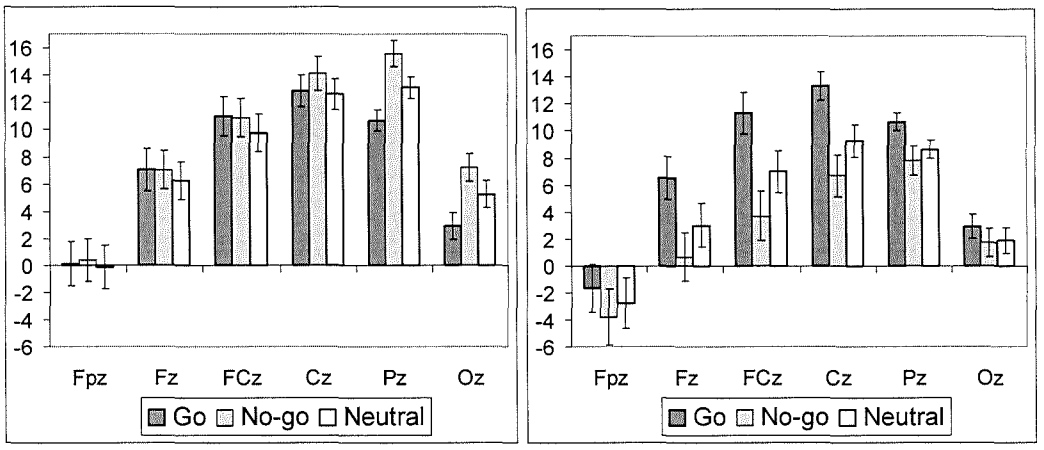

Figure 5.14: Average amplitude for midline electrodes in third time window dependent on prime type for go targets (left panel) and no-go targets (right panel).

Summarising the data from the third time window (235ms to 385ms post target onset), there appeared to be two main effects driving the differences in this period. The amplitude at frontal electrodes appeared to vary dependent on prime type for no-go target trials only, while a parietal difference was evident only for go target trials. Re-examining the ERPs presented in figure 5.10 it seems clear that the differences at frontal electrodes reflect modulation of the frontal no-go N2 which peaks in this time window. As described in the previous section, this component was found to be greatest for no-go trials preceded by a no-go prime (congruent no-go), and smallest for no-go trials following a go prime. The parietal modulation for go trials appears to reflect modulation of a parietal P300 component which is greater for go compared to no-go trials, with its onset apparently modulated by prime type. The finding that amplitudes significantly differed on the upward slope of this component indeed confirms that this component varied as a function of prime type for go trials.

In the final time window (385 to 585ms) there was no main effect of target ($F(1,15)=3.75$, $p=0.07$) and no main effect of prime ($F(1.7,26.6)=3.4$, $p=0.054$). There was however a significant target x prime interaction and a significant target x prime x anterior-posterior interaction ($F(3.7,54.8)=17$, $p<0.001$). Figure 5.15 shows the average amplitude for the fourth time window dependent on prime type, for go and no-go target trials. While for go target trials go primes show more positive amplitude in comparison to no-go

primes this effect is reversed for no-go target trials such that no-go primes appear more positive. In addition, while these differences for go target trials appear greatest at posterior sites, the no-go target trials differ at more anterior electrode locations. T-tests confirmed that for go target trials, no-go primes were significantly more negative than neutral and go-primes at electrode Oz ($P<0.001$) and marginally significant at Pz ($p<0.006$), with no other comparisons approaching significance. For no-go target trials no-go prime trials were significantly (at $p<0.001$) more positive than neutral and go prime trials at electrodes FCz, Cz and Pz.

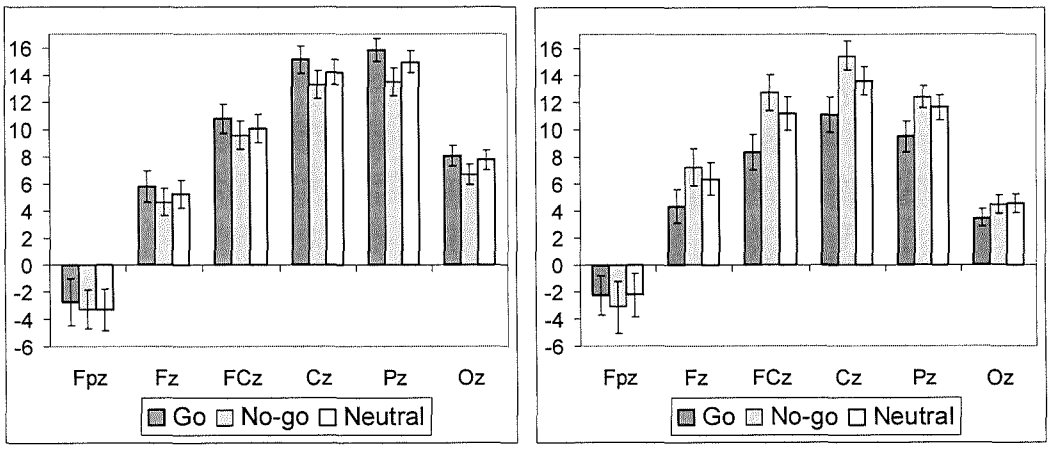

Figure 5.15: Average amplitude for midline electrodes in fourth time window dependent on prime type for go targets (left panel) and no-go targets (right panel).

The parietal effects in this final time window likely once again reflect the modulation of the parietal P300 for go trials, with this period encompassing the falling edge of this component and thus showing the reverse direction to the effect in the previous time window. The more anterior difference on no-go trials most likely reflects modulation of the frontocentral no-go P3 that, as explored above, was modulated by prime type on no-go trials.

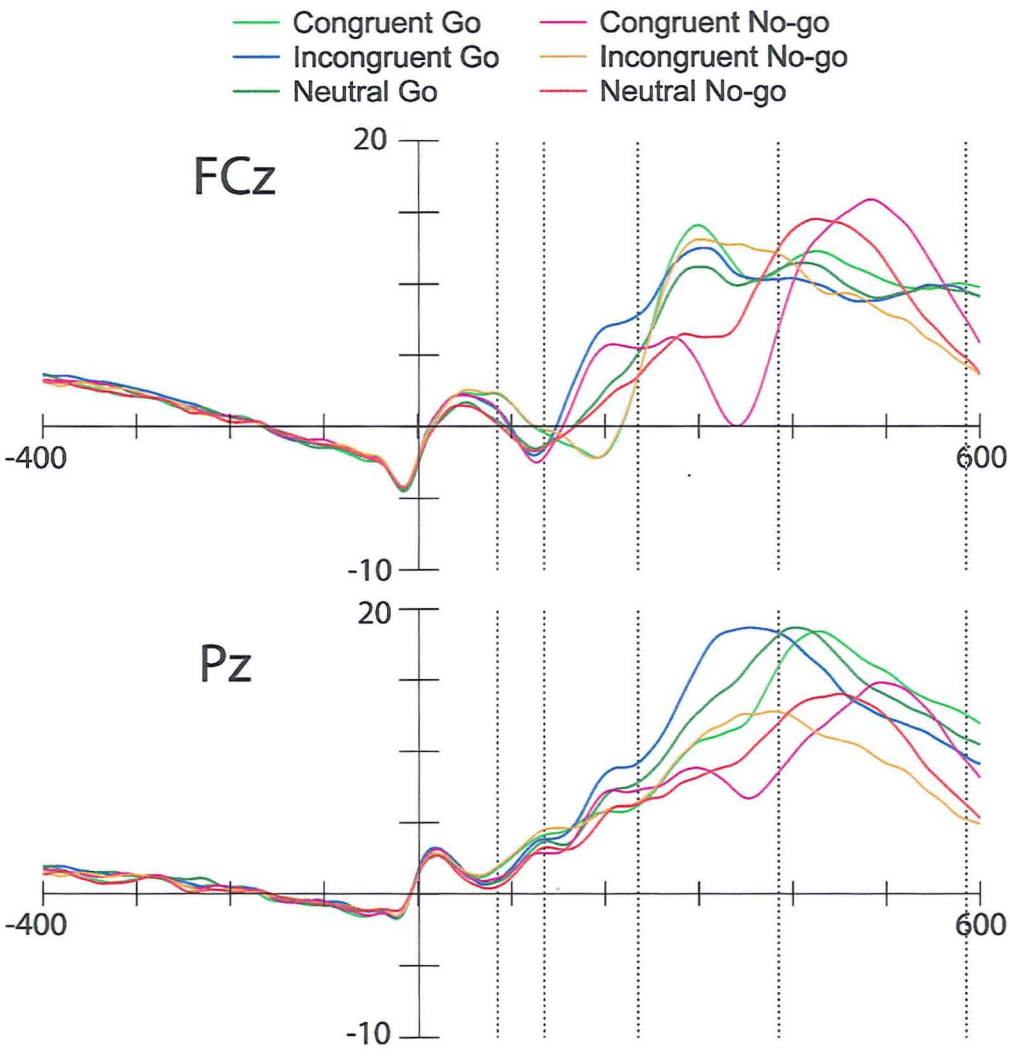

Figure 5.16: Grand average ERPs at electrode FCz and Pz.

Figure 5.16 shows the grand average ERPs for electrodes FCz and Pz with the four time windows separated by the dashed lines. These two waveforms provide a summary of all the major finding in the comprehensive analysis of the entire epoch described above. The effects in the first time window were confined to frontal electrodes where trials with a go prime (congruent go and incongruent no-go) showed an increased positivity. In the second time window these effects reversed such that no-go primes now showed more positive amplitude, with go primes showing increased negative amplitude. Importantly, in both these time windows the waveforms at frontal electrodes were uniquely determined by the unconscious prime. This second time window also began to include differences at parietal electrodes associated with increased P300 for go trials in comparison to no-go trials.

The third time window centred on the no-go N2 at frontal electrodes and the rising bank of the P300 at parietal electrodes. At frontal electrodes the no-go N2 was found to be significantly more negative for congruent no-go trials than neutral no-go trials, while the N2 for the incongruent no-go condition appeared to be altogether absent in this time window. At parietal electrodes the modulation of the P300 component for go target trials was highlighted by significantly increased amplitude for incongruent go trials. This dissociation between no-go effects at frontal electrodes and go effects at Pz is important with respect to the N2 topography shown in the previous section. In the topographic maps presented in this analysis (Figure 5.6), two contributions to the go/no-go differences were identified. Thus while the parietal contribution to the difference topographies reflects P300 differences between go and no-go trials, the frontal effect reflects the no-go N2. Finally, the fourth time window was focused on the period around the no-go P3 on frontal electrodes, and the falling bank of the P300 at parietal electrodes. The frontal no-go P3 was found to be largest on congruent no-go trials, while amplitudes at Pz once again separated based on the modulation of the P300.

LRP and go/no-go differences

To explore the possibility that the effects observed at central electrodes were projections of movement related activity at lateral electrodes, grand average ERPs were computed separately for left and right hand responses. Since Praamstra and Seiss (2005) showed that a pseudo N2 was produced in a choice reaction time version of this experiment that caused by averaging together right and left responses, it is important to consider the contribution of these lateralised motor effects in the current experiment. Figures 5.17 shows grand average ERPs for left and right hand trials separately at electrodes C3, C4 and Cz for congruent and incongruent no-go conditions. Examination of this figure reveals that there are lateralised differences present at these electrodes. Most notably, from around 200ms after stimulus onset the grey and black lines showing more negative amplitudes over FC3 and the converse at FC4. These lateralised motor effects reflect the partial

onset of an LRP for congruent no-go conditions from around 180ms after stimulus onset highlighted in the earlier LRP analysis (see figure 5.2). Despite, this slight lateralisation of activity at central electrodes the main effect of the N2 is apparent at both left and right electrodes for both left and right hand responses and therefore cannot be produced by averaging together left and right responses as was the case in Praamstra and Seiss (2005). In addition, an earlier lateralised effect is also evident overlapping with the prime-related effects, but once again, since the difference between the prime conditions is different at both electrode locations and for both conditions, this effect is not caused by the motor related asymmetries.

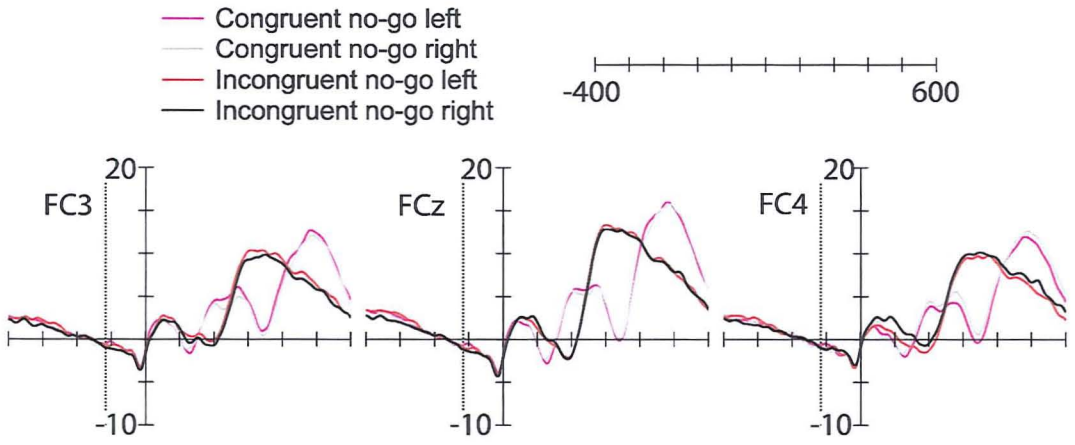

Figure 5.17: Grand average ERPs for congruent no-go and incongruent no-go trials separated by response hand.

Finally figure 5.18 shows the relationship between the no-go N2 at electrode Fz alongside the LRP for all conditions (with neutral go omitted from the N2 waveforms as they represent difference waveforms from this condition). The dotted line represents the average onset of the N2 for the trials with a no-go prime-mask effect (left line), and the trials with a no-go target (right line). Following these lines down to the LRP reveals a strong relationship between the onset of the negative component and the motor related activity. Specifically the onset of the early no-go N2 for those trials with a no-go prime-mask effect appears to coincide very closely with the time at which the early LRP activation begins to return to baseline. Similarly, for congruent no-go trials, the N2 appears to onset around the same time that the LRP in this condition returns begins to return to baseline.

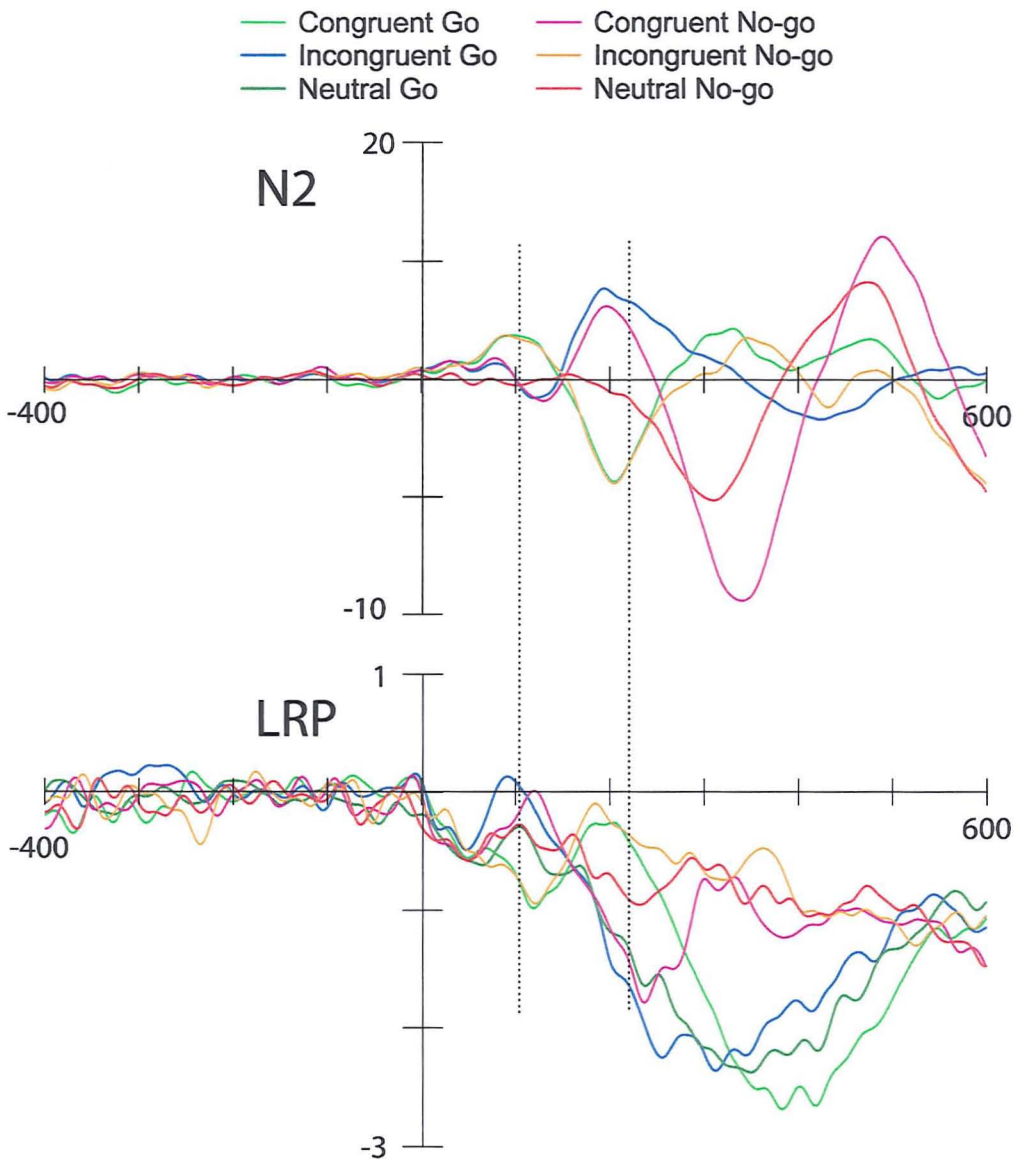

Figure 5.18: N2 difference waveforms viewed alongside the lateralised readiness potential.

Discussion

This investigation aimed to determine whether endogenous motor inhibition can be initiated unconsciously. While Eimer and Schlaghecken (1998) have shown that exogenous inhibition may be elicited in response to unconscious primes, this has been shown not to be modulated by frontal control mechanisms (Praamstra & Seiss, 2005). Leuthold and Kopp (Leuthold & Kopp, 1998) did show N2 modulation elicited by subliminal primes, but this component exhibited a parietal rather than a frontal topography. This experiment, shows for the first time that the frontal No-Go N2 can be modulated by an unconscious prime. This is in contrast to Eimer and

Schlaghecken's (2003) proposal that this mechanism can only be engaged by a conscious stimulus.

The current experiment varied Eimer and Schlaghecken's (1998) masked priming paradigm to explore unconscious priming of go/no-go differences. Participants were required to press a button as quickly as possible following a go stimulus, and to withhold their response following a no-go stimulus. In order to build up a readiness to respond participants were required to respond to go targets within 500ms of their onset. Reaction times on go trials were significantly affected by the presence of unconscious masked primes, such that participants responded more quickly when a go target was preceded by a no-go prime (incongruent go trial). Similarly, error rates on both go (misses) and no-go (false alarms) were greatest for congruent trials. These behavioural effects follow the negative compatibility effects (NCE) described by Eimer and Schlaghecken (1998).

These findings were evident despite the fact that participants were below the objective threshold for awareness. Performance in the prime identification and prime detection tasks did not significantly differ from chance. However, performance on the long duration primes also did not differ from chance despite clearly being above the subjective threshold for awareness. Further analysis of these trials showed that participants either performed significantly above or below chance. Since in the current experiment, the presentation of the mask reverses the effect of the prime participants effectively perceive both left and right pointing arrows in these trials. In fact, a number of participants reported that they did see the arrows quickly reversing in this task. Clearly, this finding is rather problematic for the use of the prime identification task as a measure of awareness of the primes, since participants may be able to reliably identify the direction but may be confused by the fact that they see one direction followed by the other. This problem is overcome in the current experiment by also asking participants to perform a prime detection task. Since in this task participants are required to detect the presence or the absence of any prime, their performance should not be affected by any reversal effects caused by the mask. Therefore, since

performance on this task was not significantly different from chance, it is unlikely that participants were aware of the briefly presented primes. This assumption is also reinforced by the finding that priming was not correlated with prime detection performance, suggesting awareness of the primes was independent of priming effects. However, it is important to note that the finding of significantly below chance performance on the long duration primes for the identification task has implications for other experiments using these stimuli. Firstly, since most of those experiments (including Eimer & Schlaghecken, 1998) use a similar prime identification task, it is possible that performance was at chance despite some awareness of the masked primes being present. Furthermore, a central claim of the exogenous inhibition account of the NCE proposed by Eimer and Schlaghecken (1998) is that negative compatibility only occurs below the threshold of awareness. The finding that negative compatibility occurred for long duration primes (which were clearly visible) in the prime identification task in the current experiment, suggests rather that it is the combination of the prime and the mask that reverses the NCE and that this reversal is independent of awareness.

The LRP also showed a similar pattern to that observed by Eimer and Schlaghecken (1998). ANOVA confirmed that an initial separation was evident such that incongruent go trials showed least LRP activation and congruent go trials showed greatest LRP activation, reflecting a motor response to the prime. These effects quickly reversed, reflecting the prime-mask effect and leading to an earlier onset of LRP for incongruent go trials, and a later onset for congruent go trials. Further analysis confirmed that these early prime-related effects were also present for no-go trials, confirming that the LRP was directly initiated by the unconscious primes. This finding supports previous reports of direct unconscious priming of a motor response (Dehaene et al. 1998; Leuthold & Kopp, 1998; Eimer & Schlaghecken, 1998).

Initial analysis of go/no-go differences focused on the specific hypotheses outlined in the introduction, namely that the subliminal primes will modulate the no-go N2 and P3 on no-go target trials. This was explored both in terms

of the onset of the N2 component, as well as the ERP amplitude in the no-go N2 and no-go P3 time window. This analysis supported the hypothesis that the unconscious primes were able to modulate frontal inhibition/control mechanisms. N2 onset was significantly earlier for incongruent no-go trials in comparison to all other conditions. Importantly, in this condition the no-go prime-mask effect coded for a no-go response, so this shift in N2 latency likely reflects a no-go N2 to the prime-mask interaction. This is supported by the fact that the latency shift is very similar to the difference in time between the prime onset and the mask onset (100ms). Importantly, the topographic distribution showed that the no-go N2 and P3 components showed a frontocentral maximum, although this was later replaced by a parietal maximum.

As with the LRP, this early N2 was observed in response to the prime and was independent of the target stimulus, suggesting that the primes were able to directly initiate this component. This conclusion was supported by the more comprehensive analysis of go/no-go differences, which also revealed early effects over frontocentral electrodes uniquely dependent on the subliminal primes. Initial activity appeared to separate based on the prime information, with neutral and no-go primes showing an increased negativity in comparison to go primes. This effect then reversed with go prime trials (no-go prime-mask effect trials) showing increased negativity and no-go prime (go prime-mask effect trials) showing increased positivity in comparison to neutral primes. Importantly, this modulation was maximally observed over frontocentral electrodes, suggesting that it reflected modulation of the no-go N2. This early N2 occurred even on congruent go trials despite the fact that ultimately a response was required to the target stimulus. This N2 (in response to the no-go prime-maskeffect) suggests that the response was successfully inhibited at this point, and later reactivated by the target stimulus. This interpretation is supported by examining the LRP and N2 for these trials, where the LRP appears to onset around the time that the N2 component begins to return to baseline. The fact that the ERP waveforms were determined entirely by the prime type in this time window suggests that the primes were able to directly initiate frontal inhibition/control

mechanisms. However, it is important to note that the functional significance of the no-go N2 is still under debate. For example it seems likely that at least part of this component reflects activity related to conflict monitoring and not an active process of inhibiting the response (see chapter 2). While the interaction between the N2 and the LRP points to an active role of this modulation it is possible that this component simply reflects detection of a conflict and not its resolution.

In addition to an increased negativity observed for the no-go prime-mask effect in this time window, an opposite deflection is present for the go prime-mask effect. A possible explanation for this is that since the neutral prime contains one feature that is relevant to a go response and another that is relevant to the no-go response it itself produces a moderate amount of conflict. This would suggest that as the degree of conflict induced by the mask increases, the modulation of this frontal component increases. Similarly, the neutral prime may partially co-activate go and no-go responses and would therefore be associated with a greater degree of inhibition than go primes (or prime-mask effects) and less than no-go prime/prime-mask effects.

In the N2 time window there was a significant modulation of no-go N2 amplitude at frontal electrodes with congruent no-go trials showing greater N2 amplitude. Importantly, a functionally distinct modulation was evident at parietal electrodes, where go target trials varied in amplitude. A significant prime by target by anterior-posterior interaction showed that the frontal modulation was exclusive to no-go target trials, while the go target trials differed only at posterior sites. Furthermore, both the frontal no-go N2 and parietal component were more positive for go target trials than no-go target trials. Therefore, since the N2 difference waveforms were computed in reference to the neutral go trials, the parietal part of the no-go N2 topographic maps in this experiment likely reflects a P300 effect and not a parietal N2. This assumption is supported by evidence that a parietal P300 effect is often observed in go/no-go tasks which partially temporally overlaps with the no-go N2 (Nieuwenhuis et al., 2003).

In the final time window, the major effect to appear was the modulation of the frontal no-go P3, also referred to as the no-go P3 anteriorization due to the shift in topography from a parietal to a frontal P3 for no-go trials. This component was found to vary as a function of the subliminal primes such that it showed greatest amplitude for congruent no-go trials and smallest amplitude for incongruent trials. Once again, this is consistent with the notion that the prime was able to initiate unconscious frontal control/inhibition processes, since this component was reduced when these processes had presumably already occurred.

Analysis of visual ERPs revealed no significant early modulation of visual P1 and N1 components. While a later separation was evident at electrode Oz this appears later than the earliest prime-related effects at frontal electrodes. This once again suggests that the unconscious primes were able to directly initiate the activity at frontal electrodes, and not through perceptual priming of the target stimulus. However, lateralised occipital and parietal effects were evident in response to the physical characteristics of the prime and the mask. Jongen, Smulders and Van der Hinden (2007) recently showed similar visual asymmetries using the same arrow stimuli as those utilised in the current experiment. As in the current task they found that these visual asymmetries were independent of the functional significance of the arrow, and simply reflected the coding of the visual features of the arrows. Despite the arrows being presented at a central location, the more salient feature of the arrow (the point) are appear asymmetrically in the direction in which the arrow points. In the current experiment this would reflect (unconscious) visual detection of lateralised visual features relevant for task processing. Since these visual asymmetries were independent of the functional significance of the arrows, as indexed by the absence of any interactions with response mapping, they do not reflect an N2pc compnent, which should show greater response to go than no-go stimuli.

The finding of early visual effects related to the physical characteristics of the prime does not compromise the conclusion that the frontal effects are

initiated by the prime, as they occur well in advance of the time at which this lateralised activity becomes dependent on the target stimulus. In addition, it seems necessary that some form of visual detection of the stimulus features must be required for a response to begin to be processed, even if this is unconscious. The possible mechanisms of the modulation observed in this experiment, and masked priming effects in general will be discussed in more detail in chapter 10.

The major limitation of this experiment is that it utilised a paradigm that produces a negative compatibility effect, thus complicating the results somewhat. In particular, the greatest prime-related effects appeared in response to the prime-mask effect. While it is likely that this effect is produced in this experiment by a physical interaction between the prime and the mask, its exact mechanism remains the subject of debate. If some inhibition based mechanism is involved in producing the NCE this might be responsible for the N2 modulation in response to the prime-mask effect. However, this seems unlikely in light of the fact that Praamstra and Seiss (2005) failed to find N2 modulation in a similar task. Nonetheless, it is important to show that a similar modulation of no-go N2 and P3 components is evident when a prime-mask combination is used that produces a positive compatibility effect.

Conclusions

This experiment aimed to show that frontal inhibition/control related ERP components could be modulated by an unconscious prime. In support of this hypothesis the frontal no-go N2 and P3 were found to vary as a function of the unconscious information. The finding that early ERP effects were uniquely determined by the nature of the prime suggests that as with motor responses, motor inhibition can be directly initiated by an unconscious stimulus. However, the choice of prime and mask in the current experiment and the reversal of the priming effects that they produced (the NCE) make these conclusions difficult to generalise to unconscious priming producing more conventional priming effects.

Chapter 6

Experiment 2 – Unconscious modulation of no-go N2 and P3 amplitude in a metacontrast masking paradigm.

Introduction

This experiment aimed to replicate the effects of experiment 1 in a paradigm that produced positive compatibility between the mask and the prime. The major complication in interpretation in experiment 1 was the reversal of the initial prime effects generating the NCE. In order to explore if the modulation of the no-go N2 and P3 components were reliable and not simply an artefact of this rather unusual effect of inverse priming, it was important to replicate the effect using a paradigm that produced a positive compatibility effect. A metacontrast masking paradigm was employed similar to that used by Neumann and Klotz (1994). They presented participants with an array of two different shapes – one square and one diamond. Participants were required to press a left button for one array (for example left diamond right square) and press a right button for the opposite array. The subliminal primes consisted of smaller versions of these shapes that fit exactly in to the internal contours of the target/mask. Neumann and Klotz (1994) found that the subliminal primes were successful in priming motor responses, such that reaction times were significantly reduced following a congruent prime. These stimuli were selected for use in the current experiment because they are known to produce a positive priming effect in the absence of awareness.

Hypotheses

Reaction times and error rates should follow a positive compatibility effect in this experiment, with fastest reaction times for congruent go trials and slowest reaction times for incongruent go trials. Similarly, accuracy should be greatest for congruent trials and lowest for incongruent trials. If the subliminal primes are able to influence frontal inhibition/control processes in

this task they should also produce positive modulation of the No-go N2 and P3. N2 and P3 amplitude should be greatest for incongruent no-go trials and smallest for congruent no-go trials. In addition, early frontal differences dependent on prime type should be evident if the unconscious primes can directly initiate inhibition of the response.

Method

Participants

Twenty first-year undergraduate psychology students (three male and 17 female) participated in exchange for course credits. All participants were right handed and had normal or corrected to normal vision. The mean age of participants was 20 years and two months, with a range of 18 to 29 years.

Experimental Procedure

All participants completed a single experimental session lasting approximately two hours. The participants completed 14 blocks of the go/no-go task followed by four blocks of the prime identification task. The go/no-go task required participants to respond as quickly as possible to one stimulus configuration, and refrain from responding to the other. Each go/no-go block contained 96 trials presented in a random order. The 14 experimental blocks were preceded by two practice blocks of 48 trials. On each trial two shapes appeared on the screen. One of the shapes was a square and one of the shapes was a diamond (see figure 6.1). The two shapes appeared randomly above or below fixation. This was in accordance with the procedure employed by Neumann and Klotz (1994). The participants responded identically regardless of whether the stimuli appeared above or below the centre of the screen. The centre of the stimulus was 4.5cm above or below the centre of the screen, with both stimuli either above or below fixation on each trial. The square shape measured 4.7cm across, with the diamond shape being the same shape but rotated by 90 degrees. The centre of each stimulus appeared 3.6cm to the left or right of fixation.

Participants were allocated to either a left go condition, which required a go response when the diamond was on the left, or a right go condition, where a right diamond signalled a go trial. Figure 6.1 shows the stimuli for a congruent, incongruent and neutral trial. Participants in the left go condition were told to respond only when the diamond was presented on the left side of the screen and to refrain from responding when the diamond was presented on the right. For these participants the stimuli in figure 6.1 would represent congruent go, incongruent go and neutral go, respectively. Conversely, participants in the right go condition were told to respond only when the diamond was presented on the right side of the screen and to refrain from responding when the diamond was presented on the left, thus the stimuli in figure 6.1 would represent congruent no-go, incongruent no-go and neutral no-go, respectively.

The response hand was varied from one block the next, with the initial hand counterbalanced across participants. The participants were informed that that they had a time limit of 500 milliseconds (ms) to respond to the go stimuli and that they should react as quickly as possible without sacrificing accuracy. Participants were given visual feedback immediately after the 500ms response window for correct responses and non-responses as well as false alarms and incorrect non-responses. Unbeknownst to the participants masked primes were presented prior to the target stimulus. The prime consisted of a pair of shapes presented at the same location as the target shapes. The primes fit exactly into the internal contours of the target and measured 3cm by 3cm. The configuration of these shapes was congruent with, incongruent with, or neutral to the target stimulus. On congruent trials the prime contained a square and a diamond in the same configuration as the target. Incongruent primes had an opposite configuration to the target, and neutral primes contained two squares. There were an equal number of congruent, incongruent and neutral trials in each block.

The primes were masked by the target stimulus. The primes fit exactly into the internal contours of both target shapes for optimized metacontrast

masking. Each trial began with a central fixation for 700 ms then the primes were presented for 16ms followed by a blank screen for 49ms and then the mask/target for 100ms. Participants had 500ms to respond, after which visual feedback was presented for 500ms. Finally, a blink pause was presented where "blink pause" appeared in the centre of the screen for 1200ms and participants were informed to use this time to blink if they needed. Participants were also informed not to blink during the trial, and to keep their eyes fixated on the centre of the screen.

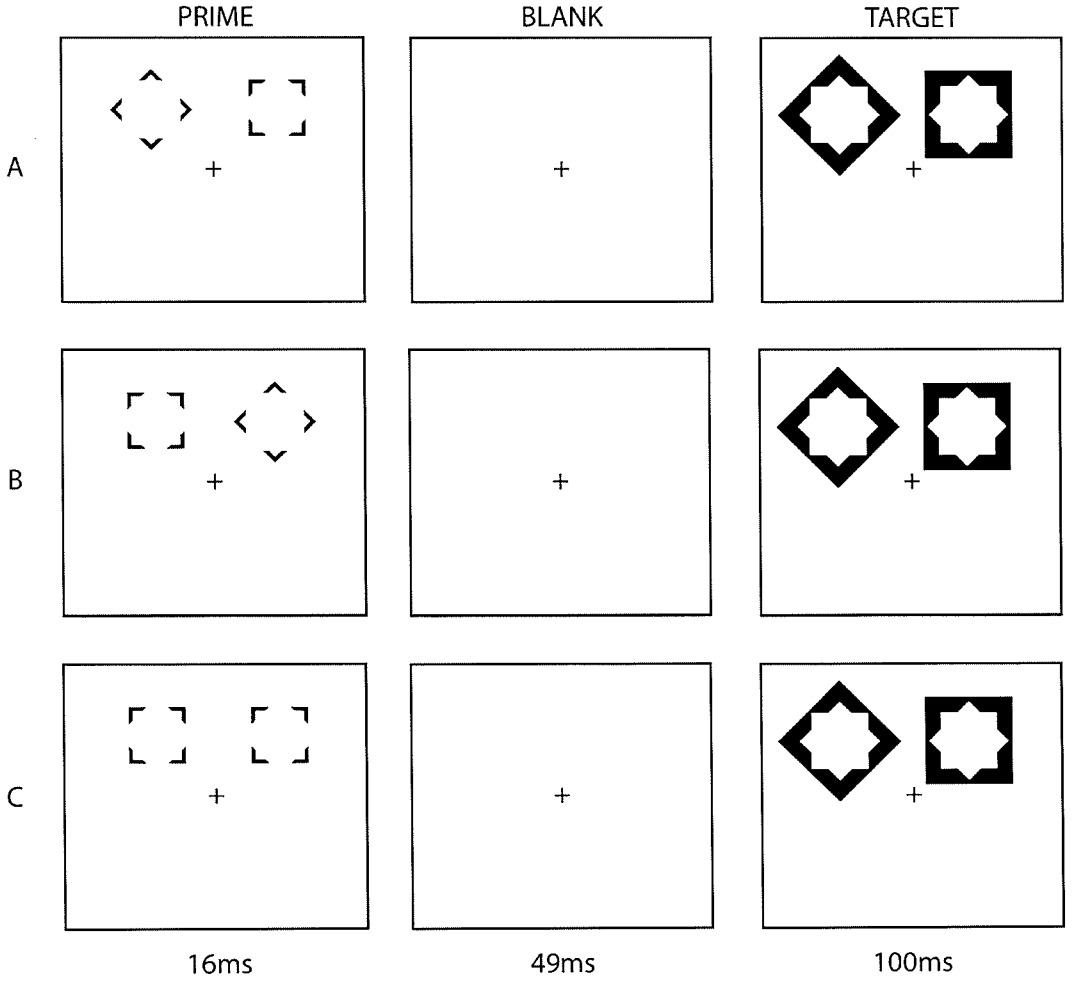

Figure 6.1: Stimuli for experiment 2. A congruent (A), incongruent (B) and neutral (C) trial.

Following the go/no-go task participants were asked the following questions: (1) Did you notice that there were stimuli presented prior to the diamonds and squares? (2) Could you tell what they were? (3) Did you notice anything flicker on the screen? Responses to these questions were noted by the experimenter. The participants were then fully informed of the nature of the

primes and were shown the sequence in slow motion. Participants were then asked (question 4) if they recognised having seen these primes during the go/no-go task. Participants then completed one practice block of 32 trials followed by four experimental blocks of the prime identification task. The exact same sequence was presented as that used in the go/no-go task, but now participants were asked to identify the location of the diamond in the prime pair. No neutral trials were presented in this task. Participants were asked to respond with the right button if the diamond was on the right and the left button if the diamond was on the left. Half the trials contained a left diamond prime and half contained a right diamond prime. Furthermore, half the trials were congruent and half were incongruent and participants were informed that using the target stimulus to make their judgement would not aid their performance. There was no time limit to respond. Participants received feedback after each trial. Following the prime identification task, participants were asked to estimate on what percentage of trials they were able to see the location of the diamond prime (question 5).

Behavioural Results

Awareness of Primes

None of the participants reported having seen any stimuli appear prior to the diamond and square targets. Table 6.1 shows participants responses to the questions regarding their subjective awareness of the masked primes.

Table 6.1: Summary of subjective awareness measures for experiment 2.

	Notice?	What?	Flash?	Recognise?	See?
NO	20	20	13	12	14%
YES	0	0	7	8	

Performance on the forced-choice ranged from 46% to 57% and averaged 51.6%. This represented a significant difference from chance ($t(19) = 2.34$, $p = 0.03$), d' scores were not significantly different from zero (mean = 0.06, $t = 2$, $p = 0.06$). Performance on the forced-choice task was not correlated

with participants self report of the number of trials where they thought the prime was visible (r=-0.097;p=0.7). The slightly above chance findings on the prime identification task suggests that some participants may have had residual awareness of the masked primes. To explore this possibility in more detail, later analysis attempted to correlate prime identification performance with the magnitude of priming effects under the assumption that residual awareness of the primes will affect both measure and will therefore show a significant correlation.

Priming

Table 6.2 shows a summary of the behavioural priming effects for experiment 2. Repeated measures ANOVA showed a significant main effect of prime-stimulus congruency on reaction times ($F(2,38)=36.7$, $p<0.001$) for go trials. A significant main effect of accuracy was observed for no-go trials ($F(2,38)=18.2$, $p<0.001$) but not for go trials($F(2,38)=0.97$, $p=0.39$). Subsequent t-tests showed a significant difference in reaction time between congruent and incongruent go trials ($t(19)=4.2$, $p<0.001$) and between congruent and neutral go trials ($t(19)=4.4$, $p<0.001$). No significant difference was observed between incongruent go and neutral go trials ($t(19)=1.8$, $p=0.08$). With regard to error rates on no-go trials, a significant difference was observed between congruent no-go and incongruent no-go ($t(19)=4.2$, $p<0.001$) and between incongruent no-go and neutral no-go ($t(19)=4.4$, $p<0.001$) for error rates such that more errors were observed for incongruent trials ($t(19)=1.8$, $p=0.08$).

Table 6.2.1: Mean Reaction times and accuracy (and Standard Deviations) for go trials

	Congruent	Incongruent	Neutral
RT	330 *(22)*	349 *(19)*	348 *(18)*
Accuracy	0.91 *(0.05)*	0.91 *(0.06)*	0.91 *(0.05)*

Table 6.2.2: Mean accuracy (and SD) for no-go trials

	Congruent	Incongruent	Neutral
Accuracy	0.94 *(0.03)*	0.86 *(0.08)*	0.95 *(0.03)*

There was no significant correlation between behavioural priming and prime identification using raw scores for percent correct and d'. In addition, absolute values of d prime and the absolute difference from 50% performance were also calculated and correlated with priming on the go/no-go task. There was a significant correlation between priming, measured as the difference in reaction time between congruent and incongruent go trials, and the absolute difference from 50% ($r=0.63$, $p<0.01$) on the prime identification task. Similarly, priming was also correlated with the absolute value of d' ($r=0.47$, $p<0.05$). There was no correlation between priming of error rates on go and no-go trials, and prime identification performance. These findings suggest that while there appeared to be an association between prime visibility and the magnitude of behavioural priming, this relationship was only observed when prime identification was measured as the absolute difference from chance. This issue will be explored in more detail in the discussion section of this chapter.

EEG Results

Two participants were excluded from the EEG analysis. One participant was excluded due to a hardware failure during recording and another was excluded due to incorrect coding of stimulus triggers. ERPs for the remaining 18 participants were formed from an average of between 77 and 85 trials per condition per response hand, with a minimum of 35 trials per condition per response hand.

Lateralised Readiness Potential

Figure 6.2 shows grand average LRP for the six conditions. Onset analysis was conducted only on go target trials. ANOVA showed a significant main effect of prime congruency on LRP onset ($F(1.9,31.5)=9.8$, $p<0.01$), with subsequent t-tests showing that LRP onset was significantly earlier for congruent trials in comparison to incongruent ($t(17)=3.59$, $p<0.005$), and neutral trials ($t(17)=3.46$, $p<0.005$). Incongruent no-go LRP onset did not differ from neutral go LRP onset.

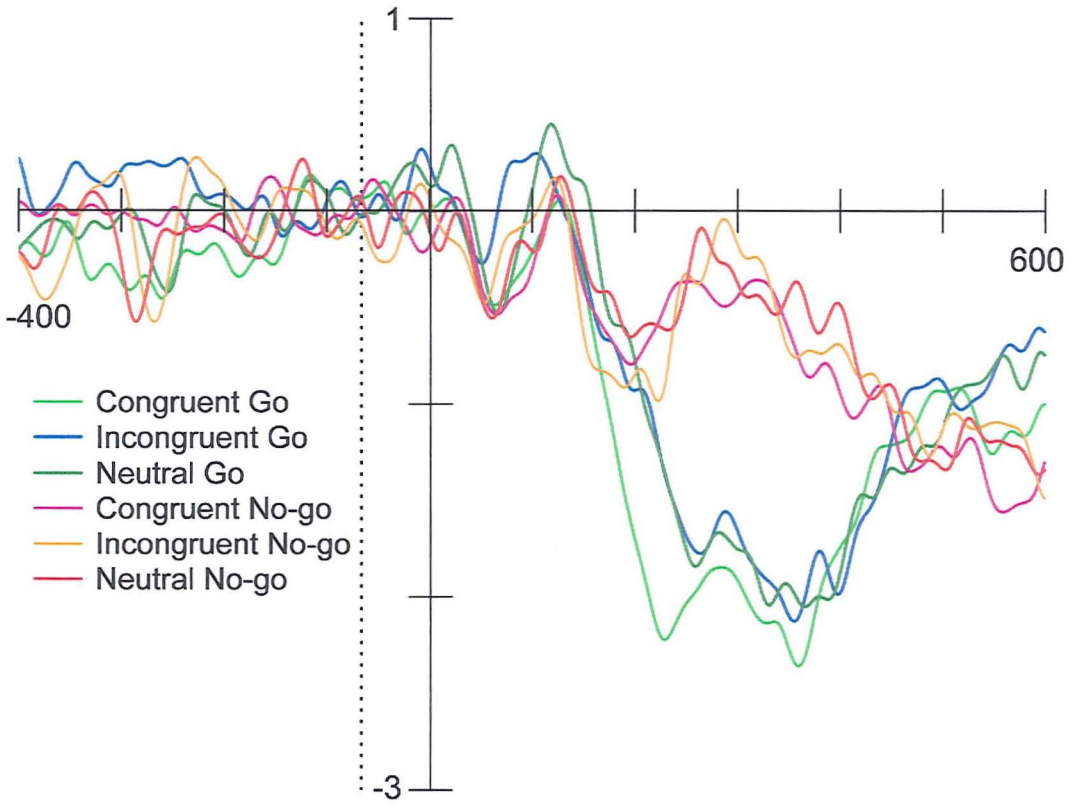

Figure 6.2: LRP for the three go conditions with respect to target onset. Prime onset at -100ms signified by dashed line.

Amplitude analysis was conducted on all six conditions with prime type (go, no-go, neutral) and target type (go, no-go) as repeated measures factors. In the 50 to 100ms time window there were no significant main effects, but there was a significant prime x target interaction ($F(2,33.2)=5.8$, $p<0.01$). Follow up t-tests revealed that this interaction was largely caused by a decreased negativity for incongruent go trials, which were significantly different to all other conditions except for incongruent no-go (at $p<0.05$). No other pairs significantly differed from one another. In a later time window (150 to 200ms) there was a significant main effect of prime ($F(2,33.2)=3.9$, $p<0.05$) but no effect of target and no prime x target interaction. Subsequent contrasts revealed that go-prime trials were significantly more negative than neutral prime trials ($F(1,17)=6.8$, $p<0.05$) but were not significantly different to go prime trials ($F(1,17)=3.3$, $p=0.085$). There was no difference between neutral and no-go prime trials ($p=0.32$). These findings suggest that the early part of the rising bank of the LRP was modulated dependent on the prime, with increased motor readiness for go primes. In a 200 to 450ms time

window there was a significant main effect of target type ($F(1,17)=67$, $p=0.001$) reflecting the increased LRP for go target trials.

Frontal no-go N2 and P3 analysis

Initial analysis focused on the hypothesised frontocentral modulation of the no-go N2 and P3. Figure 6.3 shows the no-go differences waveforms for each of the three no-go conditions compared to the neutral go condition. Two separate one-way ANOVAs were conducted at electrode Fz for the three no-go difference waveforms to explore the amplitude of the no-go N2 and P3. The N2 time window (200ms to 300ms) was chosen to encompass the period around the peak latency of the no-go N2. Similarly the P3 time window (375ms to 475ms) was meant to capture any differences in average amplitude of the no-go P3. Each ANOVA (one for each time window) included prime type as a repeated measures factor (congruent, incongruent and neutral).

In the N2 time window there was a significant main effect of prime congruency ($F(1.4,23.5)=19.9$, $p<0.001$). Follow up t-tests confirmed that incongruent no-go trials ($m=-5.3$; $std=2.8$) showed significantly more negative amplitude than neutral no-go trials ($m=-2.9$; $std=3$; $t(15)=5$, $p<0.001$) and congruent no-go trials ($m=-2.7$; $std=2.8$; $t(15)=4.5$, $p<0.001$). Neutral no-go N2 amplitude did not significantly differ from congruent no-go N2 ($t(15)=0.43$, $p=0.68$).

In the P3 time window there was once again a significant main effect of prime congruency ($F(1.8,30.4)=3.91$, $p<0.05$). Follow up t-tests confirmed that no-go P3 average amplitude was significantly greater for incongruent no-go trials ($m=4.2$; $std=5.6$) in comparison to neutral no-go trials ($m=2.96$; $std=5.4$; $t(17)=2.55$, $p<0.05$), and showed a non-significant trend in comparison to congruent no-go trials ($m=3.1$; $std=5.2$; $t(15)=2.02$, $p=0.059$). Neutral no-go P3 amplitude was not significantly different to congruent no-go P3 amplitude ($m=2.7$; $std=3.3$; $t(17)=-1.2$, $p=0.25$).

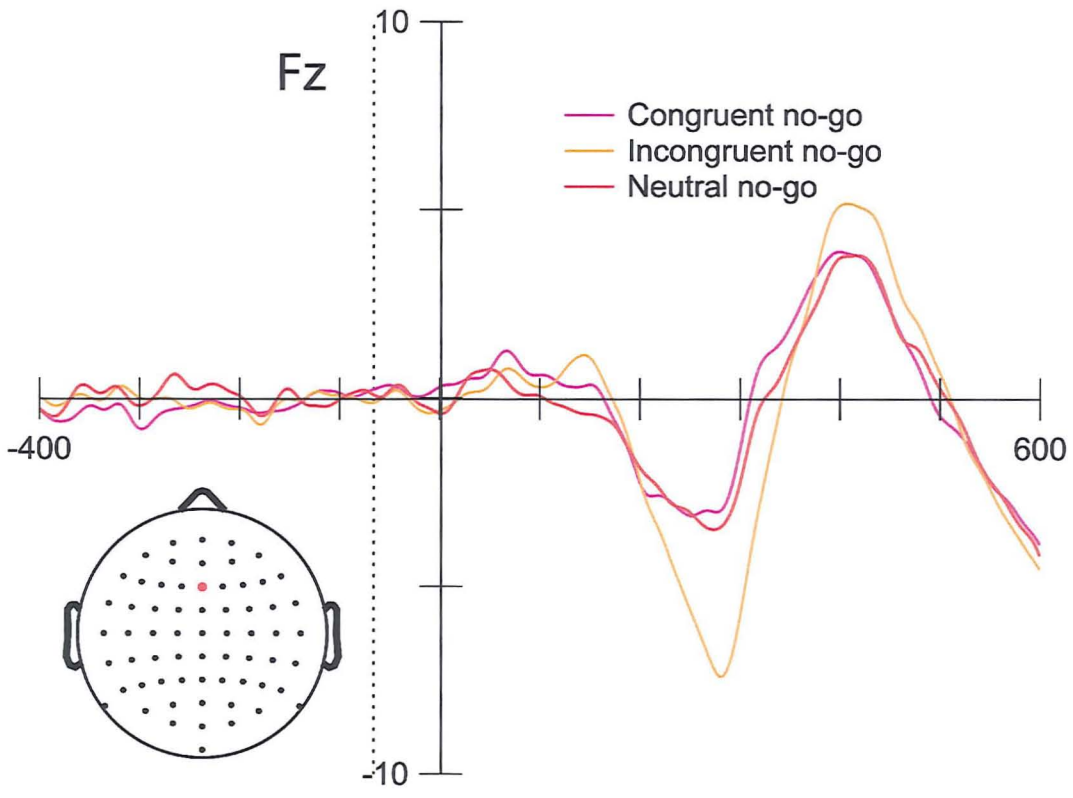

Figure 6.3: Difference ERP waveforms for the three no-go conditions at electrode Fz.

No-go N2 peak latency ($F(1.9,31.9)=5.7$, $p<0.01$) and amplitude ($F(1.3,22.5)=31.7$, $p<0.001$) were found to vary as a function of prime congruency. Subsequent t-tests confirmed that the N2 peak latency was significantly later for incongruent trials (m=287; std=27.4) in comparison to both neutral (m=261; std=36.1; $t(17)=2.8$, $p<0.05$) and congruent trials (m=256; std=37.8; $t(17)=3.3$, $p<0.01$). There was no significant modulation of P3 peak latency, but there was a significant effect of P3 peak amplitude ($F(1.7,29.6)=4.6$, $p<0.05$), with incongruent trials showing significantly greater P3 peak amplitude (m=7.2; std=6.2) in comparison to neutral trials (m=5.5; std=5.8; $t(17)=2.8$, $p<0.05$).

Further analysis was conducted to explore the possibility that the magnitude of the N2 and P3 effects were affected by participants' scores on the forced-choice tasks. Correlations were calculated between the three no-go difference amplitudes in each time window and performance on the two forced-choice tasks measured by both percentages correct and d' as well as

the absolute differences from chance. There were no significant correlations between N2 or P3 and performance on the prime identification task.

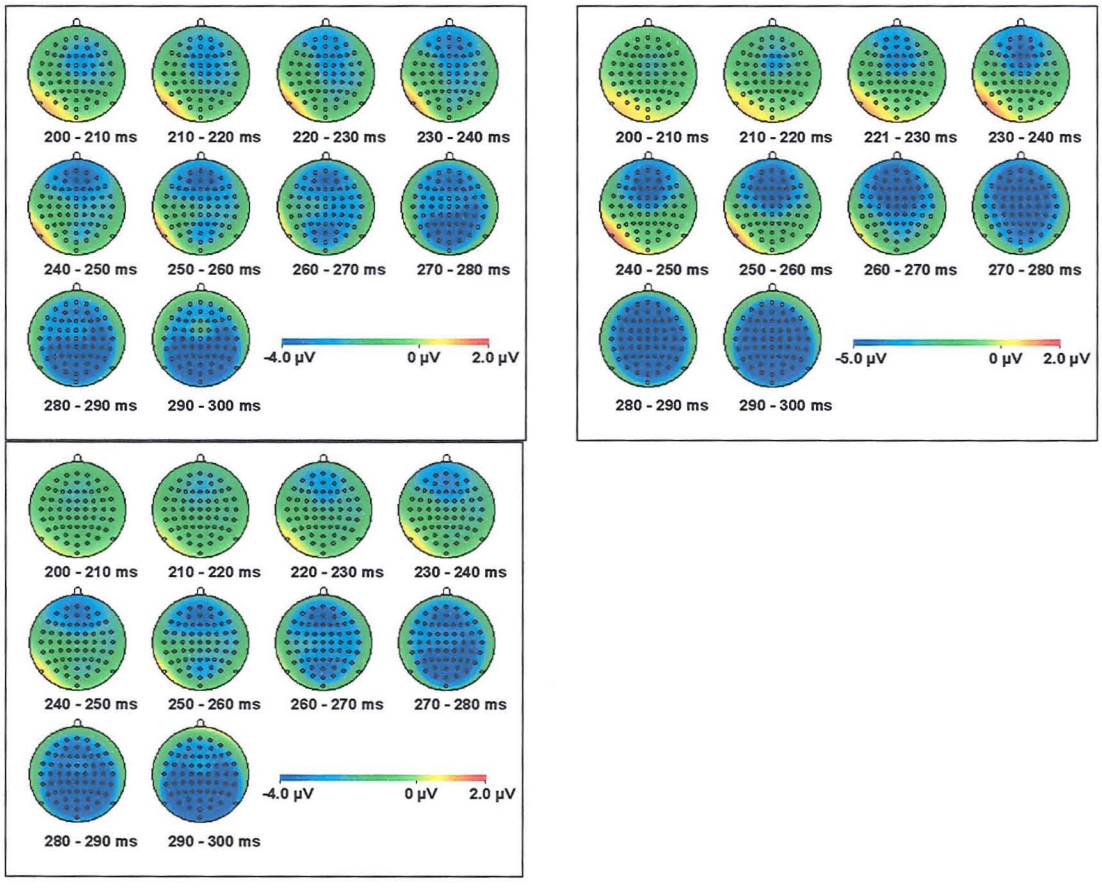

Figure 6.4: Scalp distribution of no-go N2 for congruent (top left), incongruent (top right) and neutral (bottom left) no-go trials. Each scalp map represents the average amplitude for the specified 10ms time window

N2 and P3 topography was explored by computing scalp maps of the difference waveforms for those conditions which showed a notable N2 or P3. Figure 6.4 shows the scalp maps for the no-go N2 for the congruent prime, incongruent and neutral prime conditions. In all three conditions the no-go N2 appears to initially show a frontal maximum, beginning around 200 ms. This initial frontal maximum then becomes rather more centrally and parietally distributed. Figure 6.5 shows the scalp distribution of the no-go P3 which appears to have a frontocentral distribution in all three conditions.

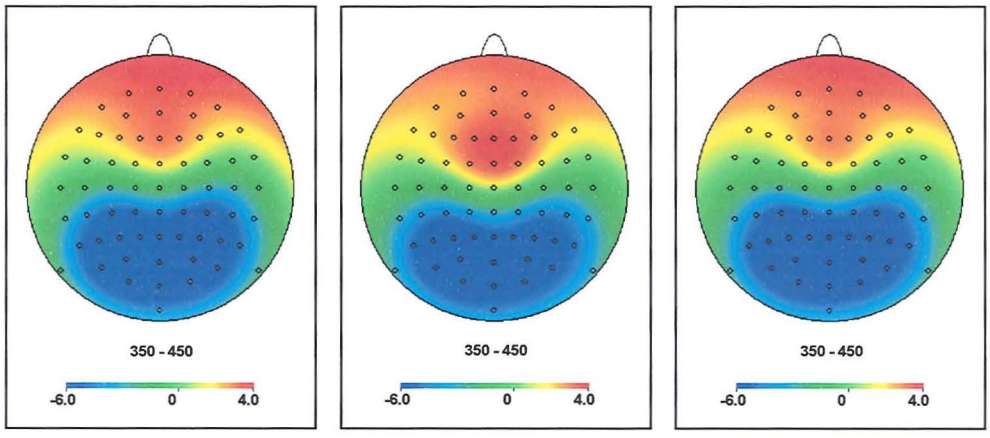

Figure 6.5: Scalp distribution of no-go P3 for congruent (left panel), incongruent (central panel) and neutral (right panel) no-go trials.

Finally, to explore the possibility that early prime-related effects may be present at electrode Fz, two-way ANOVA was conducted with prime type (go, no-go, neutral) and target type (go, no-go). Figure 6.6 shows the grand average ERPs at electrode Fz for all six conditions. Visual inspection reveals a clear no-go N2 peaking at around 350ms for no-go target trials, followed by a no-go P3 peaking at around 520ms (both explored above). In addition, a prime-related difference appears to be present overlapping a negative peak at around 150ms. ANOVA from 120 to 180ms revealed a significant main effect of prime type ($F(1.8,31.4)=9.2$, $p<0.001$), but no main effect of target and no target x prime interaction. Contrasts revealed that neutral prime trials were significantly more negative than go prime trials ($F(1,17)=14.9$, $p<0.001$) and no-go prime trials ($F(1,17)=7.6$, $p<0.05$). In addition, no-go prime trials showed a non-significant trend towards being more negative than go prime trials ($F(1,17)=3.6$, $p=0.075$). These early prime-related differences suggest that the unconscious primes were able to directly initiate frontal modulation of go/no-go ERP differences. However, the comparison between go and no-go primes failed to reach statistical significance, with neutral prime trials instead exhibiting statistical differences from both other conditions.

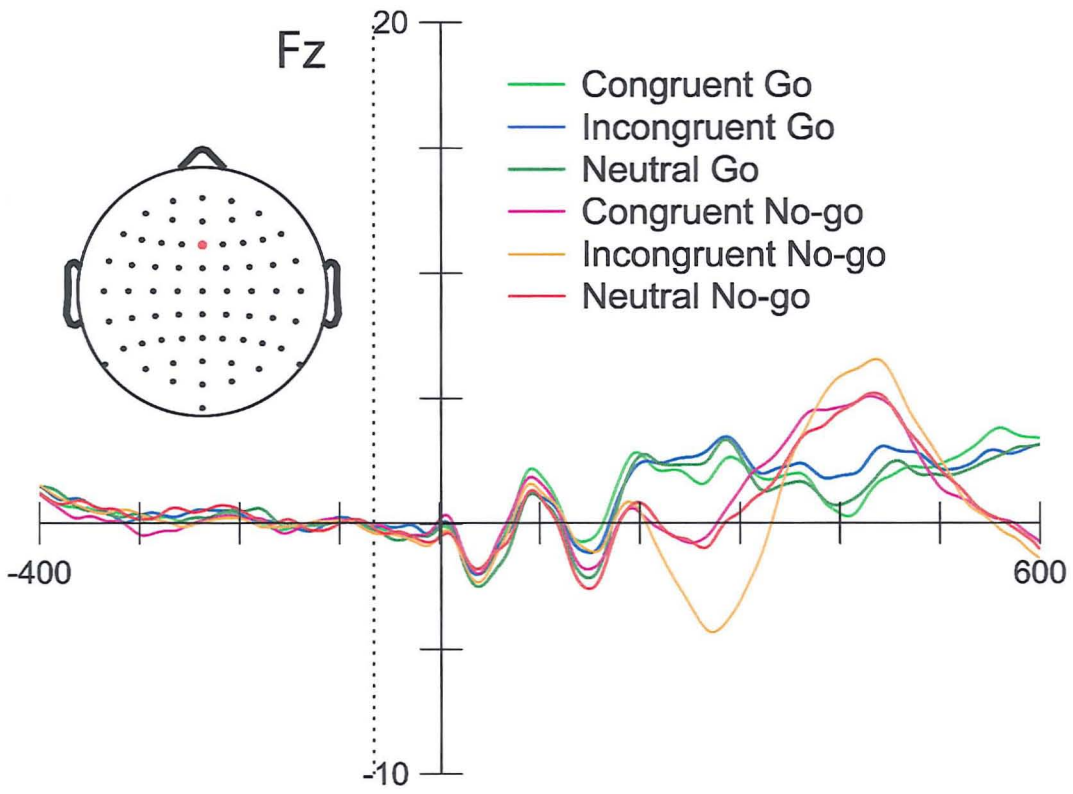

Figure 6.6: Grand average ERPs for the six conditions at electrode Fz

Early visual ERP effects

Figure 6.7 shows the grand average ERPs at electrode Oz for all six conditions. Repeated measures ANOVA with prime type (go, no-go and neutral) and target type (go, no-go) revealed a significant main effect of target for P1 (15 to 65ms) amplitude ($F(1,17)=5.26$, $p<0.05$). Follow up comparisons revealed that go target trials showed significantly greater P1 amplitude than no-go target trials ($t(17)=2.29$, $p<0.05$). In the N1 time window (90 to 125ms), ANOVA revealed a significant prime x target interaction for N1 amplitude ($F(1.7,29.5)=4.47$, $p<0.05$). Figure 6.7 shows that N1 amplitude appears to be greatest on congruent go trials and congruent no-go trials. In fact this difference is evident in the waveforms from as early as 60ms after stimulus onset. This may reflect an earlier onset of this component for congruent trials. Linear comparisons revealed significantly greater N1 amplitude for congruent trials (m=-3.3; std=4) in comparison to both neutral (m=-2.6; std=4.2; $t(17)=4.5$, $p<0.001$) and incongruent trials (m=-2.7; std=4.2; $t(17)=-1.2$, $p<0.05$). In addition,

incongruent go trials appear to have a greater amplitude than incongruent no-go trials and both neutral prime conditions, however paired t-tests revealed that these differences were not significant.

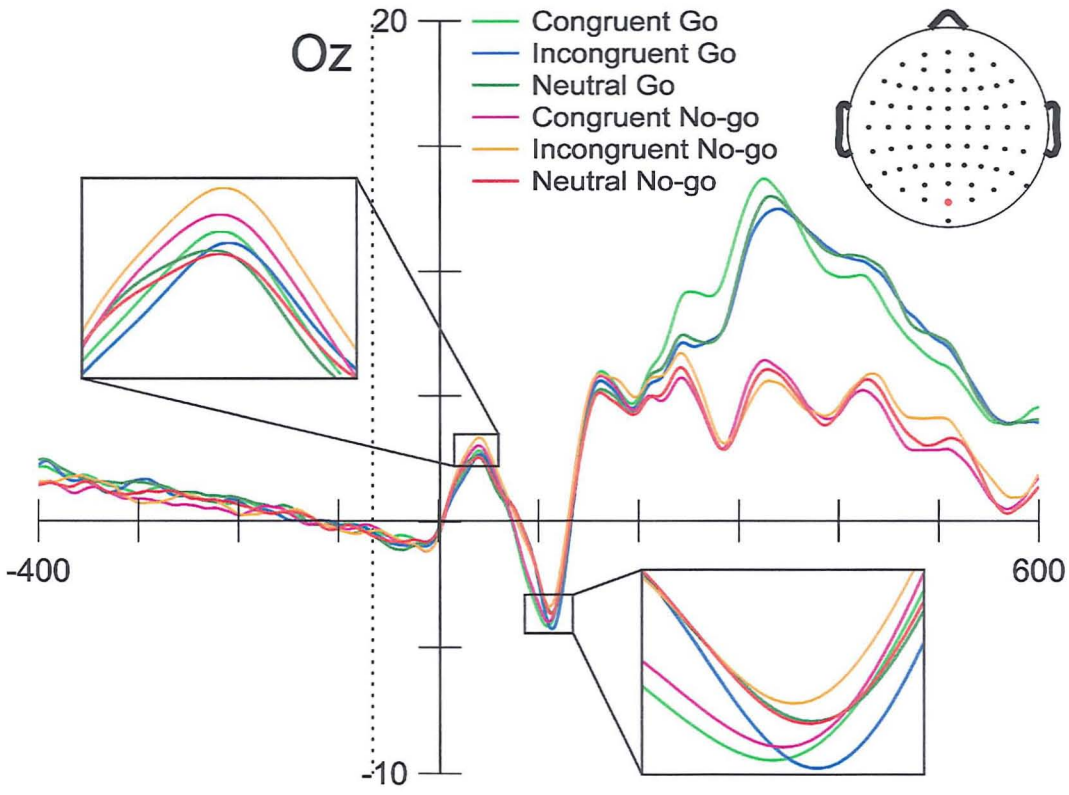

Figure 6.7: ERP waveforms at electrode Oz.

Further analysis was conducted to explore possible lateralised visual effects related to the physical target. Since targets are presented to both the left and right hemisphere it is likely that visual ERPs would be lateralised dependent on the particular stimulus array. Figure 6.8 shows grand average ERPs computed dependent on the physical stimulus properties regardless of the response requirements. The conditions are labelled with respect to the side on which a diamond was presented. For example left left indicates that a diamond prime was presented on the left and a diamond target was also presented in the left. Neutral primes consisted of two squares, so the neutral left condition would be one where two squares are presented followed by diamond target on the left. A clear separation between left prime trials, neutral prime trials and right prime trials is evident from around 20ms after

stimulus onset such that trials with a right prime appear more positive than trials with a left prime, with neutral primes in between. This time window corresponds roughly to the peak of the P1 component observed at electrode Oz (figure 6.7 above). An increased positivity in the difference waveforms below (figure 6.8) would thus reflect greater amplitude over left occipito/parietal regions in comparison to right sided electrodes. In the P1 time there appears to be general increased amplitude over right sided electrodes, but this is more pronounced when the prime stimulus (a diamond) appeared on the left side of the visual presentation. Similarly this effect is least pronounced following a left sided prime stimulus.

ANOVA was conducted to explore these effects in more detail. Each analysis included prime type (left, right, neutral) and target type (left,right) as repeated measures factors and response mapping as an independent factor. Response mapping was included as a factor to explore whether these lateralised effects were dependent on the meaning of the stimulus rather than its physical characteristics. Since the ERPs were formed by combining subjects with different response mappings to create ERPs dependent on the physical aspects of the prime, each ERP contains both left go and right go response mappings. Therefore, if lateralised responses were dependent on the nature of the stimulus then this should result in a significant interaction involving response mapping. ANOVA for average amplitude in the 20ms to 40ms time window showed that a near significant effect of prime ($F(1.6,25.3)=3.1$, $p=0.075$) and no effects involving target or response mapping. A significant linear contrast was present for prime type ($F(1,16)=7.2$, $p<0.05$), with right prime trials showing greatest positive amplitude and left prime trials showing least positive amplitude, confirming lateralised prime-related visual effects in this time window.

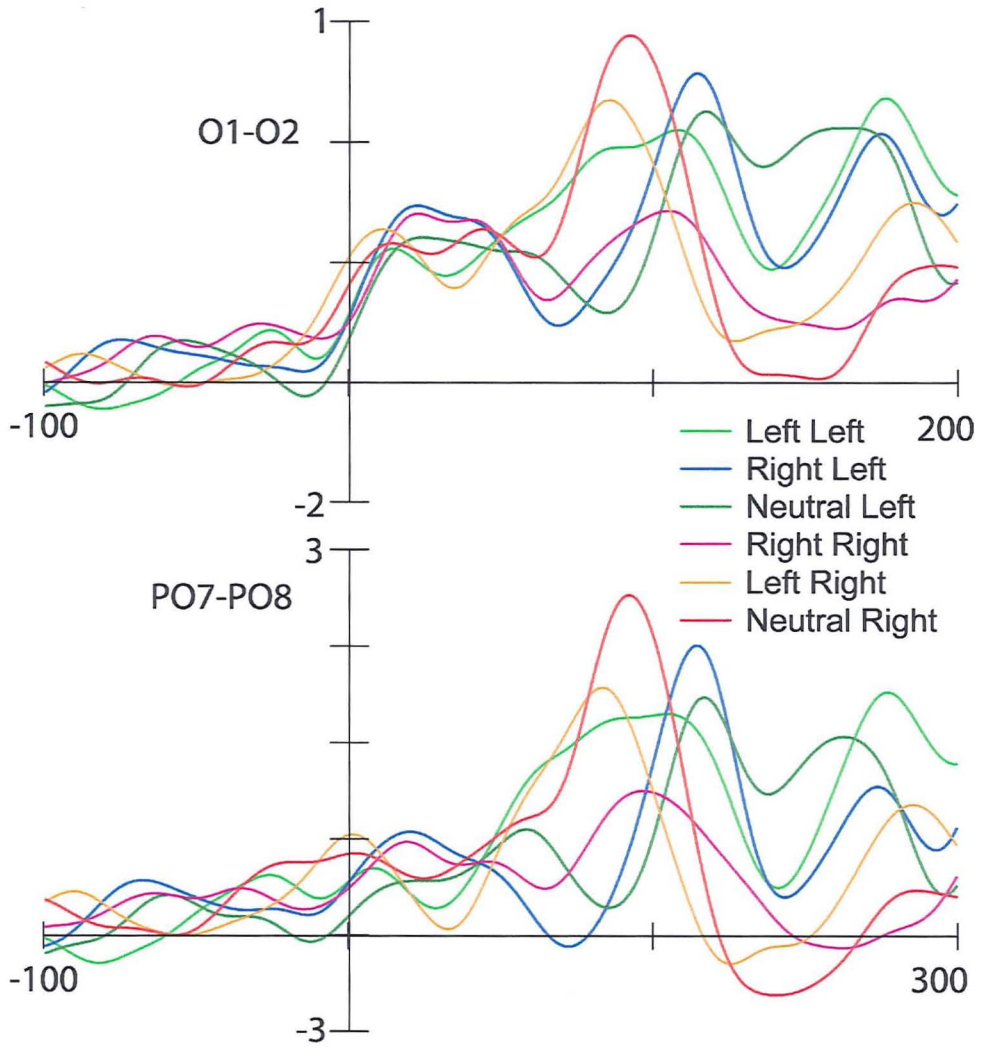

Figure 6.8: Lateralised occipital/parietal effects dependent on the physical stimuli presented in experiment 2.

In a later time window (60ms to 100ms) ANOVA revealed a significant main effect of prime ($F(1.5,23.8)=8.5$, $p<0.05$) as well as a significant main effect of target ($F(1,16)=18.6$, $p<0.001$) and a target x prime interaction ($F(1.9,30.5)=6.5$, $p<0.01$). There was no significant effect of response mapping. Visual inspection of the difference ERPs reveals that the waveforms separate based on prime type for left and right sided diamond primes, with right sided primes showing greater amplitude than left diamond primes. However, rather than remaining in the middle of the two different types of target stimuli, the neutral primes appear to separate based on target type with neutral left target trials showing greater positive amplitude than neutral right target trials. This means that while left-left left-right and neutral-

right group together, right-left right-right and neutral-left group together. Subsequent t-tests confirmed that all pair-wise comparisons between these groupings were significant (at $p<0.01$) while those within the groupings were not significant. One common difference between these groupings is that the former all have a square prime on the right, the latter all have a square prime on the left. However, this does not explain why the neutral primes separate as they do since they both contain two squares. From 100ms onwards the waveforms separated dependent on the target stimulus ($F(1,16)=7.9$, $p<0.001$), with no effects of response mapping. All the effects described above for the O1-O2 difference were identical for the PO7-PO8 difference.

Go/no-go Differences

This section provides a more comprehensive analysis of all the go/no-go differences observed in the current experiment. Figure 6.9 shows the raw ERP waveforms for the six conditions. The no-go N2 is clearly evident as a negative deflection beginning around 200 ms after stimulus presentation. In addition, a parietal separation is evident between go and no-go trials but does not appear to be modulated on no-go trials. Amplitude analysis of go/no-go differences was conducted using a five-way repeated measures ANOVA with prime type (go,no-go,neutral), target type (go,no-go), hemisphere (left,right), anterior-posterior (Fp, F, FC, C, P, and O) and time (120-180, 180-350, 350-550) as within-subjects factors. The early time window (120-180ms after target onset; 186-246ms after prime onset) was selected to explore any early differences in the ERPs associated with the unconscious primes. The second and third time windows were centred on the no-go N2 and no-go P3 respectively. The initial five-way ANOVA showed no main effects of hemisphere and no significant interactions involving hemisphere and target or prime type. Therefore, further analysis was conducted on the six midline electrodes only (Fpz, Fz, FCz, Cz, Pz, and Oz). Since there was a significant four way interaction between the other four factors ($F(4.9,84)=9.7$, $p<0.001$), further analysis explored the three way interactions between prime type, target type and anterior-posterior separately for each time window.

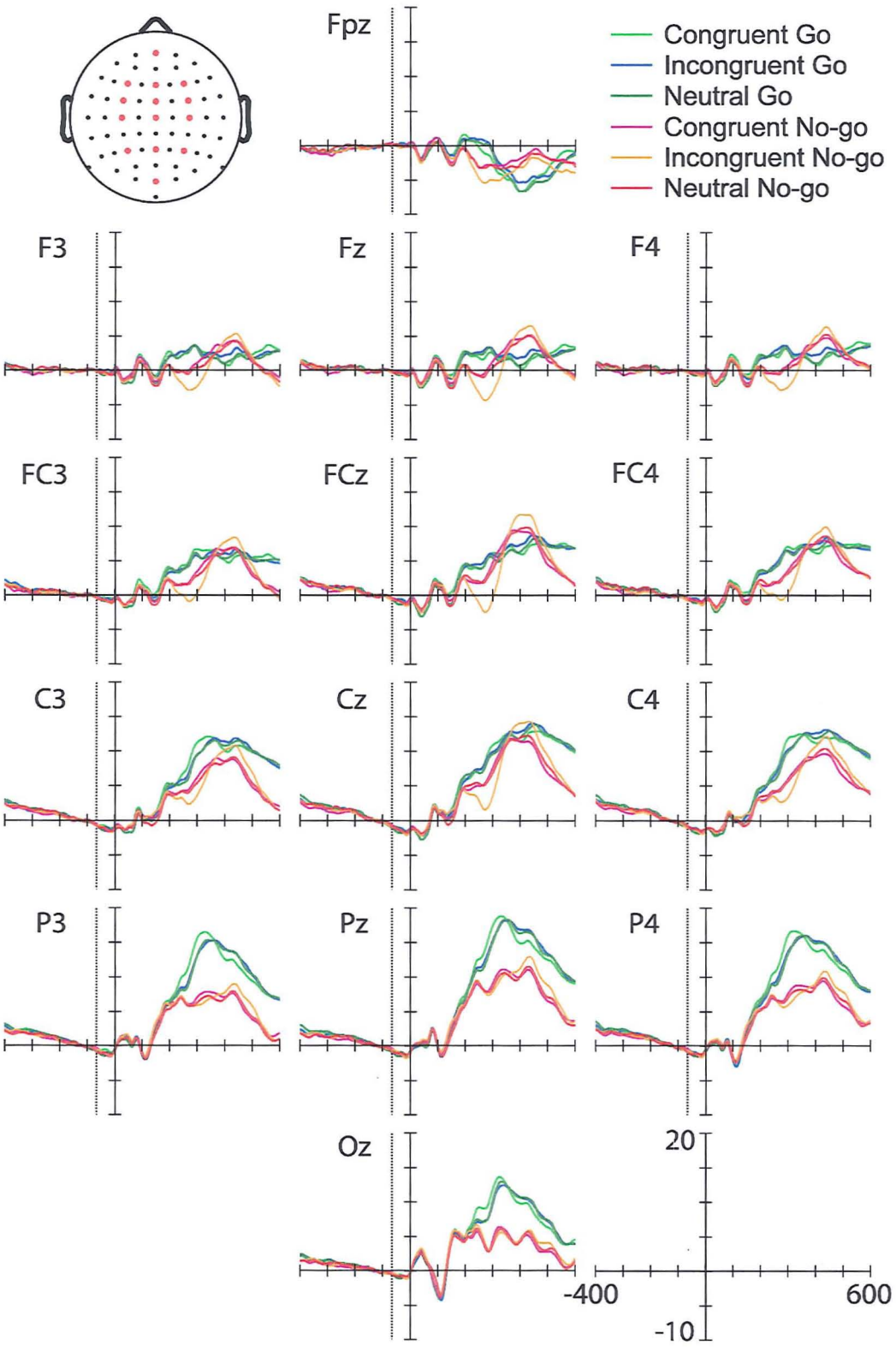

Figure 6.9: Raw ERP waveforms for experiment 2

In this first time window (120-180ms), there was a significant main effect of prime type ($F(1.9,32.4)=132.2$, $p<0.001$), but no significant effect of target type, and no significant interactions. Figure 6.10 shows the average amplitude for go prime, no-go prime and neutral prime trials collapsed across target type. The main effect of prime appears to be caused by a widespread effect of greater negative amplitude for neutral prime trials in comparison to go prime trials, which in turn are more negative than no-go prime trials. Follow up ANOVAs confirmed that a significant main effect of prime was present at all six electrode locations, with follow up contrasts revealing significant (at $p<0.01$) differences between neutral prime trials and go prime trials at electrodes Fz, FCz, and Cz, and maximal at FCz. In addition, no-go prime trials appeared to be somewhat more negative in this time window in comparison to go trials. This effect was maximal over electrode Fz although, as highlighted in the earlier analysis focused on Fz, it failed to reach statistical significance ($F(1,17)=3.61$, $p=0.075$).

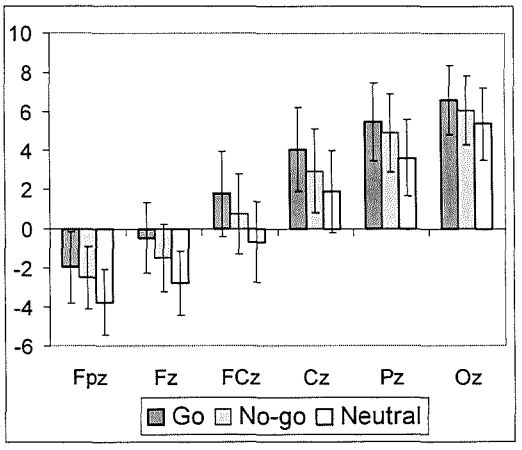

Figure 6.10: Average amplitude for midline electrodes in the first time window dependent on prime type collapsed across go and no-go target trials.

Amplitude analysis in the second time window (180ms to 350ms) revealed a significant main effect of target type($F(1,17)=28.5$, $p<0.001$) and a significant main effect prime type($F(1.7,28.7)=4.2$, $p<0.05$) as well as a prime x target interaction ($F(1.7,29.2)=11.1$, $p<0.001$). Furthermore, there was a marginally significant prime x target x anterior-posterior interaction ($F(2.4,40.2)=2.98$, $p=0.054$). Figure 6.11 shows the average amplitude in the second time window dependent on prime type for go trials and no-go trials separately.

For go trials the only significant difference occurred at electrode Pz, where go prime trials showed significantly greater positive amplitude than neutral prime trials. In contrast, for no-go trials, go prime trials showed significantly increased negativity in comparison to no-go prime trials at electrode Fpz Fz FCz and Cz and in comparison to neutral prime trials at Fz and FCz. These differences were greatest at FCz and likely reflect modulation of the no-go N2 for no-go trials.

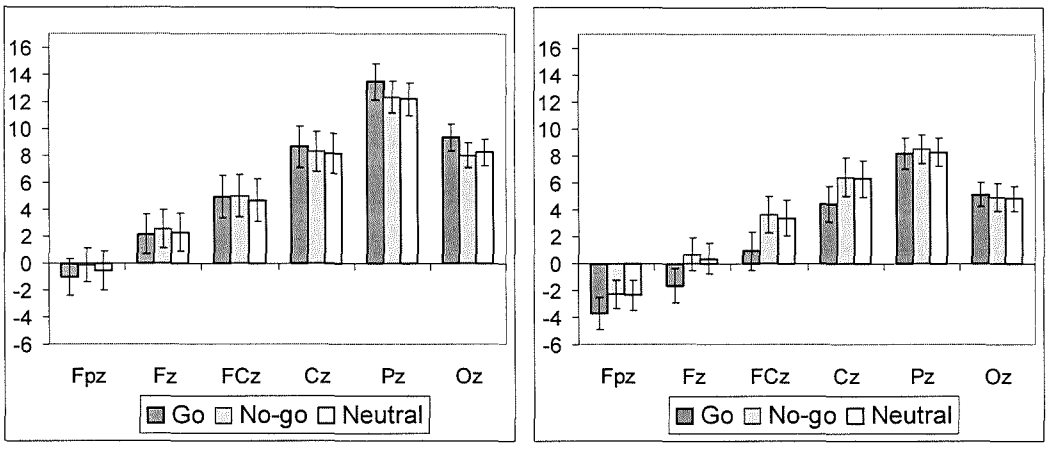

Figure 6.11: Average amplitude for midline electrodes in the second time window dependent on prime type for go targets (left panel) and no-go targets (right panel).

In the final time window (350 to 550ms) there was no main effect of target and no main effect of prime. However, there was a significant target x anterior-posterior interaction ($F(2.2,37.6)=5.6$, $p<0.05$) as well as a significant prime x target interaction ($F(1.7,28.9)=5.6$, $p<0.05$) and a significant prime x target x anterior-posterior interaction ($F(3.5,59.1)=7.6$, $p<0.001$). Figure 6.12 shows the average amplitude at each electrode dependent on prime type for go target trials (left panel) and no-go target trials (right panel). Inspection of these two graphs together reveals that the interaction between target type and anterior-posterior electrode location appears to be driven by differences between go and no-go target trials at posterior electrodes, visible as greater amplitude for target go trials (left panel) in comparison to no-go target trials (right panel). Follow up comparisons confirmed that go target trials shows significantly greater positive amplitude than no-go target trials at electrodes Pz and Oz. This difference likely reflects modulation of the parietal P300 component. The left panel of figure 6.12 shows the average amplitude at each electrode for go

target trials dependent on prime type. T-tests revealed that there were no significant differences at any electrode sites for go target trials. The right panel of figure 6.12 shows that average amplitude for no-go target trials in the final time window. It is evident that go prime trials show a significantly greater positive amplitude in comparison to neutral and no-go prime trials over central and anterior electrode sites. This effect only reached statistical significance (at $p<0.001$) at electrode Cz and likely reflects modulation of the no-go P3.

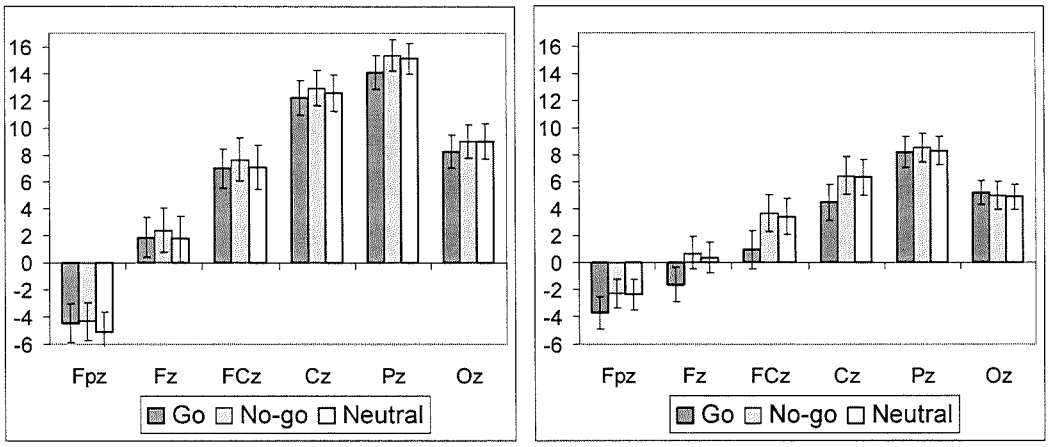

Figure 6.12: Average amplitude for midline electrodes in the final time window dependent on prime type for go targets (left panel) and no-go targets (right panel).

LRP and go/no-go differences

To explore the possibility that the effects observed at central electrodes were projections of movement related activity at lateral electrodes, grand average ERPs were computed separately for left and right hand responses. Figure 6.13 shows grand average ERPs for left and right hand trials separately at electrodes FC3, FC4 and FCz for congruent and incongruent no-go conditions. It is evident that the effects described above are not simply projections of lateralised movement activity. The two squares showing the ERPs between 100 and 200ms reveal that the early prime effects are not caused by lateralised effects which are then averaged together, since the no-go prime conditions is more negative at both lateral electrode sites. Similarly, the increased no-go N2 for incongruent trials is evident both for both left and right hand responses over both left and right hemisphere. The amplitude at FCz appears to be greater than that observed over the lateral

sites suggesting a central topography to the effects, again ruling out the possibility of contamination from lateralised movement activity.

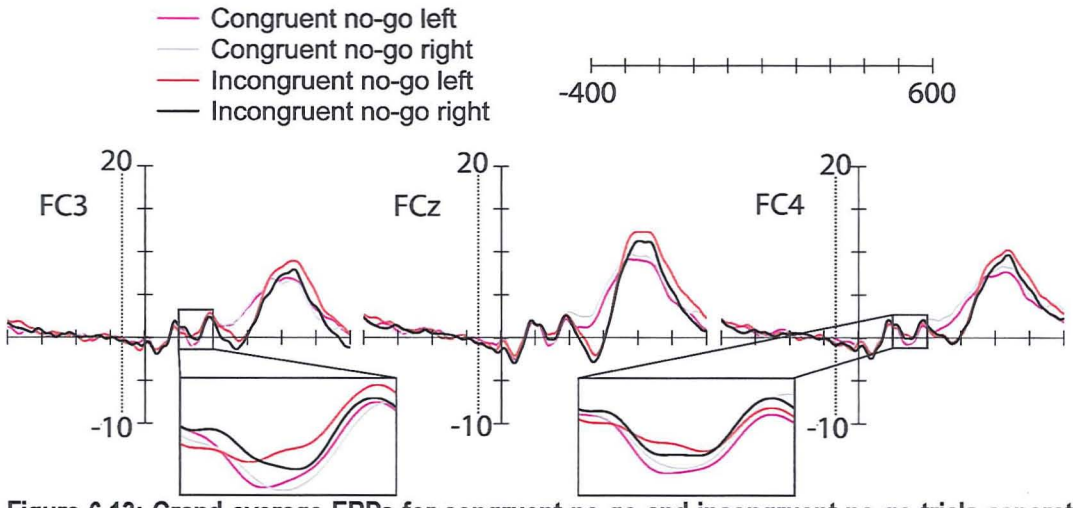

Figure 6.13: Grand average ERPs for congruent no-go and incongruent no-go trials separated by response hand.

Discussion

This investigation aimed to replicate the effects observed in the experiment 1: that subliminal primes were able modulate the no-go N2 and P3 components and further that they were able to directly initiate these processes. Participants were presented with a diamond and a square on each trial and were asked to make a go response for one combination of the targets and a no-go response to the opposite stimulus array. Unconscious primes presented in advance of these stimuli were shown to influence reaction times to go targets, with slower reaction times for incongruent primes and faster reaction times for congruent primes. However, there was no significant difference in reaction times between incongruent go trials and neutral go trials, suggesting that the neutral prime acted in a similar way to a no-go prime. Similarly, for error rates there were significant differences between incongruent no-go trials and both neutral and congruent no-go trials, but the latter two conditions did not differ from one another. This shows that once again the neutral primes appeared to behave much like no-go primes.

Similarly, analysis of the frontal no-go N2 and P3 revealed that both these components showed significantly greater amplitude for incongruent no-go trials in comparison to neutral no-go trials, but that neutral and no-go prime trials did not differ. A possible explanation for this is that although participants were instructed to attend to both stimuli (a diamond on one side codes for go and the other side for no-go), the task is easily accomplished by simply attending to the side where a go stimulus will be present. For example those participants who were instructed to go in response to a left diamond and not go following a right diamond could simply attend to the left and go if a diamond was presented, but not go if a square was presented. This would mean that on the side where attention is allocated the neutral prime and no-go prime would be identical (a square) since neutral primes are always two squares and incongruent primes are a square and a diamond with the diamond on the opposite side. This is highlighted by examining figure 6.1 and looking at the sequence of stimuli presented left of fixation. Comparing only the left of the stimulus array of an incongruent trial (B) and a neutral trial (C) reveals that the stimulus sequence presented on this side of the screen were essentially identical.

The more in-depth analysis of ERP go/no-go differences replicated many of the effects observed in the first experiment. Crucially, for the hypothesis that the primes could directly initiate frontal control mechanisms, there was once again a significant main effect of prime in the early time window. This appeared to be largely driven by greater negative amplitude for neutral prime trials in comparison to go prime trials. No-go prime trials also appeared somewhat negative in relation to go prime trials, showing a maximum at electrode FCz, although this contrast only reached a significance level of $p=0.075$. Even disregarding this trend, the finding that neutral prime trials showed increased negativity in this time window is consistent with the explanation above that these trials effectively acted as no-go trials. However, this effect must also be treated with some caution since due to the complications observed with the neutral trials in the current experiment. These early prime-related effects at frontal electrodes appear to support the hypothesis that the subliminal prime was able to directly initiate frontal

control mechanisms in this task. While some of these early effects were significant it is noteworthy that these effects are much smaller than those observed in the first experiment.

The effects in the second and third time window were very similar to those observed in experiment 1. Firstly, a similar dissociation was observed between prime-related modulations on go and no-go trials. For no-go trials the effect of prime was maximal over frontocentral electrodes, while for go trials this effect was limited to electrode Pz only. However, similar to the initial analysis at frontal electrodes these differences were only evident between go and no-go primes as well as go and neutral primes, with activity for no-go and neutral primes grouping together. Similarly in the third and final time window neutral/no-go and go prime trials showed significant modulation at anterior electrodes related to the no-go P3.

LRP analysis revealed a significant effect of LRP onset for go trials. Two early amplitude modulations were also evident. The early effect, just 50ms after stimulus onset was caused by a decreased LRP activation for incongruent go trials. However, since the same decreased was not evident for congruent no-go trials, this effect was classified by a prime x target interaction. A more reliable effect was also evident in the 150 to 250ms time window, where the LRP separated dependent on the nature of the prime. As with the other prime-related effects in this experiment, go primes were different from neutral and no-go primes, with neutral and no-go primes showing no difference. This finding once again supports the assumption that neutral primes acted as no-go primes in the current experiment, and provides further support for the assumption that a motor response can be directly prepared by a subliminal prime.

Examination of visual ERPs revealed a significant effect of prime congruency at electrode Oz from around 60ms after stimulus onset with greater negative amplitude for the rising bank of the N1 component for congruent trials. This suggests that the difference between congruent and incongruent trials was detected in the visual system extremely early. Further analysis of lateralised

ERPs at occipital and parietal electrodes also revealed an early separation dependent on prime type, with increased visual P1 and N1 components contralateral to a diamond prime. These findings suggest that the locations of the different prime stimuli were able to initiate lateralised visual effects related to either perception of the target or increased attention towards one location or the other. The congruency effects at Oz suggest that perhaps these early lateralised responses to the prime modulated attention such that a greater visual response was then evident for congruent trials, since the prime had successfully directed attention toward that location. Consistent with this interpretation there is evidence to suggest that increased attention toward a spatial location of a subsequent stimulus increases the amplitude of the visual response (Martinez et al., 2001) from around 70ms after stimulus onset. However, since these lateralised effects were purely dependent on the physical characteristics of the primes and did not vary dependent on the functional significance of the primes, this suggests that these effects reflect visual detection of the different targets. The fact that these effects occurred despite the two different prime stimuli being simply a 90 degree rotation of one another suggests that this visual discrimination must have involved some selection of target relevant dimensions to discriminate which of the two stimuli were presented.

The finding that congruency related visual ERP effects emerged so early is slightly problematic for the interpretation that the early ERP effects at frontal electrodes reflected direct effects of the unconscious primes, since it is possible that this effect was produced by an earlier response to the target due to the increased attention toward the target location. However, this explanation seems unlikely given the early prime-related modulation at electrode Fz. For example if the priming of the no-go response was simply caused by earlier categorisation of the target due to increased attention or faster visual categorisation one would expect an earlier N2 for congruent no-go trials but no prime-related negativity for incongruent go trials. This would manifest as a prime x target interaction in the early time window at Fz. However, in this time window the ERPs separated entirely dependent on the prime with no target effects and no interaction, supporting the assumption

that the modulation of the no-go response was caused by a direct partial activation of the no-go response following a no-go prime. It is worth reiterating once again, however, that this early prime-related effect at Fz was only partially reliable, and therefore can not entirely rule out a simple attentional explanation for the priming effects observed in the current experiment.

Finally, performance on the Prime identification task was found to be marginally above chance when measured as the difference from 50% accuracy on the forced-choice task but not when measured using d'. In addition there was no significant correlation between either of these measures and priming in the go/no-go task, suggesting that the priming effects were not driven by residual awareness. Interestingly there was a significant correlation between the absolute difference from chance in the prime identification task and the amount of priming. These absolute measures reflected the difference from chance regardless of the direction of this distance. For example, 53% accuracy and 47% accuracy were both 3% away from chance performance. The correlation between these measures suggests that there may have been at least some aspect of performance in the two tasks that were related. It is important to note that participants received feedback on an individual trial basis in the forced-choice task, so it should be expected that if participants were able to detect some subtle differences between the two primes then they would have been able to use the feedback to perform above chance rather than below chance. However, as outlined in chapter 3, there is some evidence to suggest that flexible adaptive responses to unconscious stimuli may not be possible (Mayr, 2004). In fact the process dissociation task works on the basis of this assumption, that while we can flexible control our responses to conscious stimuli in order to exclude them under certain instructions, we are not able to do the same for unconscious stimuli (Jacoby, 1991). Under this assumption it would be possible to conclude that the residual discrimination of the prime which correlated with priming performance was driven by unconscious processes. If participants were conscious of the stimulus, they could presumably have utilised the feedback to perform consistently above chance. If however,

unconscious discrimination of the prime in the identification task could not be controlled, then it would be expected to differ from chance in a systematic way, but not such that it was always above chance since participants would be able to reliably respond one way or another, but would not be able to adjust their response dependent on feedback. This is precisely what was observed in the current experiment and therefore it seems likely that the marginally above chance performance on the forced-choice task were not driven by conscious awareness of the stimulus. While there are a number of problems regarding this position (which will be discussed further in chapter 10), it is certainly noteworthy that this type of analysis could provide interesting insight into the relative contribution of conscious and unconscious processes in the prime identification task.

Conclusions

This experiment replicated a number of effects observed in the first experiment. No-go N2 and P3 amplitude was found to vary as a function of the unconscious prime. Importantly, in this experiment this modulation reflected a positive compatibility effect such that congruent go trials showed significantly reduced N2 and P3 amplitude in comparison to neutral trials. In addition, a significant early frontal modulation was evident dependent on the nature of the subliminal prime, suggesting it was able to directly initiate frontal control processes. However, this modulation was markedly smaller than in Experiment 1 with the specific contrasts of interest failing to reach strict significance. A further problem with the current experiment was that neutral primes appeared to group together with no go primes, possibly due to increased attention toward the side where the go stimulus would appear. In summary, the present experiment replicated the modulation of target-related N2 and P3 components dependent on the nature of the unconscious prime observed in Experiment 1, and provided further evidence of direct unconscious engagement of frontal inhibition/control mechanisms.

Chapter 7

Experiment 3 - Unconscious priming of a no-go response in a choice reaction time task.

Introduction

Experiment 3 aimed to overcome the problem caused with the neutral primes in experiment 2. This problem was attributed to the fact that participants could easily complete the task by attending to one side of the visual display, which in turn changed the utility of the neutral primes. In the current experiment, in order to ensure that participants attended to both sides, the stimulus-response parameters were slightly altered. Participants were required to respond with their left hand to a left side diamond and their right hand to a right side diamond. Two squares indicated a no-go trial. The stimuli were otherwise identical to experiment 2, with the exception that neutral primes were a combination of features from the two possible targets. Since participants would have to determine on which side of the screen a target stimulus was presented they must attend to both sides of the screen to complete the task. Unlike in experiment 2 the task instructions cannot be easily recoded to a rule that involves one side of the screen. While a diamond on one side codes for a go response with the relevant hand, a square to one side could either mean an opposite hand response or a no-go trial.

This manipulation should also allow exploration of priming of go and no-go responses alongside priming of specific hand responses. This will allow direct exploration of the type of effects observed in the many paradigms involving left and right hand response priming, alongside go/no-go priming. Of particular interest, is Leuthold and Kopp's (1999) finding that a parietal N2 was observed for incongruent go trials. Eimer and Schlaghecken (2000) have suggested that while this more parietal N2 is possible in response to unconscious primes, modulation of a frontal N2 is not. The current experiment will be able to explore these two components together.

Hypotheses

As in experiment 1 and 2 the presence of subliminal primes which code for a no-go response should directly modulate ERP amplitude at frontal electrode sites. This modulation should be evident on the no-go N2 and P3 such that congruent no-go trials will show reduced amplitude on these components in comparison to neutral trials, which in turn will be reduce in comparison to incongruent no-go trials. In addition, if the primes are able to directly initiate the frontal no-go N2/P3 complex then some prime-related modulation should be evident in advance of the target-related N2 and P3. Additionally the current experiment will explore whether a parietal N2 is evident for trials where a response with one hand is primed, but a response to the other hand is ultimately required (incongruent go trials).

Method

Participants

Fourteen undergraduate psychology students (seven male and seven female) participated in exchange for course credits. All participants were right handed and had normal or corrected to normal vision. The mean age of participants was 20 years and 10 months, with a range of 18 to 28 years.

Experimental Procedure

All participants completed a single experimental session lasting approximately two hours. The participants completed 12 blocks of the go/no-go task followed by four blocks of the prime identification task. Each go/no-go block contained 128 trials presented in a random order. The 12 experimental blocks were preceded by one practice block of 48 trials. On each trial two shapes appeared on the screen. On half the trials there were two squares whilst on the other half a diamond was presented on the left or the right. The two shapes appeared randomly above or below fixation. Participants were required to respond with their right hand to a right sided diamond and their left hand for a left sided diamond. If no diamond was presented then participants were asked to refrain from responding (no-go condition). The participants were informed that that they had a time limit of

500ms to respond to the go stimuli and that they should react as quickly as possible without sacrificing accuracy. Participants were given visual feedback immediately after the 500ms response window for correct responses, incorrect responses and non-response as well as false alarms and incorrect non-responses.

Masked primes were presented prior to the target stimulus. The prime consisted of a pair of shapes presented at the same location as the target shapes. The configuration of these shapes was congruent, incongruent, or neutral with respect to the target stimulus. On congruent go trials the prime contained a square and a diamond in the same configuration as the target, whilst incongruent primes had an opposite configuration to the target. No-go primes consisted of two squares. Neutral primes consisted of a square on one side and a neutral prime on the other side. The neutral prime was made up of two features of the square prime and two features of the target prime. The four different neutral primes are presented in figure 7.1.

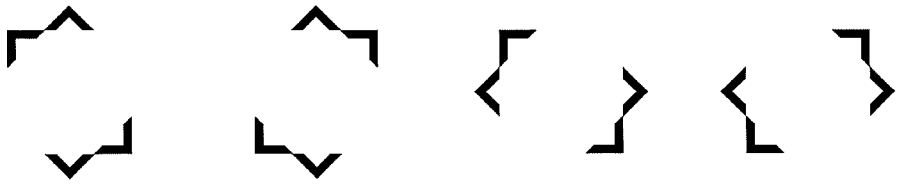

Figure 7.1: The four neutral primes for experiment 3

There were an equal number of congruent, incongruent and neutral trials in each block. Some examples of these different conditions are presented in figure 7.2. The primes were masked by the target stimulus. The primes and target were the identical size to those in experiment 2. The primes fit exactly into the internal contours of both target shapes for optimized metacontrast masking.

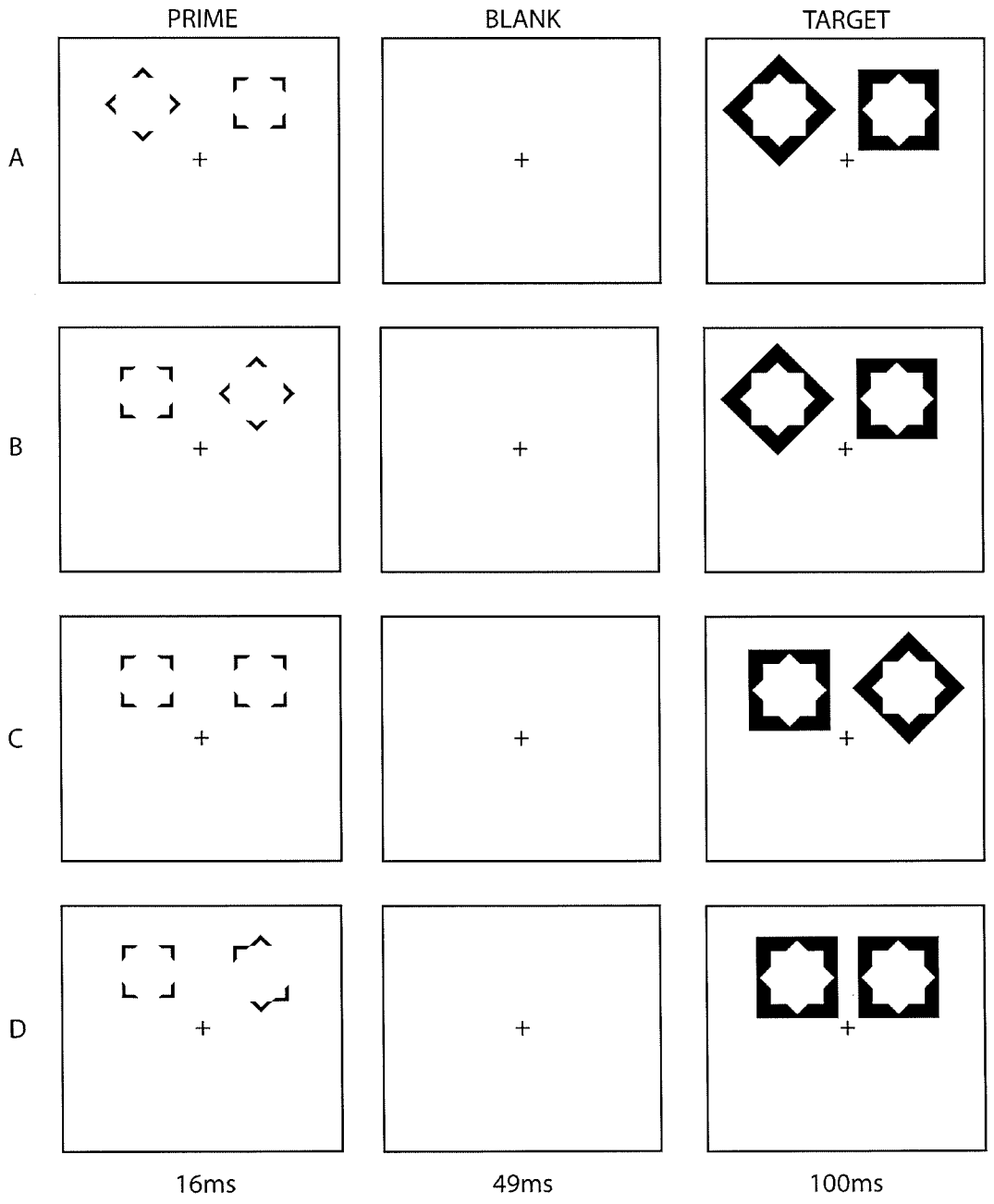

Figure 7.2: A congruent Go (A), Incongruent Go (B), No-go Go (C) and neutral No-go trial for experiment 3

Each trial began with a central fixation for 800 ms and then the primes were presented for 16ms followed by a blank screen for 49ms and then the mask/target for 100ms. Participants had 500ms to respond, after which visual feedback was presented for 400ms. Finally, "blink pause" was presented in the centre of the screen for 1000ms and participants were informed to use this time to blink if they needed. Participants were also informed not to blink during the trial, and to keep their eyes fixated on the centre of the screen.

Following the go/no-go task the participants were asked if they saw anything appear before the target, and if so what they saw. In addition, they were asked if they saw a flash on the screen in advance of the target. Participants' were then shown the sequence in slow motion. After seeing the slowed sequence participants were asked if they recognised having seen any of the primes during the go/no-go task. The prime identification task consisted of one practice block and four experimental blocks of 96 trials. Left diamond, right diamond and two squares primes each made up a third of trials. No neutral primes were presented during this task. Similarly a third of all trials showed a diamond left target, a diamond right target and a two squares target. The prime and target were congruent on a third of trials, and participants were informed that responding according to the target would not improve their accuracy. Participants were informed that they should press the far left button if the diamond prime was on the left, the far right button if the diamond prime was on the right, or the middle button if two squares were presented. Participants had no time limit to respond, and received feedback at the end of each trial.

Behavioural Results

Awareness of Primes

Table 1 shows the frequencies for the responses to questions 1 to 4. Although three participants reported that they noticed something before the target stimulus, none were able to report what had been presented. The most reliable indicator of performance in the prime identification task was whether participants recognised having seen the prime after being shown the slow motion sequence. Three out of five participants who later performed above chance reported recognising the primes, with only one of the nine unaware subjects reporting recognition.

Table 7.1: summary of responses to the four questions

	Notice	What	Flash	Recognise
No	11	14	7	10
Yes	3	0	7	4

Overall performance on the prime identification task was significantly above chance ($t(13) = 2.25$, $p < 0.05$). When five participants with accuracy of over 37%, were excluded, performance for the remaining nine participants did not significantly differ from chance ($t(8) = 0.52$, $p = 0.62$). Subsequent analysis was conducted on all 14 participants with correlations between prime identification performance and priming calculated at each step. In addition, to ensure that any observed effects were truly unconscious, all major analyses were repeated with only the nine participants who performed at chance level.

Priming

A summary of the reaction times and error rates for go and no-go trials is shown in table 7.2. Repeated measures ANOVA showed a significant main effect of prime congruency on reaction time ($F(1.6,20.2)=43.7$, $p<0.001$) and error rates ($F(1.8,23.2)=14.1$, $p<0.001$) for the four go conditions. Subsequent t-tests showed that RT were significantly reduced for congruent trials in comparison to incongruent ($t(13)=6.9$, $p<0.001$) neutral ($t(13)=8.2$, $p<0.001$) and no-go ($t(13)=9.5$, $p<0.001$) go trials. There was also a significant difference between RT on incongruent and neutral go trials ($t(13)=3.8$, $p<0.01$). Accuracy was significantly higher for congruent go trials in comparison to incongruent ($t(13)=4.4$, $p<0.001$) and neutral ($t(13)=3.5$, $p<0.005$) go trials. For no-go trials there was a significant main effect of prime congruency on error rates ($F(1.2,16.1)=6.3$, $p<0.05$), with follow up comparisons showing that congruent no-go trials had significantly fewer errors than neutral no-go trials ($t(13)=3.6$, $p<0.005$) and incongruent no-go trials ($t(13)=2.6$, $p<0.05$).

Table 7.2.1: Mean Reaction times and accuracy (and Standard Deviations) for go trials

	Congruent	Incongruent	Neutral	No-Go
RT	348 *(30)*	375 *(29)*	366 *(29)*	370 *(30)*
Acc	0.93 *(0.04)*	0.86 *(0.9)*	0.89 *(0.07)*	0.93 *(0.04)*

Table 7.2.2: Mean accuracy (and SD) for no-go trials

	Congruent	Incongruent	Neutral
Acc	0.99 *(0.004)*	0.96 *(0.04)*	0.97 *(0.02)*

There was no significant correlation between behavioural priming and prime identification using raw scores for percent correct or the absolute difference from chance. In addition running the above analysis with only those nine participants who performed at chance level produced the same results with the exception that the error rates for no-go trials now marginally failed to reach significance. These findings confirm that the priming effects were unrelated to the difference from chance in the prime identification task, and that the priming effects were present in the complete absence of awareness.

Further examination of neutral trials revealed that the side of the presentation of the neutral trial greatly influenced participants' responses. On each neutral trial one of the neutral primes was presented on one side, with a square prime always presented on the other side. Table 7.3 shows the reaction times to go target trials dependent on the congruency between the location of the target and the location of the neutral prime. When the side of the target was congruent with the side of the neutral prime reaction times were significantly faster than when the neutral prime was on the opposite side to the target. Repeated measure ANOVA with neutral prime location as one factor (left/right) and go target location (left/right) revealed a significant main effect of target ($F(1,13)=57.5$, $p<0.001$) as well as a significant interaction between the two factors ($F(1,13)=5.1$, $p<0.05$). Follow up t-tests confirmed that neutral left go left trials were significantly faster than neutral right go left trials ($t(13)=9.4$, $p<0.001$). Similarly neutral right go right trials were significantly faster than neutral left go right trials ($t(13)=5.8$, $p<0.001$).

Table 7.3: Reaction times for neutral trials dependent on side of neutral prime

	Mean	Standard Deviation
Neutral Left- Go Left	**358.1**	**34.6**
Neutral Left- Go Right	**379.9**	**27.1**
Neutral Right- Go Right	**343.9**	**34.5**
Neutral Right- Go Left	**376.3**	**32.8**

EEG Results

ERP analysis was conducted on all 14 participants. Grand average ERPs were formed for each condition with trial numbers averaging between 130 and 142 trials for each condition. Additionally these grand averages were formed of approximately equal numbers of left (average of 67 trials) and right (average of 69 trials) hand responses and contained at least 35 trials per hand.

LRP Analysis

Figure 7.3 shows the grand average LRP for the four go conditions in experiment 3. LRP onset varied as a function of prime congruency ($F(2.3,29.8)=4.7$, $p<0.05$). Subsequent t-tests showed that LRP onset was significantly earlier for congruent go trials in comparison to incongruent ($t(13)=3.6$, $p<0.005$) neutral ($t(13)=2.8$, $p<0.05$) and no-go go trials ($t(13)=3.2$, $p<0.01$). Neutral go, incongruent go, and no-go go LRP were not significantly different from one another. ANOVA revealed no significant amplitude differences between 50 and 100ms and 100 to 150ms after stimulus onset. From 150 to 200ms there was a significant main effect of prime congruency ($F(2.9,37.1)=7.7$, $p<0.001$), reflecting the difference in LRP onset, with congruent go trials showing significantly increased amplitude in comparison to the other three conditions. As is clearly evident from figure 7.3, there is also a significant effect of LRP amplitude from 200 to 300ms ($F(2.2,29.1)=11.8$, $p<0.001$), with neutral trials showing significantly reduced amplitude in comparison to the other three conditions.

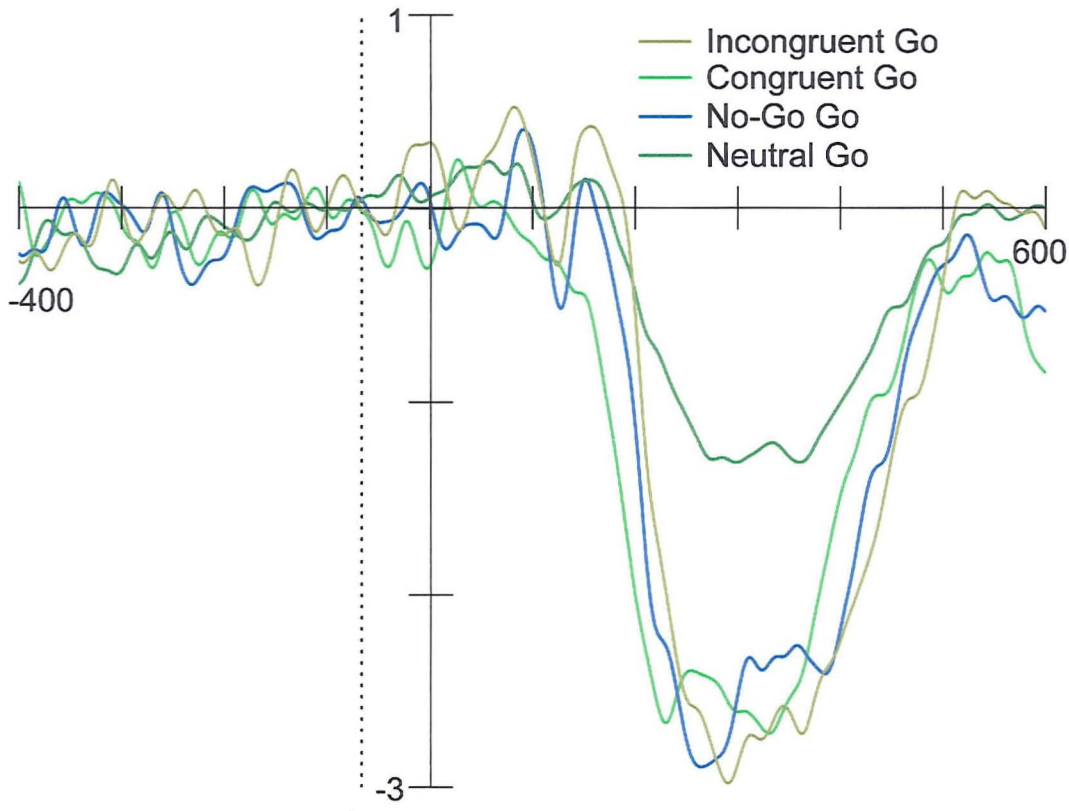

Figure 7.3: LRP for go trials in experiment 3.

Frontal No-go N2 and P3 Analysis

Initial analysis focused on the hypothesised frontocentral modulation of the no-go N2 and P3. Figure 7.4 shows the no-go difference waveforms for each of the three no-go conditions compared to the neutral go condition. It is worth noting that due to the problems described above with the neutral conditions, the more comprehensive analysis described in the next chapter excluded neutral trials. However, the no-go N2 and P3 are still presented here as difference waveforms from neutral go trials to allow easy comparison between experiments. Since all the waveforms are taken as differences from the same neutral go condition the statistical analysis of the difference between congruent and incongruent no-go trials will be identical whether conducted on raw waveforms or difference waveforms.

A clear no-go N2 and P3 are evident for the incongruent no-go condition and the neutral no-go condition, but is absent for the congruent no-go condition. Two separate one-way ANOVAs were conducted at electrode Fz for the three no-go difference waveforms to explore the amplitude of the no-go N2

and P3. The N2 time window (200ms to 300ms) was chosen to encompass the period around the peak latency of the no-go N2. Similarly the P3 time window (375ms to 475ms) was meant to capture any differences in average amplitude of the no-go P3. Each ANOVA (one for each time window) included prime congruency as a repeated measures factor (congruent, incongruent and neutral).

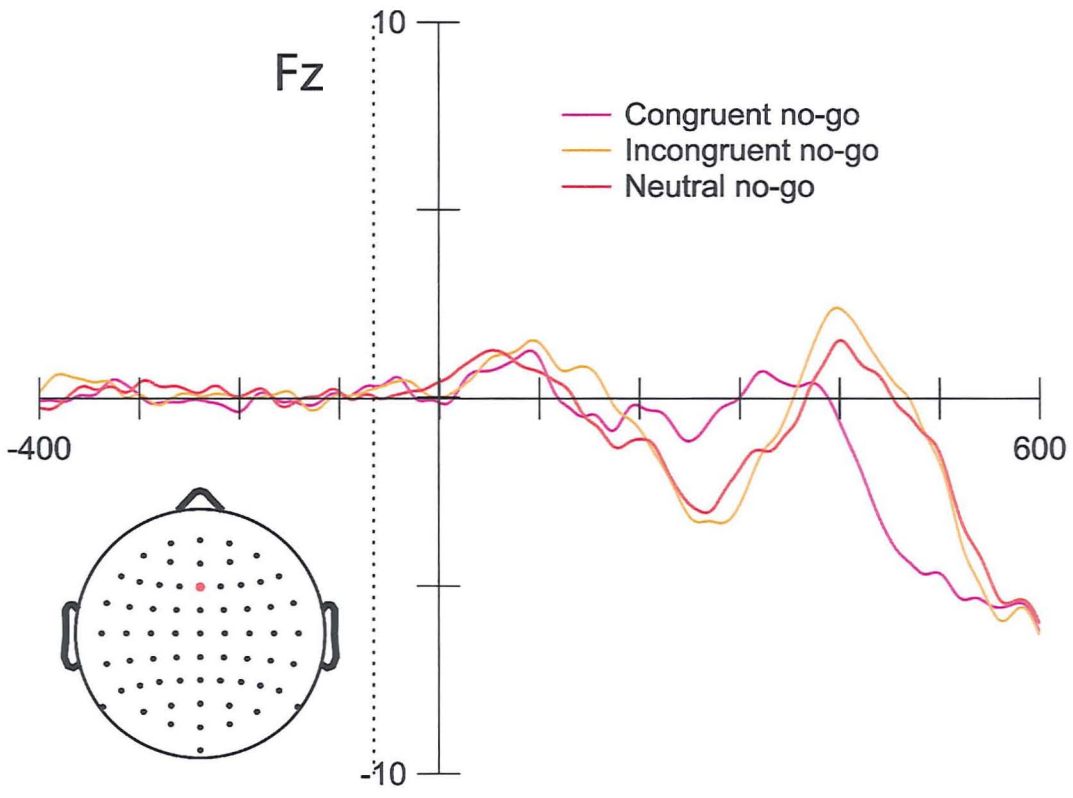

Figure 7.4: Difference ERP waveforms for the four no-go conditions at electrode Fz with respect to target onset. Prime onset at -66ms signified by dotted line.

In the N2 time window there was a significant main effect of prime congruency ($F(1.4,18.1)=10.8$, $p<0.05$). Follow up t-tests confirmed that incongruent no-go trials (m=-2.8; std=1.8) showed significantly more negative amplitude than congruent no-go trials (m=-0.8; std=3.1; $t(13)=3.8$, $p<0.005$). In addition neutral no-go trials showed greater N2 amplitude (m=-2.3; std=2.1) in comparison to incongruent no-go trials ($t(13)=3$, $p<0.05$). Neutral no-go N2 amplitude did not significantly differ from congruent no-go N2 amplitude ($t(15)=1.7$, $p=0.12$).

In the P3 time window there was once again a significant main effect of prime congruency ($F(1.3,17.1)=22.8$, $p<0.001$). Follow up t-tests confirmed that incongruent no-go trials (m=1.1; std=5.2) showed significantly more positive amplitude than congruent no-go trials (m=-2.3; std=5.9; $t(13)=5.2$, $p<0.001$). In addition neutral no-go trials showed greater P3 amplitude (m=0.5; std=5.1) in comparison to incongruent no-go trials ($t(13)=4.7$, $p<0.001$). Neutral no-go P3 amplitude did not significantly differ from congruent no-go N2 amplitude ($t(13)=2$, $p=0.07$). Figure 7.5 shows the topographic distribution of the no-go N2 and P3 components. The N2 initially appears at anterior electrodes before moving to more posterior sites. Similarly, the no-go P3 is maximal at frontocentral electrodes.

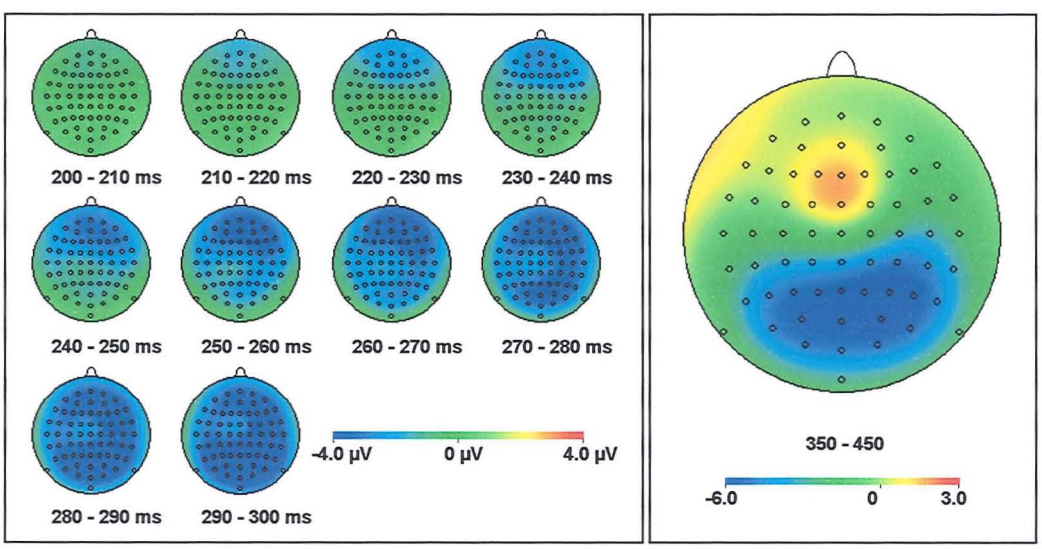

Figure 7.5: N2 and P3 topography for incongruent no-go condition.

Further analysis was conducted to explore the possibility that the magnitude of the N2 and P3 effects was affected by participants' scores on the forced-choice tasks. N2 and P3 priming effects were calculated by subtracting congruent no-go amplitude from incongruent no-go amplitude in the two time windows. There were no significant correlations between either of these measures and performance on the prime identification task, with the greatest correlation reaching $r=0.2$, with an associated p value of 0.4. Furthermore, the above N2 and P3 analysis was repeated using only those nine participants who performed at chance level in the prime identification task resulting in the identical significant effects to those described above. These

results confirm that the modulation of the no-go N2 and P3 were independent of prime identification performance, and that this modulation occurred even in those subjects who failed to identify the primes in the control task.

To further explore the differences of interest, EEG analysis was initially conducted on the same conditions as those utilised in the previous experiments, with the exception that neutral trials were excluded from analysis. Since the current experiment employed a somewhat different procedure the breakdown of different trial types is also different. For example an incongruent go trial in the current experiment signifies a trial where the prime coded for a go response with one hand while the target coded for a go response with the alternative hand. Since in the previous experiments the response hand was fixed in each block, they did not include such a condition. Therefore initial analysis did not include this condition allowing a factorial analysis of prime type and target type to be conducted as in the previous experiments. Prime type was explored on two levels (go and no-go) and target type was also on two levels (go and no-go). These four conditions in this experiment were labelled congruent go, no-go go, congruent no-go and incongruent no-go. Neutral trials were excluded from further analysis due to analysis above suggesting that they represent a number of subgroups of differentially primed conditions, averaging to give what appeared to be a neutral condition. Later analysis will return to explore the difference between congruent and incongruent go trials.

To explore possible early modulation of frontal no-go N2 related activity, ANOVA was conducted at electrode Fz with prime type (go, no-go) and target type (go, no-go) as repeated measure factors. Figure 7.6 shows the raw ERP amplitude at Fz for the four conditions. Visual inspection reveals the presence of a small no-go N2/P3 complex for incongruent no-go trials. In addition an early negativity is also evident for the congruent no-go trials, peaking at around 150ms after stimulus onset (216ms after prime onset), possibly reflecting an early N2 to the prime. ANOVA in a 120 to 180ms time window showed a significant main effect of prime ($F(1,13)=5.99$, $p<0.05$) and a significant main effect of target ($F(1,13)=10.1$, $p<0.01$) but no prime x

target interaction. Subsequent t-tests revealed that congruent no-go trials were significantly more negative that all the other conditions (P<0.01), while there were no other pair-wise differences. This finding suggests that while the prime appeared to successfully elicit an early N2 for congruent no-go trials, this early modulation did not occur in response to the no-go prime for incongruent go trials. Therefore rather than reflecting the direct activation of frontal control/inhibition mechanisms, this early negativity likely reflects an early onsetting target-related negativity on congruent no-go trials.

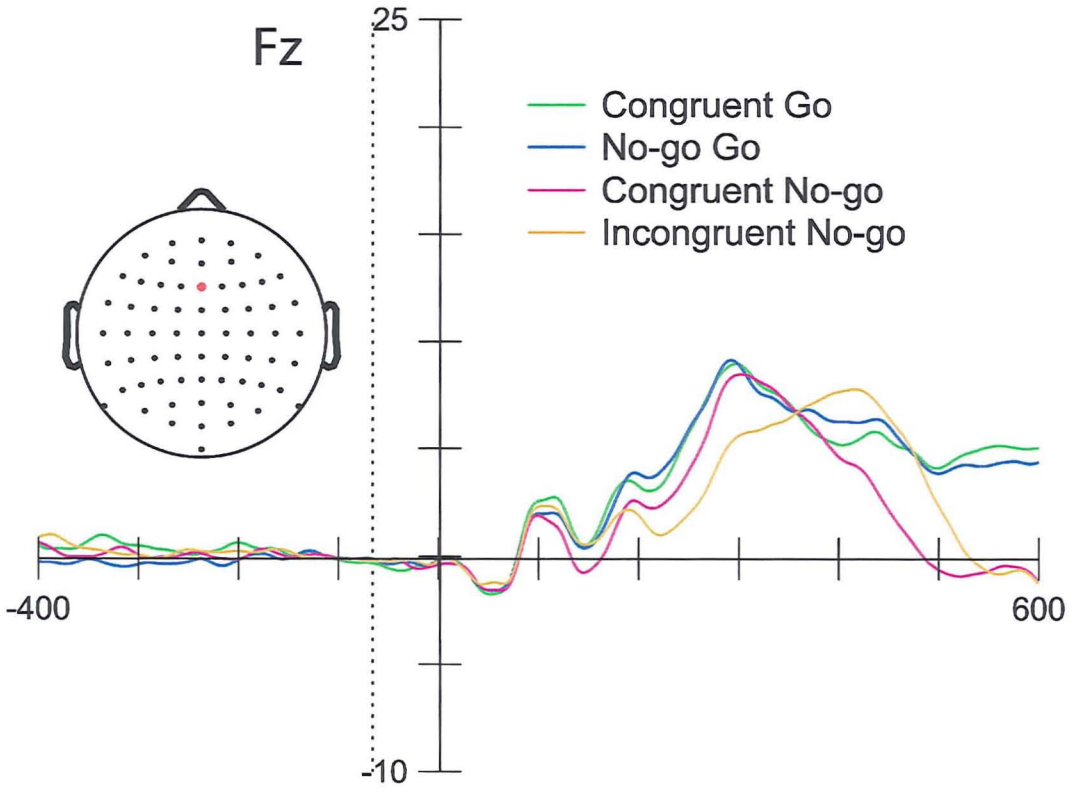

Figure 7.6: ERP waveforms at electrode Fz.

Early visual ERP effects

Figure 7.7 shows the grand average ERPs at electrode Oz for all four conditions. Repeated measure ANOVA with prime type (go, no-go) and target type (go, no-go) revealed no significant effects in the P1 time window (15 to 65ms). In the N1 time window (90 to 125ms), ANOVA revealed a significant prime x target interaction for N1 amplitude ($F(1,13)=9.29$, $p<0.01$). Follow up t-tests confirmed that congruent go trials showed significantly greater N1 amplitude (m=-4.1; std=3.2) in comparison to Incongruent no-go

trials (m=-3.1; std=3.2; t(13)=4.3, p<0.001) and No-go Go trials (m=-3.5; std=3.5 t(13)=2.3, p<0.05).

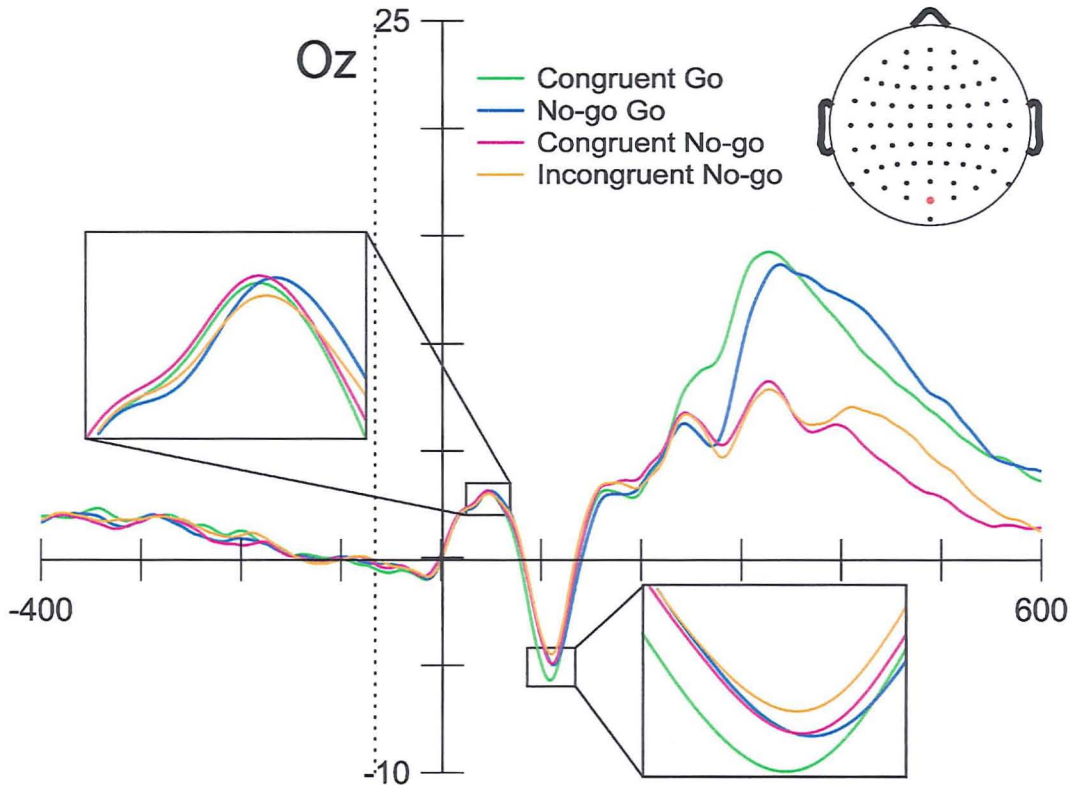

Figure 7.7: ERP waveforms at electrode Oz.

Visual ERPs were explored further at lateral electrode sites. Unlike the previous experiments all participants made the same responses to a particular set of visual stimuli. A left diamond always coded for a left response and a right diamond for a right response. Therefore lateralised ERP effects were explored by calculating O1-O2 and PO7-PO8 difference waveforms for each participant. Visual inspection of figure 7.8 reveals that an early separation is evident based on the side of the go stimulus with left go stimuli associated with increased left hemisphere amplitude and right go stimuli associated with greater right hemisphere amplitude. These differences were confirmed by a significant effect of prime type (left versus right) between 40 and 100ms for both difference waveforms ($F(1,13)=19.7$, $p<0.001$ for O1-O2). Since these differences roughly coincide with the visual N1 at electrode Oz they likely reflect an increased negativity contralateral to the target stimulus.

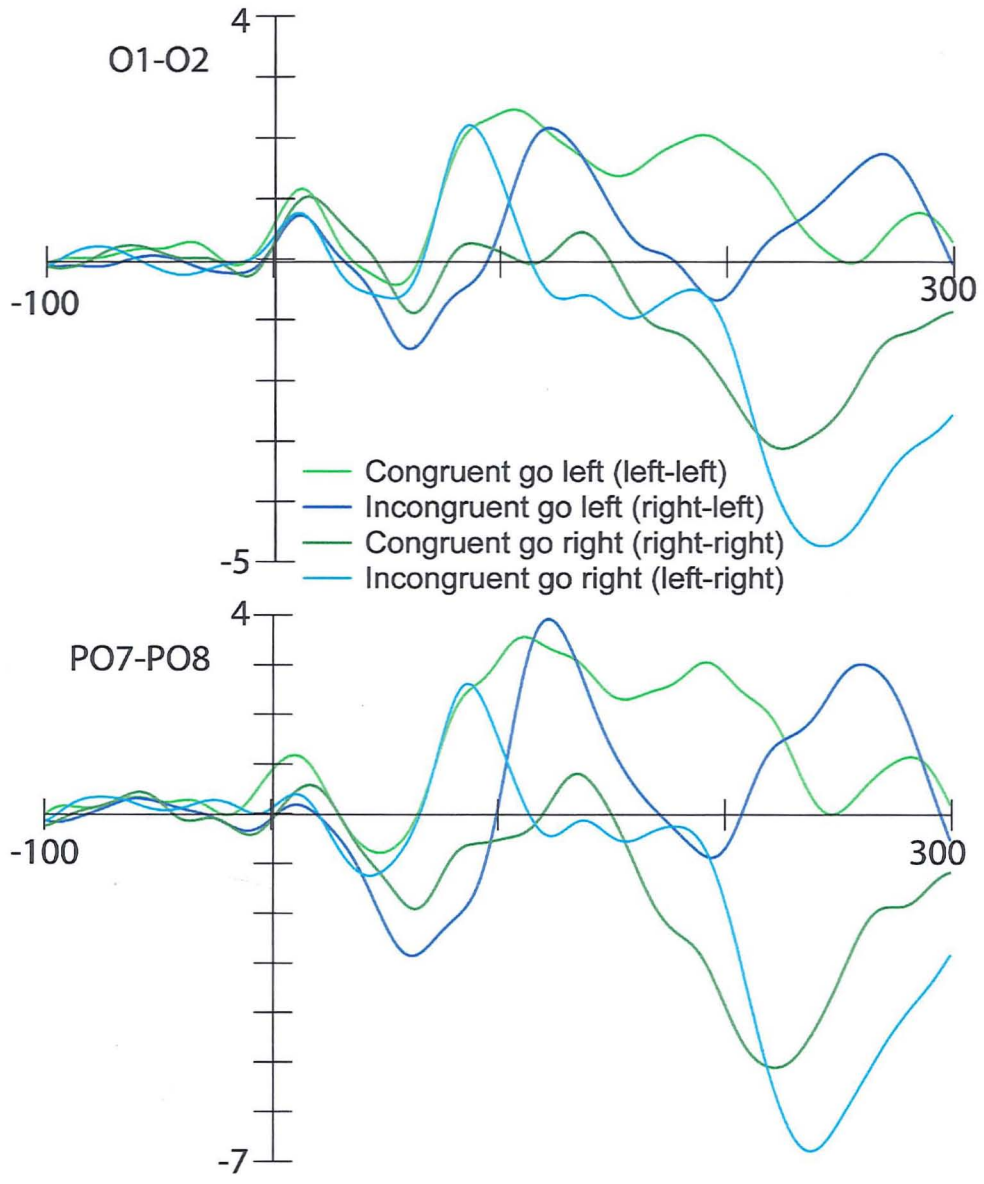

Figure 7.8: O1-O2 and PO7-PO8 difference waveforms for experiment 3.

In the 100 to 210ms time window and 210 to 300ms time windows there were significant main effects of target ($p<0.001$) as well as significant target x prime interactions ($p<0.001$). Visual inspection of figure 7.8 reveals that the lateralised ERP effects were generally much greater in this experiment in comparison to experiments 1 and 2. Importantly, in the previous experiments response hand was varied from one block to the next while the response characteristics remained the same, such that while a left diamond (or arrow) always coded for a go response, the required hand varied from block to block. This manipulation ensured that lateralised visual components

would average out and would not contaminate other ERP components. In the current experiment the lateralised visual effects would also average out in the combined ERPs observed for the go/no-go analysis, however due to the way the LRP is calculated they may contaminate this component. To explore this possibility in more detail LRP type waveforms were calculated at electrodes O1 and O2 in the same way in which the LRP is calculated for the four go conditions using electrodes C3 and C4. Thus the following waveforms were computed for both for the four go conditions:

$$LRP = [Mean(O_2\text{-}O_1)_{\text{left diamond}} + Mean(O_1\text{-}O_2)_{\text{right diamond}}]/2$$

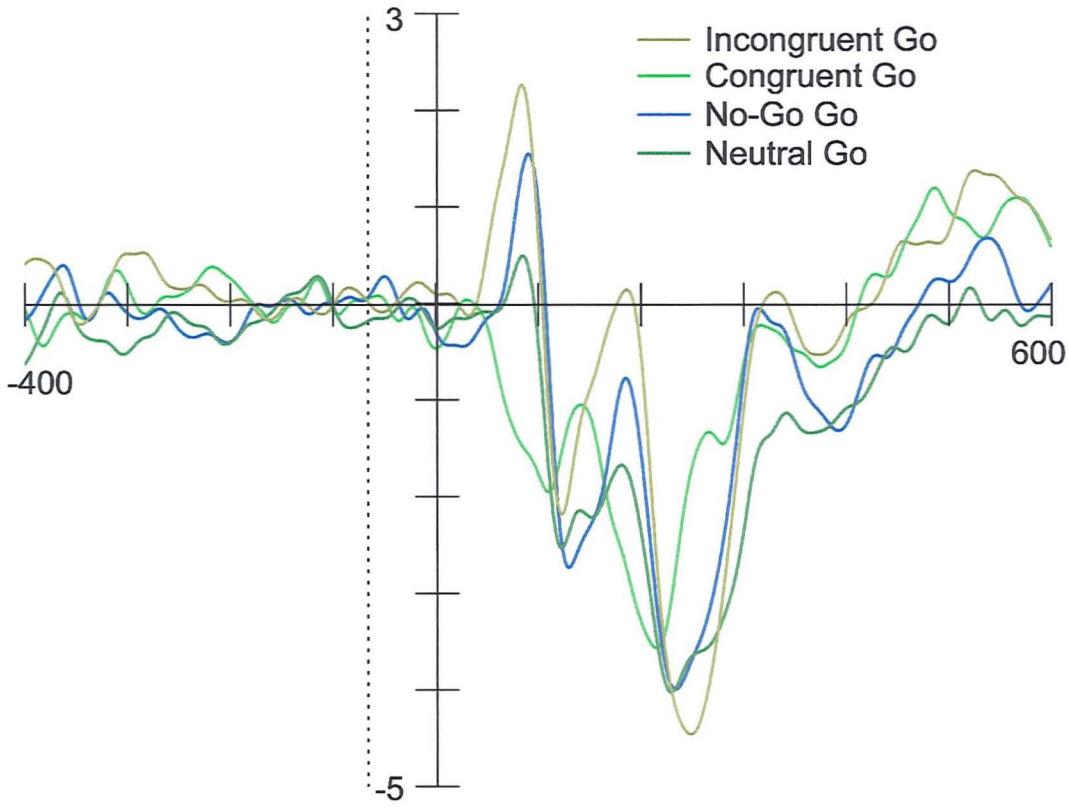

Figure 7.9: Lateralised visual ERP components for experiment 3.

Figure 7.9 shows the LRP type calculation for the four go conditions at electrodes O1 and O2. It is clear from this figure that the LRP at C3 and C4 is likely contaminated by these lateralised visual effects. The large positive deflections for No-Go Go and incongruent go (at around 100ms) and its immediate reversal can clearly be seen in the LRP waveforms (figure7.3). Later components are also evident in figure 7.9 which are of larger amplitude

than even the LRP itself and therefore likely contaminate even target-related LRP effects which begin from around 200ms.

Go/No-go differences

Figure 7.10 shows the raw ERP waveforms for two go and two no-go conditions. Visual inspection of the waveforms reveals that the no-go N2 is less well defined than in experiments 1 and 2. This is likely due to the fact that although 50% of trials were no-go trials, this made up the largest single response set (compared to 25% go left and 25% go right). This means that the default mode of participants was likely to be no-go and therefore it should be easier to withhold the response. Despite this there is still a clear negative deflection for no-go go trials in comparison to all other conditions, with typical frontocentral topography. Similarly, although the no-go P3 is reduced it is clearly still evident on electrodes Fz and Fcz. In addition, a large difference is evident between go and no-go target trials at posterior electrode sites, which also appears to be modulated by prime type for go target trials.

Statistical analysis was conducted with a five-way ANOVA with target type (go, no-go) prime type (go, no-go) hemisphere (left, right) anterior-posterior (Fp, F, FC, C, P, O) and time (120 to 180ms, 180 to 350ms and 350 to 550ms) as repeated measure factors. As in the previous experiment, the early time window (120-180ms after target onset; 186-246ms after prime onset) was selected to explore any early differences in the ERPs associated with the unconscious primes. The second and third time windows were centred on the no-go N2 and no-go P3 respectively. The initial five-way ANOVA showed a significant main effect of hemisphere ($F(1,13)=7.8$, $p<0.05$), with right hemisphere electrodes showing greater ERP average amplitude. Furthermore, there was as a significant hemisphere x target interaction ($F(1,13)=6$, $p<0.05$) and a significant hemisphere x target x time x anterior-posterior interaction ($F(3,38.4)=2.9$, $p<0.05$). There was no significant interaction involving prime and hemisphere, suggesting that while the target type differences may have varied for the lateral electrodes prime differences did not. Further analysis was conducted to explore the nature of these lateralised effects. Three separate four way ANOVAs were conducted,

one for each time window. Each ANOVA had prime type, target type, anterior-posterior and hemisphere as repeated measures factors. In the first time window there was no main effect of hemisphere and no interactions involving hemisphere. In the second time window there was a significant interaction between target and hemisphere ($F(1,13)=6$, $p<0.05$). Follow up contrasts revealed that although significantly increased right hemisphere amplitude was evident for both no-go ($F(1,13)=11.5$, $p<0.01$) and go target trials, the difference was larger for go trials (11.9 versus 7.3). However, since go target amplitude was greater than no-go target amplitude in this time window, this difference likely reflects the additive effect of increased right hemisphere ERP amplitudes.

In the final time window, there was a significant target x hemisphere x anterior-posterior interaction ($F(2.6,33.8)=3.3$, $p<0.05$). Further analysis revealed that there was a significant target x hemisphere interaction at parietal electrodes only ($F(1,13)=15.2$, $p<0.01$). Once again this interaction appeared to reflect an additive effect of increased right sided ERP amplitude over right hemisphere in comparison to left hemisphere. In summary, the hemisphere effects observed in this experiment reflect a general increase in right hemisphere ERP amplitude. This effect was particularly prominent in the second and third time window where it appears to be focused over parietal electrodes where the largest ERP component (the P300) is evident, and is more increased for go target trials than for no-go target trials. Visual inspection of figure 7.10 reveals that despite the evident laterality of ERP amplitudes, the largest ERPs are still observed over central electrodes. Therefore further analysis of go/no-go differences was conducted on the six midline electrodes (Fpz, Fz, FCz, Cz, Pz, and Oz).

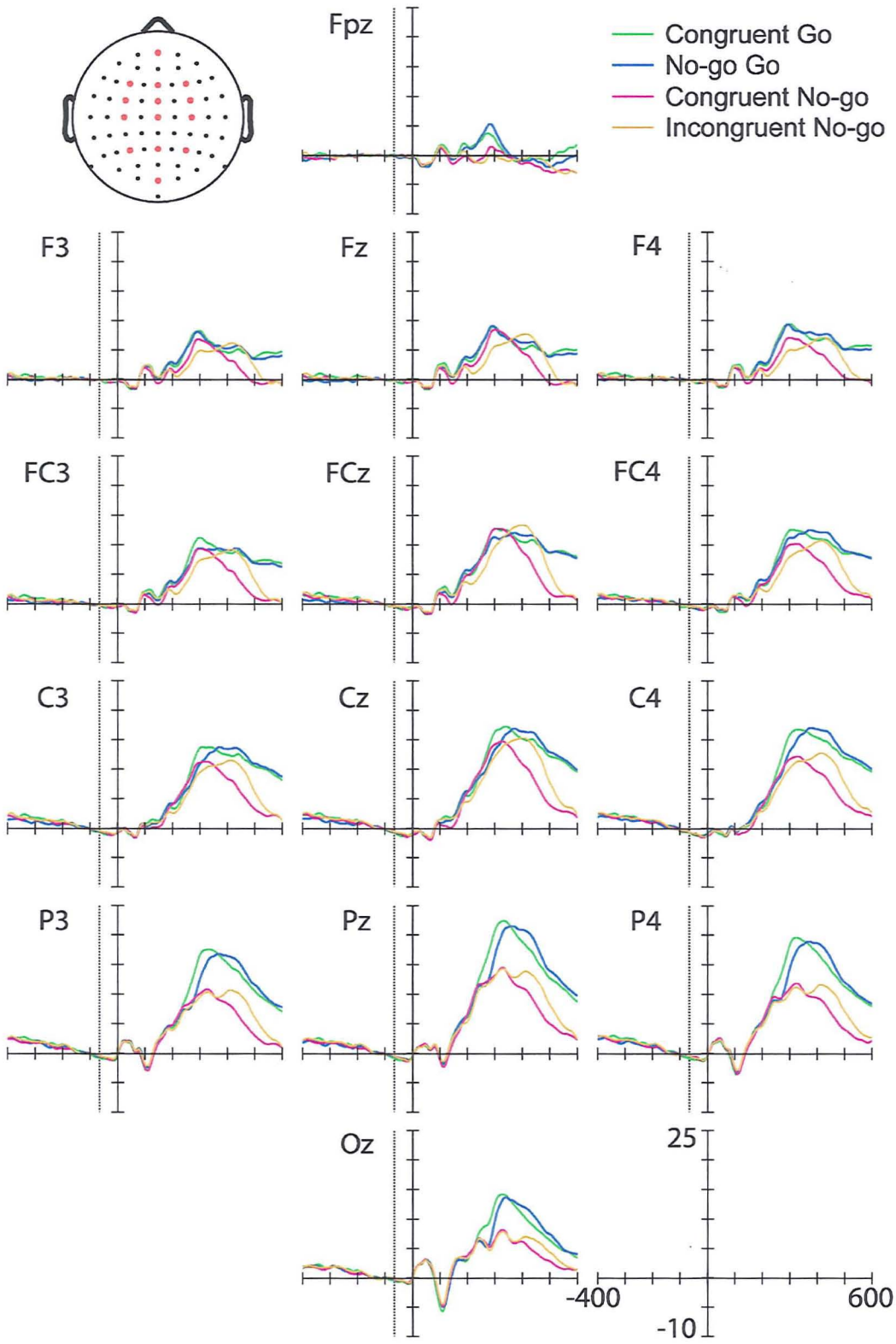

Figure 7.10: Raw ERP waveforms for experiment 2

A Four-way repeated measure ANOVA at central electrodes revealed a significant prime x target x anterior-posterior x time interaction ($F(2.8,36.6)=20.1$, $p<0.001$). Further analysis explored the three way

interactions between prime type, target type and anterior-posterior separately for each time window. In the early time window there was a significant main effect of prime ($F(1,13)=5.2$, $p<0.05$) and a significant main effect of target ($F(1,13)=4.7$, $p=0.05$) but no significant interactions. Contrasts at each electrode location revealed that the main effect of prime was maximal at FCz, where no-go primes showed significantly more negative amplitude in comparison to go primes ($F(1,13)=5.3$, $p<0.05$, uncorrected). The main effect of target was maximal at Pz, with contrasts revealing a significant effect of target (at $p<0.01$) at electrode Pz and Cz such that go target trials were more positive than no-go target trials. Thus the effect of prime type at frontal electrodes likely reflects modulation of frontal early no-go N2 related activity while the more posterior target-related effects probably reflect the onset of the parietal P300 effect.

In the second time window there was a significant main effect of target ($F(1,13)=22.4$, $p<0.001$) as well as a target x prime ($F(1,13)=10.6$, $p<0.01$) interaction and a significant target x prime x anterior-posterior ($F(2.3,30.1)=6.3$, $p<0.01$) interaction. The main effect of target reflects the no-go N2 at anterior sites, and modulation of the P300 at posterior sites. The left panel of figure 7.11 shows that, for go target trials go and no-go primes separate at posterior electrodes only, where go primes have increased amplitude in comparison to no-go primes. T-tests confirmed that go and no-go primes were significantly different at Pz only (at $p<0.001$) and marginally significant at Cz and Oz ($p<0.01$). For no-go target trials there is an opposite modulation of ERP amplitude dependent on prime type, such that go prime trials show more negative amplitude in comparison to no-go prime trials. This modulation is greatest at Fz and FCz where it is significant (at $p<0.003$) and is also marginally significant at Cz ($p<0.02$). As in experiments 1 and 2 this second time window shows a functional dissociation between modulation on go and no-go trials, with no-go trials varying at frontal electrodes (no-go N2 modulation) and go trials varying at parietal electrodes (P300 modulation).

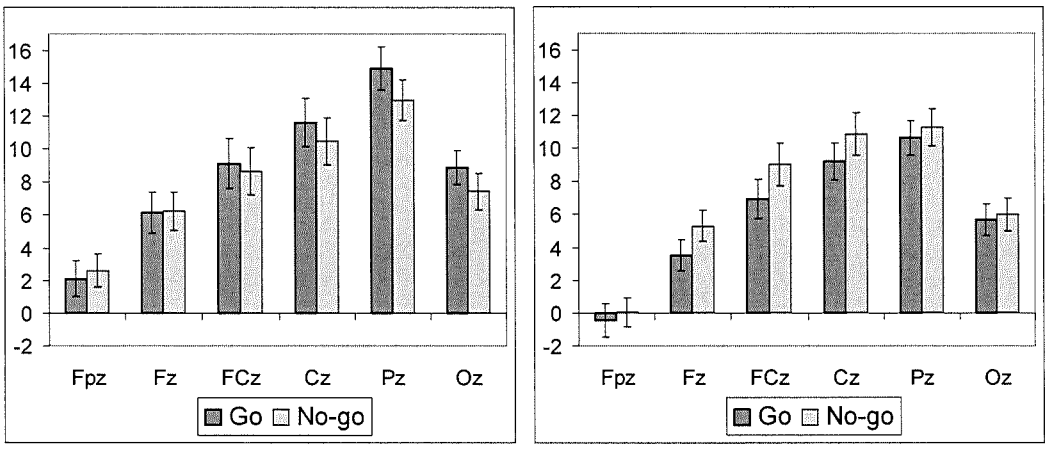

Figure 7.11: Average amplitude for midline electrodes in the second time window dependent on prime type for go targets (left panel) and no-go targets (right panel).

In the third time window there was a significant main effect of prime ($F(1,13)=23.9$, $p<0.001$) and a main effect of target ($F(1,13)=11.3$, $p<0.01$). In addition, there was a significant prime x target interaction ($F(1,13)=54.4$, $p<0.001$) and a significant prime x target x anterior-posterior interaction ($F(2.8,37)=20.7$, $p<0.001$). The main effect of target was caused more positive amplitude for go target trials in comparison to no-go target trials. Figure 7.12 shows the average ERP amplitude in the final time window dependent on target type. For go target trials there was a significant difference between go and no-go prime trials at electrodes Pz and Oz (at $p<0.001$) and a moderately significant effect at Cz (at $p<0.01$). This anterior modulation is in the opposite direction to that observed in the second time window. This reversal appears to reflect the earlier onset of the P300 condition for congruent go trials, with the second time window coinciding with the rising bank of this component and the third time window centered in the falling bank. For no-go target trials, no-go prime trials showed greater positive amplitude than go trials. This effect likely reflects the no-go P3. Modulation of this component was observed over Fz, FCz, Cz and Pz (significantly different at $p<0.001$).

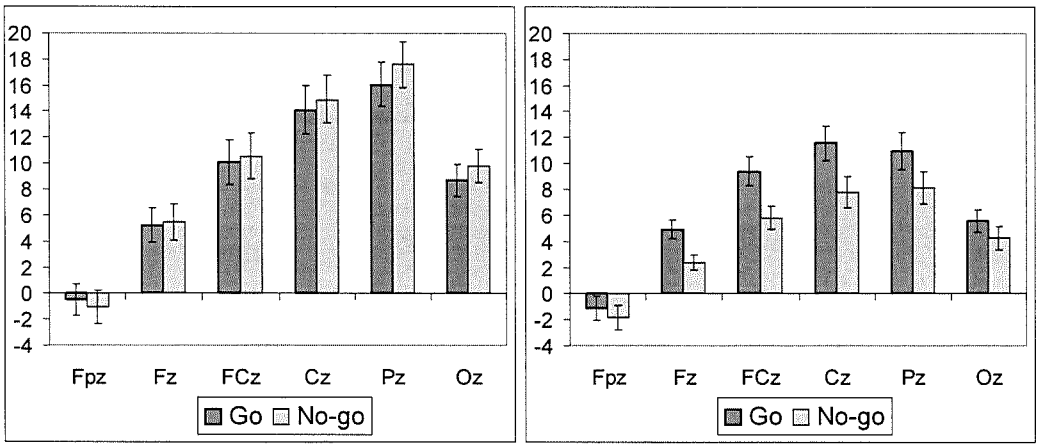

Figure 7.12: Average amplitude for midline electrodes in the third time window dependent on prime type for go targets (left panel) and no-go targets (right panel).

LRP and go/no-go differences

Since the go/no-go differences were explored by averaging together left and right response trials those effects should be uncontaminated by the visual and motor asymmetries that were evident in this experiment. To confirm that the effects remained when the left and right responses were averaged separately, ERPs were averaged separately for left and right conditions. Figure 7.13 shows these grand average ERPs for electrodes FC3, FC4 and FCz as well as P3, P4 and Pz for congruent go trials, no-go go trials and congruent no-go trials. These conditions were selected to highlight the early differences apparent at electrodes FCz for the congruent no-go condition. The two boxes show the most important areas for the hypothesis that frontal no-go effects were elicited by the prime, where the early N2 occurs on congruent no-go trials. Over FC3 both FC4 congruent no-go conditions are consistently more negative than all other conditions.

The earlier analysis of the early go/no-go differences highlighted that while there was a negativity associated with congruent no-go trials; this was not the case for no-go go trials. Further examination of the light blue and dark blue lines in figure 7.13 reveals that there is no consistent early asymmetry for no-go go trials. This is to be expected because the prime codes for a no-go response and therefore no motor activation should occur. For congruent go trials however, the congruent go left condition shows consistently greater

negativity over right sided electrodes in the early time window. This effect then persists throughout the epoch.

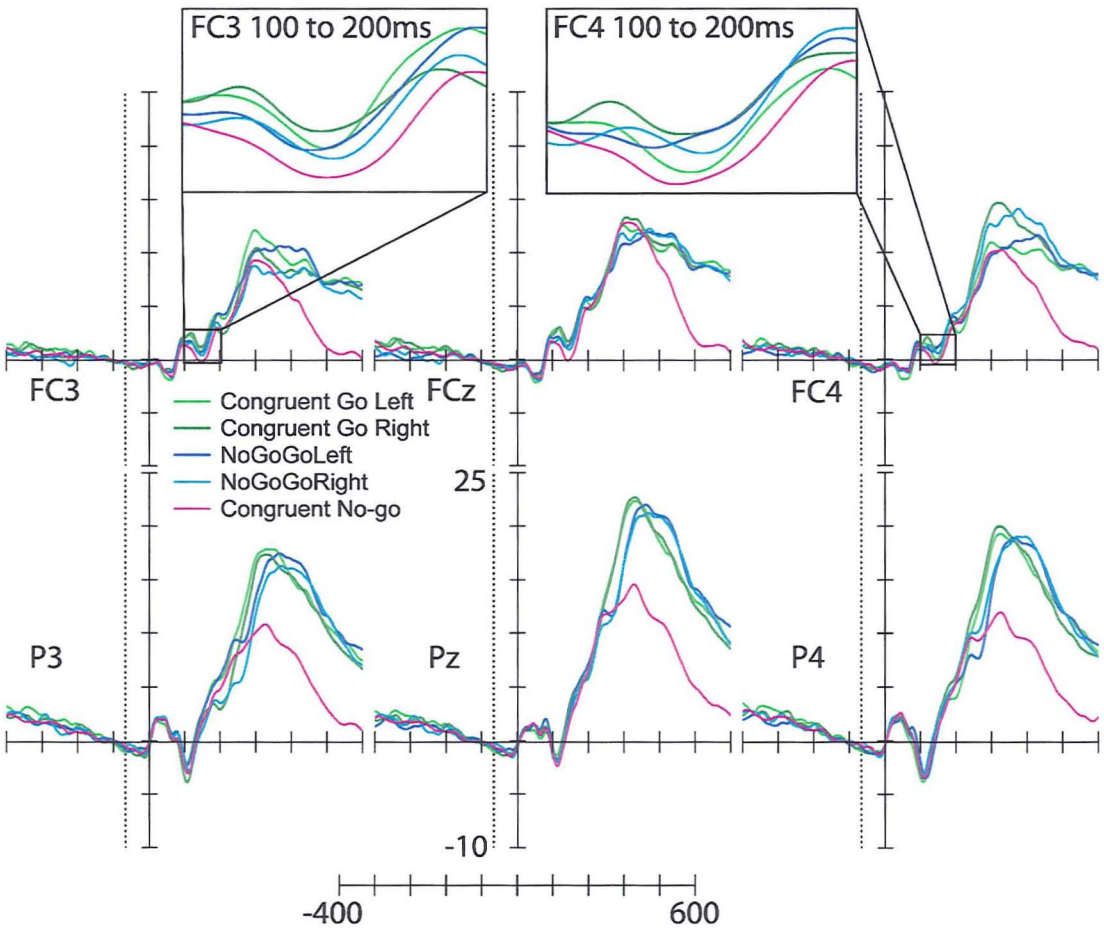

Figure 7.13: Effects of lateralised components on early go/no-go differences in experiment 3.

It is important to note that in this experiment, where forward compatibility effects are observed the motor related effects occur in the opposite direction to the go/no-go differences. For example any contamination at central sites from the early contralateral negativity in response to congruent go left trials would serve to decrease ERP amplitude for this condition, thus reducing the go/no-go differences not artificially creating them. The same would also be true for the non lateralised motor activity (the readiness potential), which is also negative going and would therefore also reduce or remove very small go/no-go differences. In the current experiment such motor related activity in response to go primes could act to abolish a frontocentral negativity for no-go go trials. The presence of an early negativity for congruent no-go trials however, suggests that the no-go related negativity for that condition was

large enough to overcome these competing factors, and therefore the same should be expected if an N2 like effect had been present for no-go go trials.

In addition to the frontal prime-related modulations, figure 7.13 shows that the target-related ERP effect at Pz, namely the early onset P300 for congruent go trials, and the increased amplitude on go trials, were both evident over the left and right hemisphere.

The overlap between response preparation negativities and frontal N2 related effects is again highlighted in figure 7.14. While the go target trials show large motor related asymmetries, onsetting around 100ms after stimulus onset, no-go target trials do not show similar asymmetries. Again, since these motor asymmetries result in an increased negativity contralateral to response the response hand, any spreading of these effects to central electrodes would result in a reduction of the no-go N2. However, since the no-go P3 is associated with increased negativity for no-go trials it is possible that this difference is partly created by increased motor negativity (see chapters 2 and 10 for further discussion on this issue).

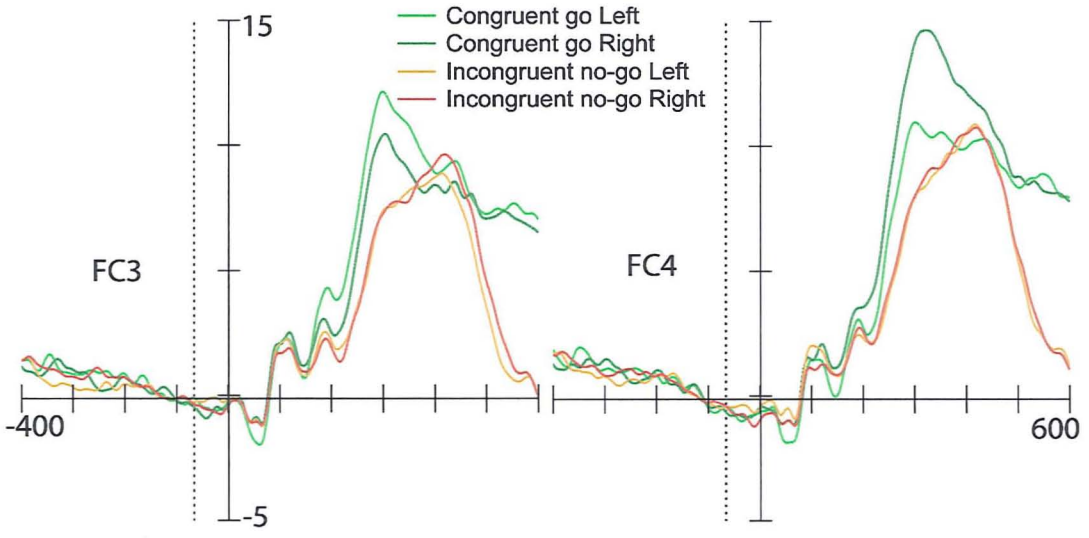

Figure 7.14: Effects of lateralised components on target-related go/no-go differences in experiment 3.

Congruent go versus incongruent go trials

Finally, to explore the possible presence of a parietal N2 for incongruent go trials, ERP grand averages were formed for congruent go, incongruent go and no-go go conditions. Figure 7.15 shows that while there is no frontal modulation for go trials there is a parietal modulation, with incongruent go trials showing significantly reduced amplitude from 250 to 370ms after target onset ($t(13)=3.86$, $p<0.002$). ANOVA at P3 ($F(1,13)=11.7$, $p<0.01$) and P4 ($F(1,13)=11.4$, $p<0.01$) with prime congruency (congruent, incongruent) and response hand (left, right) as repeated measures factors further revealed that a main effect of prime congruency was evident at both lateral electrodes.

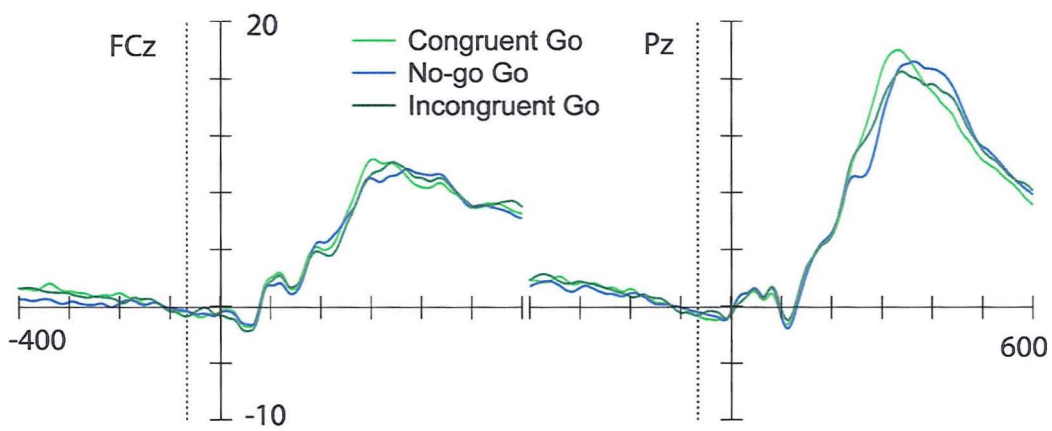

Figure 7.15: Effects of lateralised components on target-related go/no-go differences in experiment 2.

Discussion

This experiment aimed to overcome the problems with the neutral primes in experiment 2 and also to explore go/no-go inhibition and selective inhibition in the same task. In particular, it aimed to show that while frontal inhibition/control mechanisms are involved in unconscious modulation of go/no-go differences, they are not associated with the resolution of unconscious conflict on incongruent go trials. While there is evidence to suggest that conflict between response alternatives can modulate these mechanisms (Van 't Ent, 2002), Leuthold and Kopp (1999) have shown that the N2 exhibits a parietal rather than frontal topography when conflict between two response alternatives is unconscious.

Behavioural effects followed the predicted direction with fastest reaction times for congruent go trials, and slowest for incongruent go and no-go go trials. Importantly, neutral prime trials were significantly different to both congruent and no-go prime trials. However, further analysis of the neutral go trials revealed that the side of the neutral prime greatly influence reaction times. More specifically, when the neutral prime was on the same side as a subsequent go target, reaction times were significant faster than when the prime was on the opposite side. A likely explanation is that since participants were required to react on the side where a diamond was present, they will be looking out for diagonal lines in the display. Although the neutral primes contained two features of each of the primes, the requirement of the task to respond to diagonals (diamonds) and not respond to straight lines would likely give the diagonals more influence. Due to this problem with the neutral primes, they were excluded from subsequent ERP analysis.

Frontal no-go N2 and P3 effects were consistent with previous experiments which showed reduced N2 and P3 for primed no-go responses. This finding once again suggests that frontal control mechanisms are modulated by unconscious primes. Moreover, an early negativity was observed for congruent no-go trials peaking around 150ms after stimulus onset. This result suggests that in this condition the N2 onset earlier in time as a result of the no-go prime. However, unlike in previous experiments this effect was limited to the congruent no-go condition such that no early negativity was observed following a no-go prime, when the subsequent target coded for a go response. The more comprehensive analysis of go/no-go differences highlighted the same early separation at frontal electrodes dependent on prime type, with no-go prime trials showing significantly more negative amplitude than go prime trials. However, it is important to note that in this analysis a target-related effect was also evident in this early time window in this experiment, which although having a more parietal maximum was also evident at frontocentral electrodes. This overlapping target-related activity suggests that the prime-related effects observed at frontal electrodes was not purely related to the prime, but also influenced by the target. These findings together suggest that the unconscious primes were not able to

directly activate frontal inhibition/control mechanisms in the current experiment.

Analysis of visual ERP effects revealed a significant prime x target interaction at electrode Oz in the N1 time window. This is similar to the effects observed in experiment 2 where congruent trials showed an increased and perhaps earlier N1 component. In addition to this congruency effect at Oz visual ERP effects were also seen to be lateralised dependent on the nature of the physical features of the unconscious prime. As in experiment 2, increased P1 and N1 responses were observed contralateral to the side of the diamond stimulus, suggesting some kind of visual detection of the stimulus features. Given the lack of any early prime-related modulation at frontal electrodes, the presence of these visual effects provides for the possibility that the modulation of target-related N2/P3 was caused by perceptual priming of the target and therefore earlier and easier categorisation of the target. For example, the presence of a diamond prime might attract participants' attention to that location, which would then result in faster classification of a diamond in the same location. This possible alternative explanation was discounted in previous experiments due to the presence of early frontal prime-related ERP modulation, which could not be accounted for by faster or better classification of the target stimuli. The absence of such an effect in the current experiment means it is impossible to rule out this perceptual/attentional account of the observed N2/P3 modulations.

The presence of lateralised visual components in the current experiment also led to complication with interpretation of LRP activity. Importantly, although previous experiments also used somewhat lateralised visual stimuli, they were counterbalanced across conditions to ensure that no such contamination could occur. In the current experiment however, the change in stimulus-response parameters meant that stimuli in different visual fields consistently coded for one response or the other. This contamination of the LRP in the current experiment makes interpretation of the lateralised motor effects impossible. One method for avoiding this type of contamination of

LRPs would be to present stimuli in central locations, or above and below the midline (as in Leuthold and Kopp, 1999) as this would prevent the appearance of lateralised visual effects. Although, these lateralised visual effects were seen to contaminate LRP waveforms they did not affect the analysis of go/no-go differences. Therefore they do not compromise the finding that no-go N2 and P3 components were modulated by the unconscious prime.

Inspection of ERPs generated for left and right hand responses separately confirmed that the N2 and P3 effects were present over both hemisphere and for both left and right hand response trials. This confirmed that the complications with the lateralised visual and motor effects did not influence the analysis of go/no-go differences. Furthermore, although motor related asymmetries were clearly evident in the individual hand waveforms, they were seen to be acting in the opposite direction to the N2 effects. Since motor readiness potentials and lateralised readiness potentials (LRP) are both negative going potentials, they will show increased negativity for go trials in comparison to no-go trials. This is the opposite of the effects found in the N2 time window where no-go trials are in fact more negative than go trials. Praamstraa and Seiss (2005) found that a pseudo N2 was observed in the negative compatibility effect for congruent no-go trials caused by averaging together left and right hand responses. This led to the inclusion of a similar check in the current experiments to determine whether the N2 effects were indeed genuine N2 effects and not projections of motor related effects. Importantly, this effect only occurred in Praamstra and Seiss's (2005) experiment because of the reversal of the initial motor priming effects. As described above, any priming of a motor response in the current experiment, and indeed in any experiment with positive compatibility, would in fact work in the opposite direction to N2 effects and would therefore reduce them, or even remove them and not, as reported by Pramstraa and Seiss (2005), create spurious N2 effects.

The comprehensive analysis of go/no-go differences also revealed a number of significantly lateralised effects, with right hemisphere electrodes showing

generally increased amplitude in comparison to left hemisphere electrodes. This effect is clearly visible on inspection of the ERP waveforms in figure 7.9. However, despite a number of significant interactions occurring between hemisphere and target type, these effects appeared to be additive such that when go target trials showed increased amplitude in comparison to no-go target trials this effect appeared to be magnified by the increased general amplitude over right sided electrodes. This suggests that rather than the components showing a significantly right sided distribution, there appeared to be significantly increased right sided ERP amplitude. Reviewing the ERP waveforms closely for experiments 1 and 2 reveals a similar pattern in each of these experiments (although to a lesser degree); therefore the significantly increased right hemisphere activity in the current experiment likely reflects a generic process for the particular task. One possible explanation for this observation is that since the task involves processing and identification of objects, this might more actively engage right hemisphere processing. Evidence in support of this interpretation comes from the finding that increased right hemisphere magnetoencephalogram (MEG) activity appears to be modulated by successful generation of object representations (Schweinberger, Kaufmann, Moratti, Keil, & Burton, 2007). Similarly, Foxe, McCourt and Javitt (2003) showed a right sided ERP bias for a line bisection task, suggesting that control of visuospatial attention, in particular with reference to objects, manifests in increased right hemisphere ERP amplitude. This suggests that the increased right hemisphere activity observed in the current experiment, and to a lesser degree in the previous experiments, is associated with right hemisphere spatial attention and object representation processes.

As in previous experiments, modulation of go trials in the N2 time window was maximal at posterior and not anterior electrode locations. Interestingly a similar parietal modulation was also observed in the current experiment between congruent and incongruent go trials. Importantly, in the current experiment incongruent go trials reflected trials where the prime coded for a go response with one hand and the target code for a response with the other hand (e.g. left go followed by right go). This condition is analogous to the

incongruent go condition in Leuthold and Kopp (1999). They found a similar parietal modulation which they interpreted as a parietal N2. However the presence of this component alongside a genuine frontal N2 confirms that it likely reflects an independent process. The most likely explanation for this parietal modulation is that it reflects modulation of the parietal P300 or P3b component. As described in the chapter 2 there is much debate as to the exact functional significance and cortical generator of this ERP component, which is measured in a wide variety of different tasks. However, it seems likely that this component reflects the interface of perception and action, where a decision is reached about how to respond to a stimulus is reached (Verleger et al., 2005). The modulation of this component for go trials in the current experiment is consistent with such an interpretation, as the incongruent or no-go prime increases both stimulus evaluation time and reaction time to the target, both processes which are reflected in the component. This issue is discussed more extensively in response to all the current experiments in the general discussion (chapter 10).

Conclusions

This experiment replicated the unconscious modulation of go/no-go ERP differences observed in experiments 1 and 2. More specifically, no-go N2 and P3 amplitude were found to vary as a function of the unconscious masked prime. In addition to this target-related modulation, an early frontal negativity was observed for congruent no-go trials. Unlike in previous experiments however, this modulation was not entirely determined by the no-go prime as this negativity was largely absent for no-go go trials. Therefore, the results from the current experiment cannot rule out the possibility that the N2 and P3 priming effects were caused by earlier or more successful classification of no-go targets when followed by a no-go prime. Moreover, the current experiment showed that unconscious conflict between two response alternatives does not exhibit a frontal no-go N2, but rather is reflected in modulation of a parietal P300 component. However, examination of these effects alongside LRP modulation was not possible due to contamination of this component from lateralised visual ERP effects. A further complication with the neutral primes also meant that only differences

between congruent and incongruent (and no-go) primes could be explored in the current experiment. In summary, the current experiment replicated the modulation of the target-related no-go N2 and P3 amplitude as a function of the unconscious prime observed in the previous experiments. Importantly, this modulation was maximal over frontal electrode sites, while an additional effect was observed at posterior sites for incongruent go trials. This finding suggests that while the frontal effects reflect modulation of frontal inhibition/control mechanisms, the parietal effects are related to the P300 elicited on go trials.

Chapter 8

Experiment 4 - Unconscious facilitation of no-go N2 and P3 ERP components.

Introduction

Experiment 4 aimed to further extend the conclusion of the first three experiments that unconscious primes could facilitate the no-go response as measured by the no-go N2 and P3. In experiments 1 to 3 this facilitation was manifested in decreased N2 and P3 amplitude for congruent no-go trials in comparison to incongruent no-go trials. However, only experiment 1 allowed direct comparison of trials with a neutral prime with congruent and incongruent trials. Given that this effect occurred in an experiment where congruent primes impeded rather than facilitated responses it is worthy of replication. The comparison between congruent and neutral no-go trials is particularly important since any modulation of no-go N2 and P3 amplitude between congruent and incongruent trials might reflect motor priming rather than priming of a no-go response. For example, when a go prime is presented this may initiate motor response preparation which then requires inhibition. When a no-go prime is presented, since no unconscious motor activation would be expected the no-go responses will be smaller in comparison. Crucially however, without a neutral prime it is not clear whether the no-go N2/P3 response is attenuated in this condition compared to baseline.

The observation in the previous experiments that the subliminal primes were able to, to differing degrees, directly initiate early frontal ERP effects associated with inhibition/control mechanisms, strongly supports the assumption that the no-go response is indeed facilitated by a no-go prime. Nonetheless, this conclusion would be further supported by showing facilitation of a no-go response in comparison to a neutral baseline. The current experiment reverted back to the simple go/no-go procedure employed in the first two experiments so as to not introduce the additional

confounds observed in experiment 3. In the current experiment participants were required to make a response to a single central arrow pointing either to the left or to the right. Unbeknownst to the participants a prime arrow was presented that was either congruent incongruent or neutral to the target. Congruent arrows pointed in the same direction as the target while incongruent arrows pointed in the opposite direction. Neutral primes consisted of arrows pointing either upwards or downwards. The primes were presented slightly above, below, left or right of fixation. Since the target was in the centre of the screen; all primes would be equidistant from the target thus ensuring that they would have equal allocation of attention.

Hypotheses

If no-go N2 facilitation occurs as a result of priming of the no-go response then N2 and P3 amplitude should be reduced for congruent no-go conditions in comparison to incongruent and neutral no-go conditions. Additionally, if these processes are directly initiated by the unconscious primes then the ERPs should show some early frontal modulation dependent on prime type.

Method

Participants

Twenty-one participants (11 male and 10 female) were recruited by means of poster advertisement. All participants were right handed and had normal or corrected to normal vision. The mean age of participants was 26 years and 0 months, with a range of 19 to 38 years. Participants were reimbursed £15 in compensation for their time.

Experimental Procedure

All participants completed a single experimental session lasting approximately two hours. The participants completed 14 blocks of the go/no-go task followed by three blocks of the prime identification task. Each go/no-go block contained 72 trials presented in a random order. The 14 experimental blocks were preceded by two practice blocks of 36 trials. Participants were required to respond if a central arrow was pointing in one

direction and to refrain from responding to arrows in the opposite direction. Half the participants were instructed to press a button in response to left pointing arrow and half were instructed to respond to right pointing arrows. The response hand was varied from one block to the next. The participants were informed that they had a time limit of 450 ms to respond to the go stimuli and that they should react as quickly as possible without sacrificing accuracy. Participants were given visual feedback immediately after the 450ms response window for correct responses, incorrect responses and non-response as well as false alarms and incorrect non-responses.

Participants were informed that a black square with a white diamond centre would be presented prior to the target stimulus. They were informed that this would help to guide their attention to the centre of the screen. Unbeknownst to the participants a brief prime was also presented prior to the diamond shape (see figure 8.1). The prime consisted of a faint arrow pointing either to the left or the right, or up or down. The primes fit exactly into the contour of the white diamond shape for maximum metacontrast masking. One third of the primes were congruent with the respect to the target stimulus and one third were incongruent. A third of trials were neutral to the target stimulus. The neutral trials either pointed up or down, with half the participants being presented with up neutral primes and half with down neutral primes. The allocation of target stimuli and neutral primes was counterbalanced across participants.

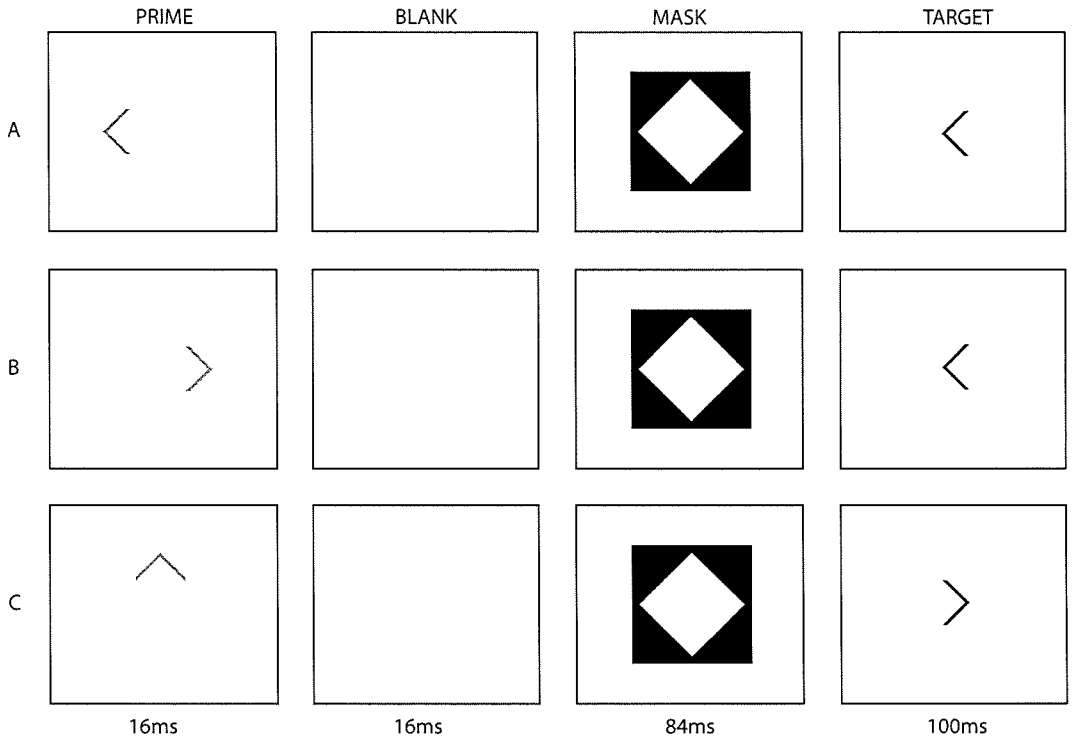

Figure 8.1: Stimuli for experiment 4. A congruent (A), incongruent (B) and neutral (C) trial.

The stimuli used for experiment 4 are shown in figure 8.1. Left and right pointing prime and target stimuli measure 0.8cm across and 1.6cm from top to bottom, with the dimensions reversed for up and down pointing arrows. Primes were presented with the outer edge 1.75cm from the centre of the screen. The outer contours of the mask measured 4cm, with the internal diamond measuring 3.5cm. The target arrow was presented in the centre of the screen such that an approximately equal number of pixels were presented marginally either side of fixation (78 pixels on the side of the arrowhead and 82 pixels on the side of the flankers)

Each trial began with a large central fixation for 200 ms which acted as a warning signal to participants that the next trial had begun. A smaller fixation cross was then presented for 800ms followed by the prime (16ms), a blank screen (16ms), the mask (84ms) and the target (100ms). Following the 450ms response window participants were given visual feedback which remained on the screen for 500ms. Finally a blink pause was presented for 800ms followed by a blank screen for a randomly selected interval between 200ms and 800ms.

Following the go/no-go task the participants were asked the same three questions as in experiment 2 and 3 and were then shown the sequence in slow motion. After seeing the slowed sequence participants were asked if they recognised having seen any of the primes during the go/no-go task. The prime identification task consisted of one practice block and three experimental blocks of 96 trials. One half of trials contained a right pointing prime arrow and the other half contained a left pointing prime arrow. No neutral primes were presented in this task and no target stimulus was presented. Participants were informed that they should press the far left button for left arrow primes and the far right button for right pointing primes. Participants had no time limit to respond, and received feedback at the end of each trial. Finally participants were asked to report whether the felt they were able to see the masked primes during the detection task.

Behavioural Results

Awareness of Primes

Table 8.1 shows the participants' responses to the four questions regarding the visibility of the primes. None of the 21 subjects reported having seen anything appear before the diamond shape. In fact four participants did not even notice the diamond shapes despite being informed of its presence. Whilst four subjects reported that they might have seen something flash, only one subject recognised having seen the prime when shown the slow motion sequence, and the same subject reported seeing the primes in the forced-choice task.

Table 8.1: Summary of responses to Questions 1 to 5

	Notice?	What?	Flash?	Recognise?	See?
NO	21	21	17	20	20
YES	0	0	4	1	1

Overall performance on the forced-choice task was not significantly different from the 50% chance level (t (20) = 1.385, p = 0.181). However, one subject (the same subject who reported having seen the primes) achieved 75% accuracy, while one other participant achieved close to 60%. These two participants were classed as possibly having some residual awareness of the prime. Initial analysis included these two participants, with all priming effects then correlated with prime identification performance. In addition, the reliability of the effects was assessed when these two participants were excluded. The remaining participants achieved an average accuracy of 50.3% which did not differ from 50% (t (18) = 0.43: p=0.68). Participants d' values did also not differ from chance (t (18) = 0.44, p=0.68).

Priming

The influence of the masked primes during the go/no-go task was assessed in the nineteen participants who showed chance recognition of the primes in the forced-choice task. Repeated measures ANOVA with prime congruency as a repeated measures factor showed a highly significant effect on prime-stimulus congruency for reaction times ($F(1.9,37.8)=25.2$, $p<0.001$) and accuracy ($F(1.9,37.3)=16.1$, $p<0.001$) on go trials. Similarly a significant main effect of prime-stimulus congruency was evident for accuracy on no-go trials ($F = 13.99$, $p<0.001$).

Table 8.2.1: Mean Reaction times and accuracy (and Standard Deviations) for go trials

	Congruent	Incongruent	Neutral
RT	342 *(15)*	354 *(13)*	347 *(15)*
Acc	0.91 *(0.06)*	0.87 *(0.07)*	0.88 *(0.07)*

Table 8.2.2: Mean accuracy (and SD) for no-go trials

	Congruent	Incongruent	Neutral
Acc	0.95 *(0.04)*	0.91 *(0.06)*	0.94 *(0.05)*

Subsequent t-tests confirmed that reaction times were significantly different between congruent and incongruent go trials ($t(20)=6.3$, $p<0.001$), congruent and neutral go trials ($t(20)=3.1$, $p<0.01$) and incongruent and neutral trials

(t(20)=4.5, p<0.001). For error rates there was a significant difference between congruent and incongruent go trials (t(20)=5.9, p<0.001) and congruent and neutral go trials (t(20)=3.9, p<0.001). No-go error rates were also significantly different for congruent and incongruent no-go trials (t(20)=-5, p<0.001) as well as congruent and neutral no-go trials (t(20)=3.5, p<0.01)

There was no significant correlation between behavioural priming and prime identification using raw scores for percent correct or the absolute difference from chance. Furthermore, running the above analysis excluding those two participants who showed possible residual awareness of the primes produced the same results as outlined above. This confirms that the priming effects were unrelated to the prime identification performance and that the priming effects were present when objective measures of awareness did not differ from zero.

EEG Results

One participant was excluded from EEG analysis due to a hardware failure during recording, leaving a total of 20 participants. ERPs were formed from an average of between 118 and 130 trials for each condition (with a minimum of 60 trials), made up of equal numbers of left and right hand response trials (approximately 62 per hand).

LRP Analysis

Figure 8.2 shows the LRP for the six conditions. Onset analysis was conducted in the three go conditions using the 50% relative criterion method and the jackknife procedure. There was no significant difference in LRP onset between the three conditions (F(1.5,29.1)=0.5). Amplitude analysis was conducted with prime type (go, no-go, neutral) and target type (go, no-go) as repeated measures factors in 50ms time windows from target onset. This revealed no significant main effects of prime and no prime x target interactions. From 200 to 250 ms there was a near significant (F(1,19)=3.4, p=0.081) main effect of target type, which then became highly significant

($p<0.001$) in each 50ms time window up until and including the 400 to 450ms time window.

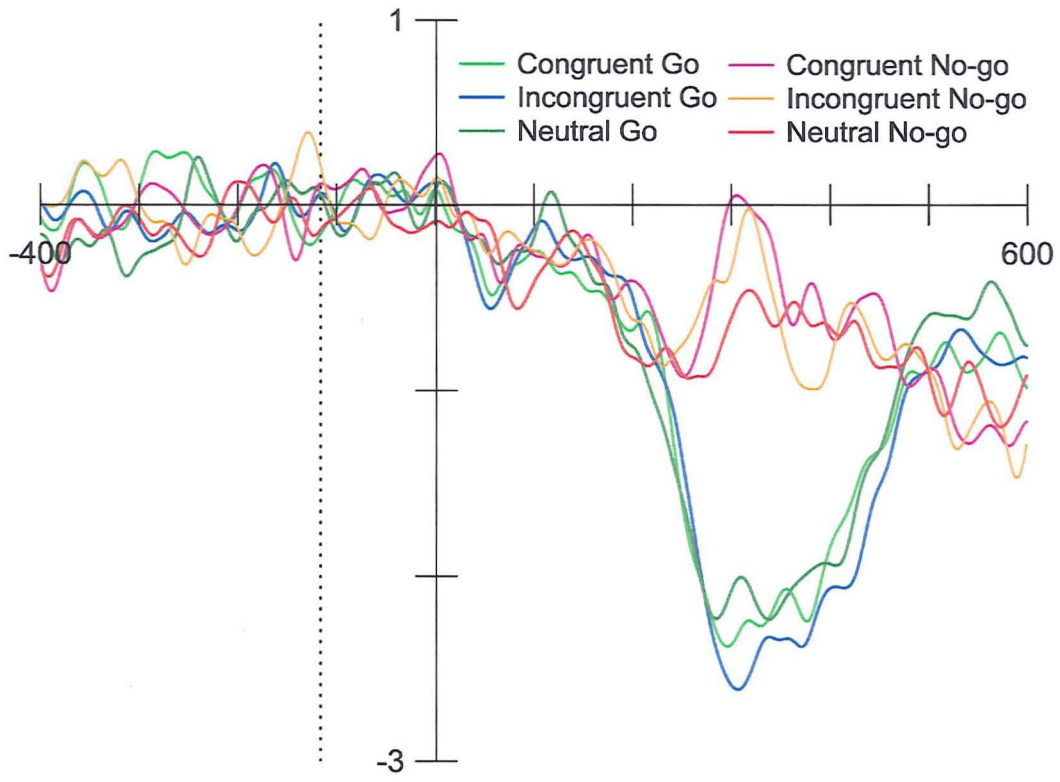

Figure 8.2: LRP waveforms for the six conditions in experiment 4 with respect to target onset. Prime onset at -116 signified by dashed line.

Frontal No-go N2 and P3 Analysis

Figure 8.3 shows the difference waveforms for each of the three no-go conditions compared to the neutral go condition. Two separate one-way ANOVAs were conducted at electrode FCz for the three no-go difference waveforms to explore the amplitude of the no-go N2 and P3. The N2 time window (275ms to 325ms) was chosen to encompass the period around the peak latency of the no-go N2. Similarly the P3 time window (400ms to 450ms) was meant to capture any differences in average amplitude of the no-go P3. The ANOVA for each time window included prime congruency as a repeated measures factor (congruent, incongruent and neutral).

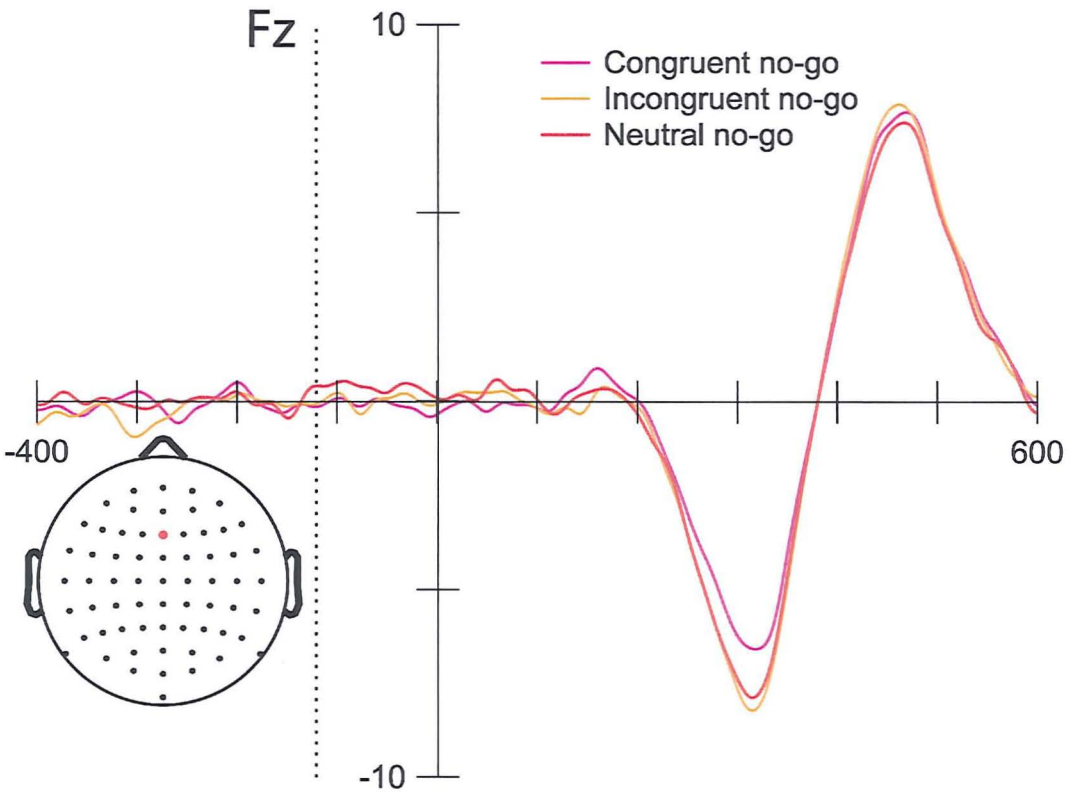

Figure 8.3: Difference ERP waveforms for the three no-go conditions at electrode Fz.

In the N2 time window there was a significant main effect of prime congruency ($F(1.9,31.5)=5.4$, $p<0.05$). Follow up t-tests confirmed that incongruent no-go trials ($m=-7.3$; $std=4.7$) showed significantly more negative amplitude than congruent no-go trials ($m=-5.9$; $std=3.8$; $t(19)=3$, $p<0.001$). In addition, neutral no-go trials showed greater N2 amplitude ($m=-7.1$; $std=4.2$) in comparison to congruent no-go trials ($t(19)=2.4$, $p<0.05$). Neutral no-go N2 amplitude did not significantly differ from congruent no-go N2 amplitude ($t(15)=0.5$, $p=0.63$). In the P3 time window there was no significant effect of prime congruency on P3 amplitude ($F(1.7,32)=0.8$).

Further analysis was conducted to explore the possibility that the magnitude of the N2 modulation was related to participants' scores on the forced-choice task. N2 priming effects were calculated by subtracting congruent no-go amplitude from incongruent no-go amplitude in the two time windows. There were no significant correlations between either of these measures and performance on the prime identification task. Furthermore, the above N2 analysis was repeated using only those 18 participants who performed at

chance level in the prime identification task resulting in the identical significant effects to those described above. These results confirm that the modulation of the no-go N2 was independent of prime identification performance, and that this modulation occurred even in those subjects who failed to identify the primes in the control task.

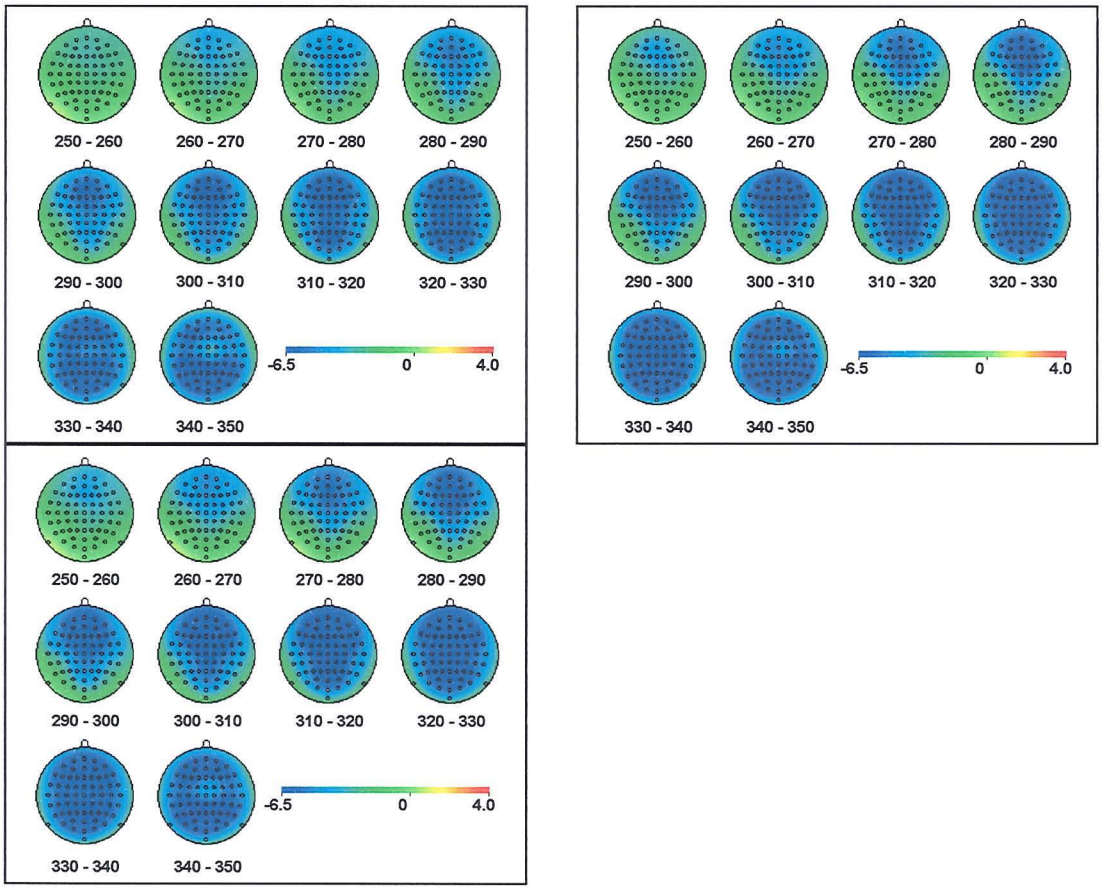

Figure 8.4: Scalp distribution of no-go N2 for congruent (top left), incongruent (top right) and neutral (bottom left) no-go trials. Each scalp map represents the average amplitude for the specified 10ms time window

Figure 8.4 shows the topography of the no-go N2 for congruent, incongruent and neutral no-go trials. The scalp maps show that the no-go N2 initially appears over frontocentral electrodes before later spreading to parietal electrodes showing a second maximum at Pz. Figure 8.5 shows the topography of the no-go P3 which also shows a frontocentral maximum.

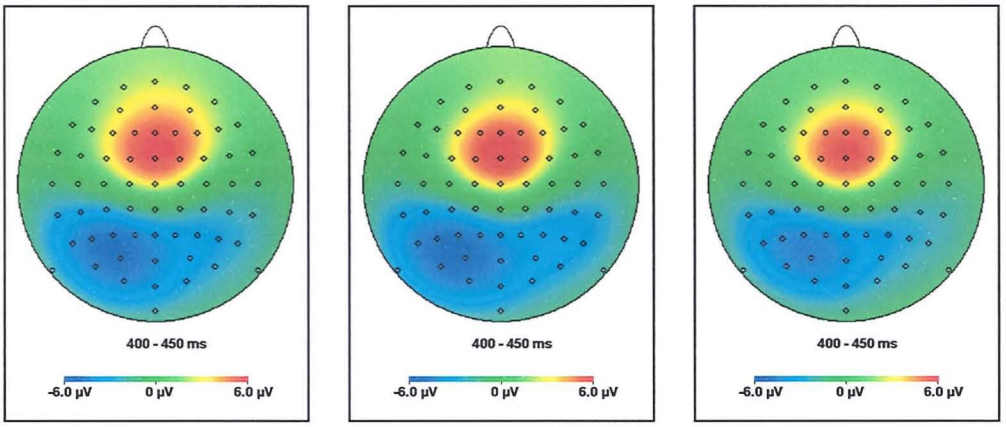

Figure 8.5: Scalp distribution of no-go P3 for congruent (left panel), incongruent (central panel) and neutral (right panel) no-go trials.

To explore any early prime-related modulation of frontal inhibition/control processes, factorial analysis was conducted on all six conditions at electrode Fz. Figure 8.6 shows the grand average ERP at electrode Fz for the six conditions. The no-go N2/P3 complex is clearly visible onsetting around 200ms after stimulus onset. Visual inspection of the ERP waveforms also reveals that there appears to be no early prime-related effects at electrode Fz. ANOVA from 120 to 180ms after stimulus onset, with prime type (go, no-go, neutral) and target type (go, no-go) revealed no significant effects.

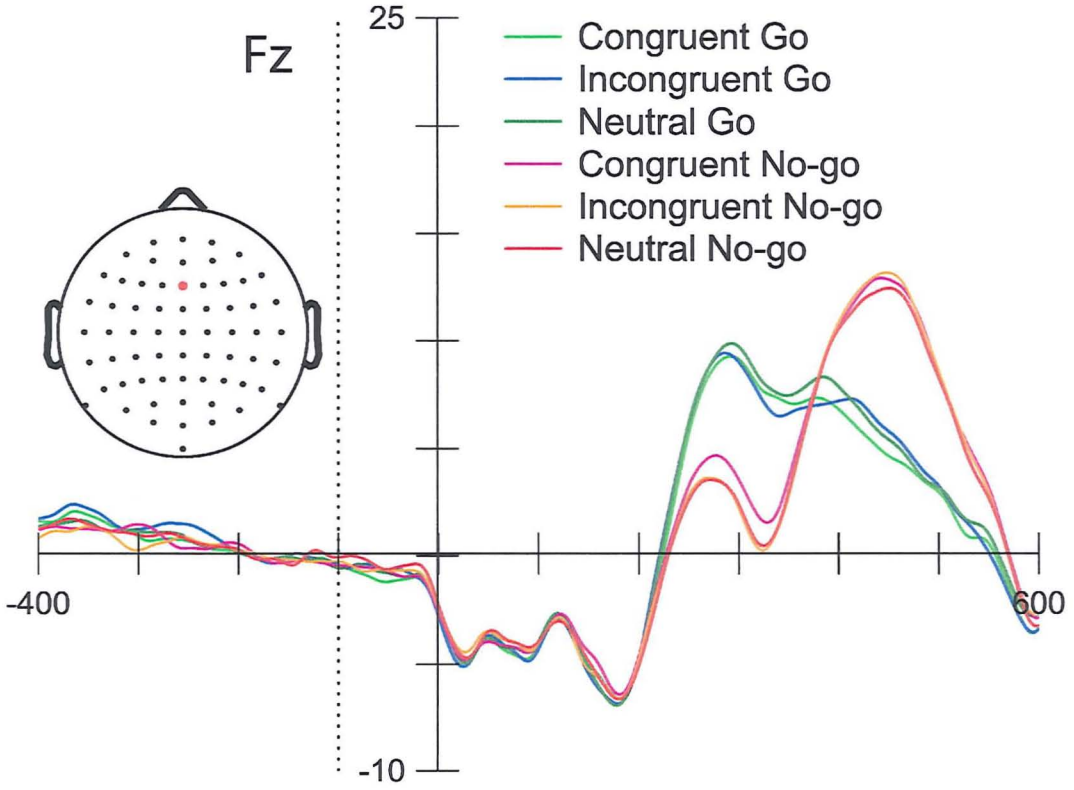

Figure 8.6: ERP waveforms for the six conditions in experiment 4 at electrode Fz.

Early visual ERP effects

Figure 8.7 shows the grand average ERPs at electrode Oz for all six conditions. Visual inspection revealed that N1 and P1 components were less well defined than in previous experiments with ERP activity showing a sequence of two positive and two negative peaks, possibly reflecting visual N1 and P1 components to the prime and target stimuli respectively. ANOVA with prime type (go, no-go, neutral) and target type (go, no-go) as repeated measures factors was conducted in four time windows centred on the observed peaks (0-30ms, 30-60ms, 60-110ms, 110-160ms). There were no significant effects in any of these time windows.

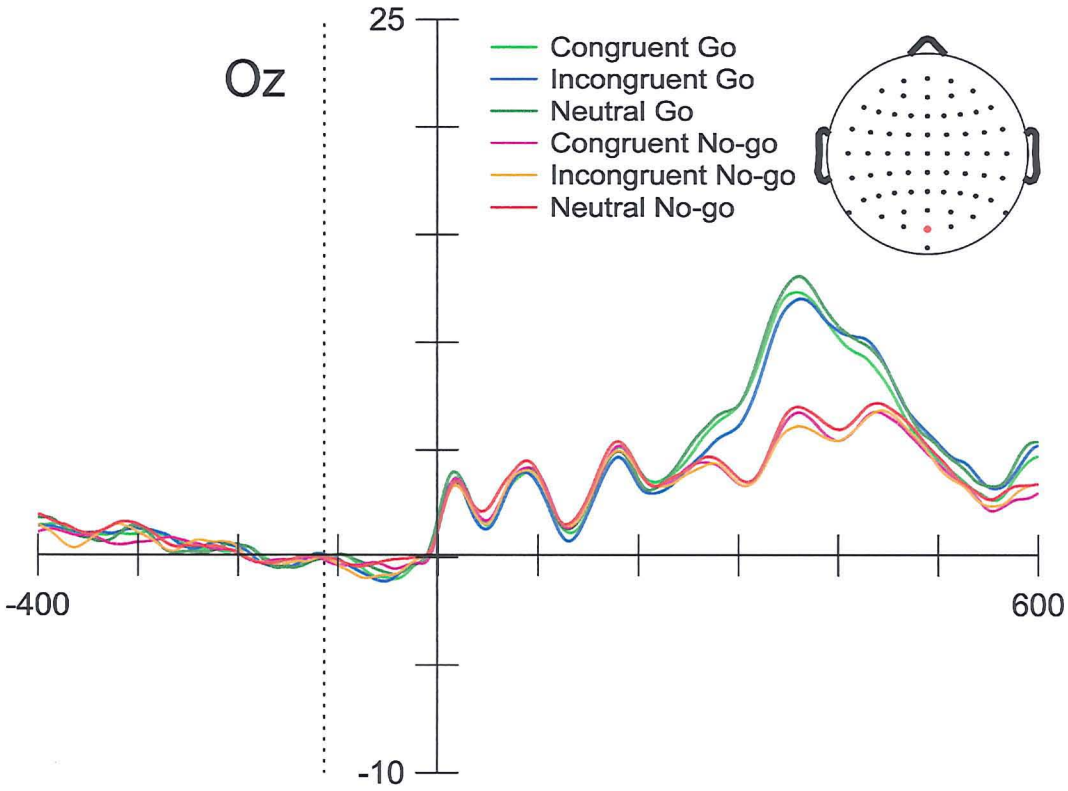

Figure 8.7: ERP waveforms at electrode Oz.

Further analysis was conducted to explore possible lateralised effects related to physical characteristics of the prime. Figure 8.8 shows grand average ERPs constructed dependent on the visual stimulus, such that they were formed from different conditions for participants with different response mappings. Visual inspection reveals that the waveforms appear to separate based on prime type at from about 20 to 50ms after target onset with left primes showing greater left hemisphere sided amplitude in comparison to

right primes. ANOVA was conducted in this time window with prime type (left, right) target type (left,right,neutral) as repeated measures factors and response mapping (left go, right go) as an independent factor. Response mapping was included to ensure that the any observed effects were truly related to the physical characteristics of the prime and not the relevance of the stimuli. A significant main effect of prime was observed for the O1-O2 difference ($F(1.9,26.9)=6.5$, $p<0.01$) and the PO7-PO8 difference ($F(1.8,28.2)=8.3$, $p<0.01$). A significant linear contrast (both $ps<0.002$) was observed for both electrode pairings, with left go primes showing greatest amplitude followed by neutral prime and then incongruent primes. There was no significant prime x target interaction, and no significant interactions with response mapping. From 100ms after stimulus onset the waveforms separated based on the primes with an initial increase for left sided targets from 100 to 150ms then being replaced by an increase in right sided targets from 150 to 200ms (confirmed by repeated measures ANOVA in these time windows showing only main effects of prime). These effects likely reflect modulation of the later positive and negative peaks observed at electrode Oz, possibly a target-related P1 and N1.

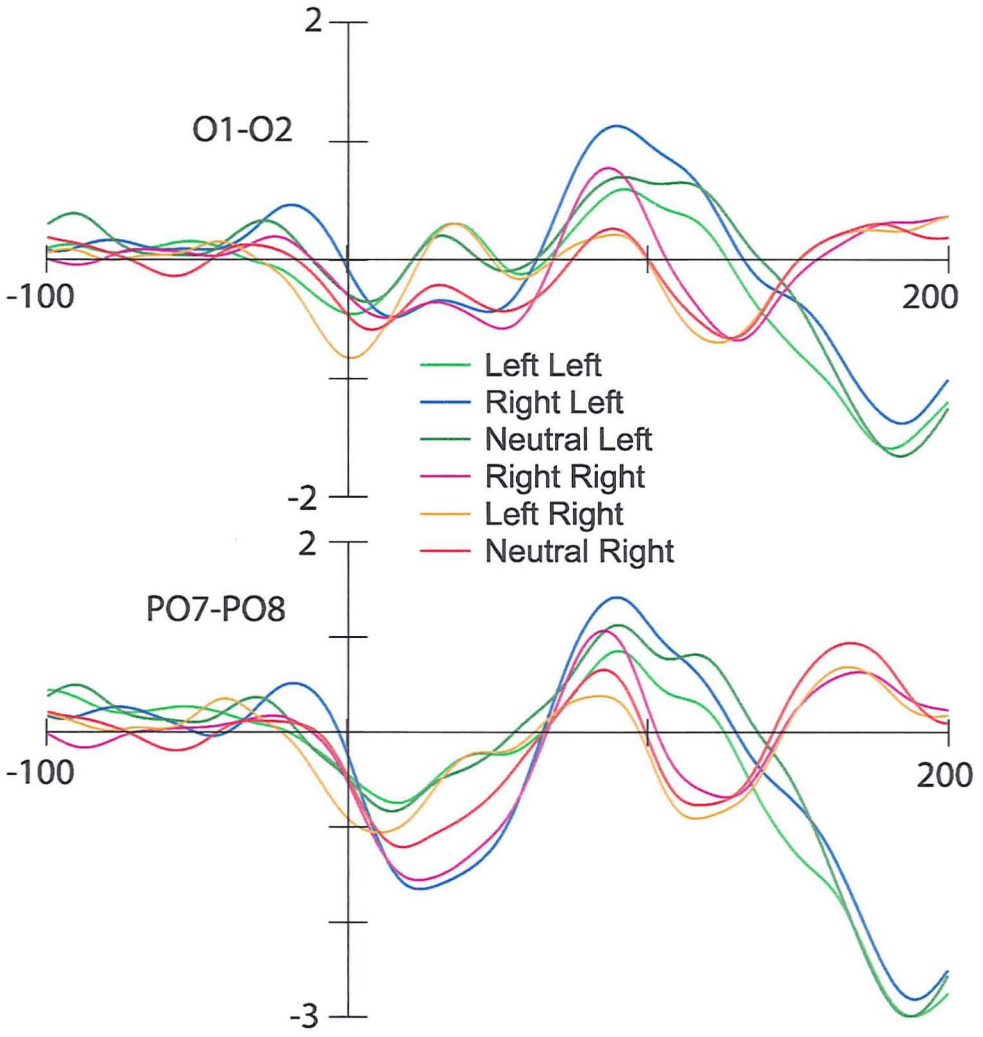

Figure 8.8: Lateralised visual ERP difference waveforms for experiment 4.

Go/No-go Differences

As in the previous experiments, amplitude analysis of go/no-go differences was conducted using a five-way repeated measures ANOVA with prime type (go,no-go,neutral), target type (go,no-go), hemisphere (left,right), anterior-posterior (Fp, F, FC, C, P, and O) and time (120-180, 180-350, 350-550) as within-subjects factors. The early time window was selected to explore any early differences in the ERPs associated with the unconscious primes. The second and third time windows were centred on the no-go N2 and no-go P3 respectively.

Initial analysis focusing on the six lateral electrode locations over the left and right hemisphere revealed no significant main effect of hemisphere ($F(1,19)=3.5$, $p=0.08$). However, there was a significant hemisphere x target

x time interaction ($F(1.6,29.7)=6.2$, $p<0.05$) as well as a near significant hemisphere x prime x time interaction ($F(2.9,54.9)=8.2$, $p=0.054$). Further analysis was conducted for each of the three time windows to try and classify these interactions. This analysis revealed that there appeared to be an interaction between prime and side in the first time window only, although this failed to reach statistical significance ($F(1.5,27.5)=6.2$, $p=0.08$). Further, since this effect appeared extremely inconsistent across the anterior-posterior dimension it most likely reflected noise. Conversely, the interaction between side and target appeared to develop more in the later time windows with a non significant trend observed in the second time window ($F(1,19)=3.6$, $p=0.07$) and a significant interaction in the final time window ($F(1,19)=6.7$, $p<0.05$).

In addition to the significant interaction between target and side, this third time window also exhibited a significant main effect of side and a significant side x anterior-posterior interaction. The main effect of side was caused by increased amplitude over the right hemisphere (m=8.1; std=0.8) in comparison to the left hemisphere (m=7.5; std=0.7). The side x anterior posterior interaction manifested in a greater right lateralisation of amplitude over frontocentral electrodes (F4, FC4 and C4; mean difference = 1.1mv) in comparison to anterior electrodes (P4 and 02; mean difference = 0.2mv). Finally, the target x side interaction was caused by a more increased right lateralisation for no-go target trials (mean difference = 0.8mv) in comparison to go target trials (mean difference =0.3). It is important to note that these laterality effects were extremely small in comparison to the target and prime-related effects (explored in more detail below). Visual inspection of figure 8.9 reveals that overall ERP amplitude was greatest at midline electrodes further suggesting that the components were largely centrally distributed. Further analysis of the prime and target-related effects was therefore explore over the six midline electrode sites (Fpz, Fz, FCz, Cz, Pz and Oz).

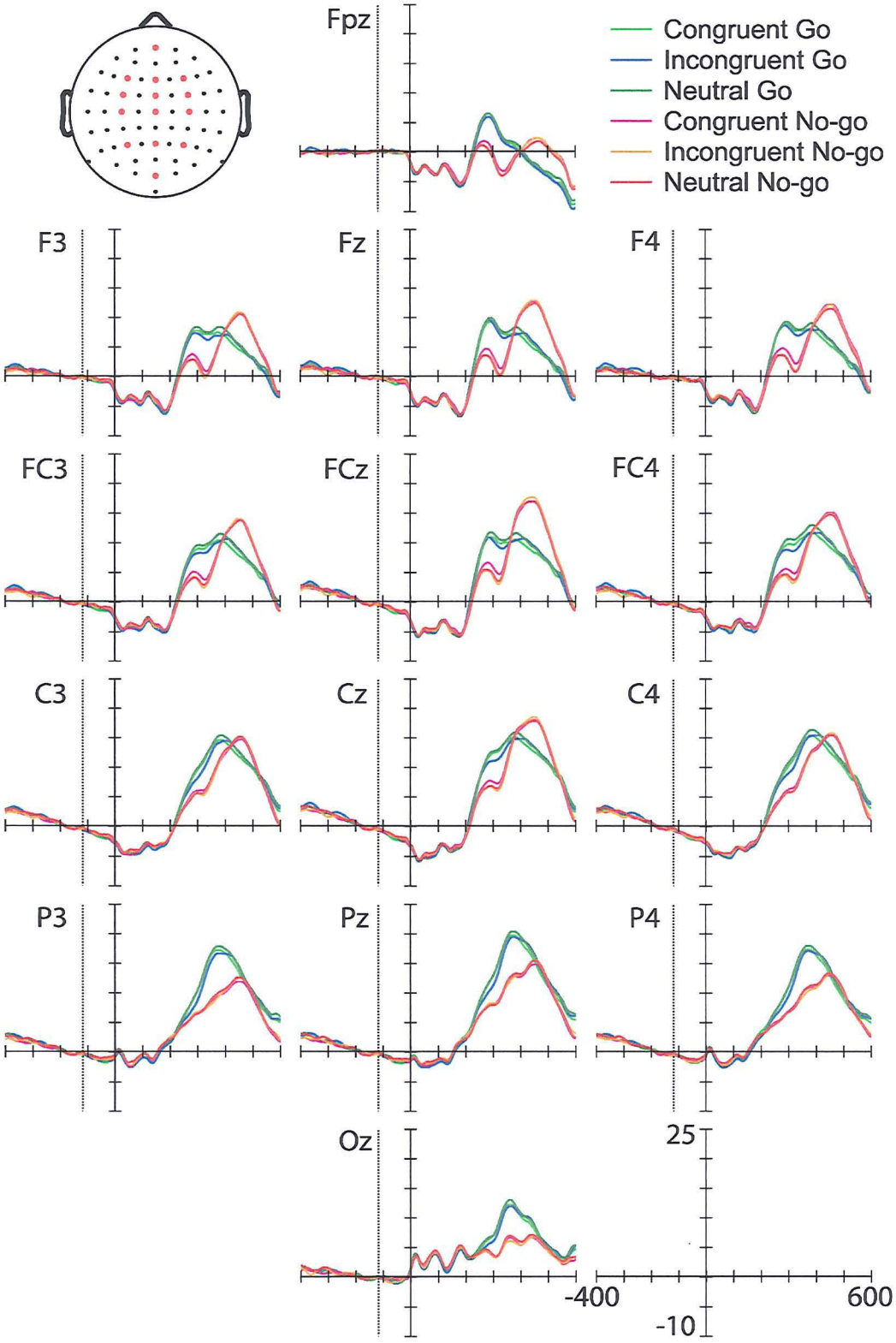

Figure 8.9: Raw ERP waveforms for experiment 4

Four-way ANOVA using the six midline electrodes (Fpz, Fz, FCz, Cz, Pz and Oz) as the anterior-posterior dimension revealed a significant target x time x anterior-posterior interaction ($F(3.4,63.9)=59.2$, $p<0.001$) as well as a prime x

time x anterior-posterior interaction ($F(6.4,122.2)=3.3$, $p<0.005$). These two effects suggest that the prime and target influenced ERPs differentially at different electrode locations and in the different time windows. However the four-way prime x target x time x anterior-posterior failed to reach statistical significance with a Greenhouse-Geisser correction applied ($F(4.5,86.1)=2$, $p=0.09$).

To explore these effects in more detail, separate three-way ANOVA was conducted for each of the three time windows. In the first time window (120ms to 180ms) there were no significant main effects or interactions involving either prime or target. In the second time window (180 to 350ms after stimulus onset) there was a significant target x prime interaction ($F(4.5,86.1)=2$, $p=0.09$) as well as a significant prime x anterior-posterior ($F(4.5,86.1)=2$, $p=0.09$) and target x anterior-posterior interaction. ($F(4.5,86.1)=2$, $p=0.09$). There was no significant three way interaction. Figure 8.10 shows these two interactions. The right panel shows that there appears to be a difference between go and no go target trials at all electrode sites. Contrasts revealed significant differences between go and no-go target trials (at $p<0.001$) at all electrode locations except Fpz, with the absence of a significant difference at Fpz likely driving the prime x anterior-posterior interaction.

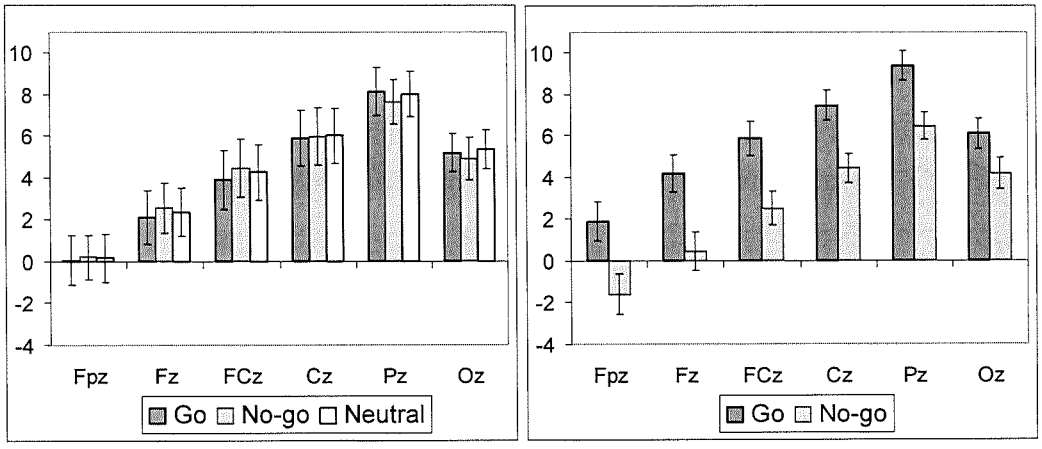

Figure 8.10: Average amplitude for midline electrodes in the middle time window dependent on prime type (left panel) and target type (right panel). Amplitude in microvolts on y axis and electrodes on x axis.

The left panel of figure 8.10 shows the amplitude in the second time window dependent on prime type. Visual inspection reveals that no-go primes are more negative than the other two prime conditions at anterior electrodes, with this effect reversed over posterior electrodes. Contrasts showed that no-go prime amplitude was significantly reduced at electrode FCz in comparison to go amplitude ($F(1,19)=4.9$, $p<0.05$). In contrast, no-go prime amplitude was significantly more positive than neutral prime amplitude at electrode Oz ($F(1,19)=5.2$, $p<0.05$). Examining the raw ERP waveforms in figure 8.8 shows that this likely reflects modulation of the no-go N2 at frontal electrodes and the P300 at parietal electrodes, with the overlapping differences at Cz and Pz cancelling one another out. The reduced N2 at frontal electrodes is characterised by a reduced no-go N2 (therefore more positive amplitude) for no-go prime trials (evident in the congruent no-go condition). At parietal electrodes the P300 is reduced in this time window for incongruent go trials manifesting in reduced amplitude for no-go primes. It is important to note however that the three way interaction between prime type, target type and anterior-posterior electrode location was not significant in this time window.

In the third time window there was a significant main effect of target ($F(1,19)=5.2$, $p<0.05$) and a target x anterior-posterior interaction ($F(2.5,47)=60$, $p<0.001$). Figure 8.11 shows the average amplitude for midline electrodes dependent on prime type. At anterior electrodes no-go target trials were significantly more positive than go target trials, with this effect reversed over electrode Pz and Oz. Contrasts confirmed significant differences (at $p<0.001$) between go and no-go target trials at all electrodes except Fpz. At Fz, FCz and Cz the no-go trials showed significantly greater amplitude while at Pz and Oz go trials showed significantly greater amplitude. This frontal modulation likely reflects the frontal no-go P3 effect, while the reversal over parietal sites reflects the increased P300 for go compared to no-go trials.

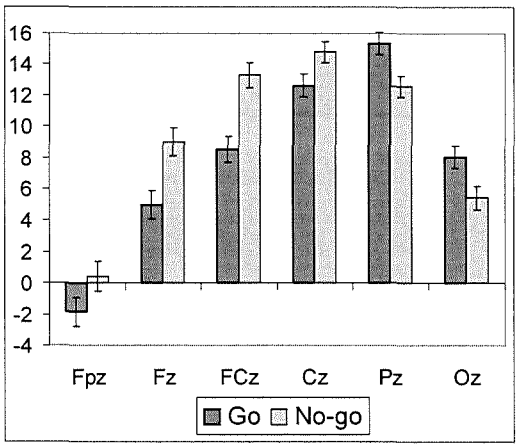

Figure 8.11: Average amplitude for midline electrodes in the late time window dependent on target type.

Discussion

Experiment 4 aimed to replicate the effects in the previous experiments while overcoming the problems previously observed with the neutral primes. Behavioural results showed that, although the magnitude of the priming effect on reaction time was notably smaller than in previous experiments, it reliably separated based on the nature of the prime. Importantly this modulation occurred between neutral and no-go primes as well as neutral and go primes with a similar effect being observed for error rates for both go and no-go trials.

The behavioural effect in this experiment was notably smaller than that observed in the other experiments. The probable explanation for this lies in the nature of the primes that were employed. Since each prime was presented at a different physical location, the prime identification task effectively amounted to a prime detection task. In this task participants could have correctly identified the type of prime simply by noticing its location (left or right of fixation). The fact that participants still performed at chance level on this task effectively meant that the primes were presented below the objective threshold for prime detection, which is known to be lower than the threshold for prime identification (Snodgrass et al., 2004). Interestingly, Snodgrass et al., (2004) also report that priming is *increased* at the objective detection threshold in comparison to the identification threshold. However,

this suggested has been vigorously contested (Holender & Duscherer, 2004; see chapter 3 for more detailed discussion). The finding that primes at the objective detection threshold produced a relatively small priming effect does not support Snodgrass et al.'s (2004) assumption. Rather the finding that an effect still emerged despite the primes being highly invisible suggests that the go/no-go priming effects are truly unconsciously driven.

In line with the relatively small behavioural priming effects observed in the current experiment, ERP differences were also reduced in comparison to previous experiments. LRP analysis revealed that there was no significant difference in the onset of the LRP between the three go conditions. Similarly, there were no significant amplitude effects related to the unconscious primes. This perhaps suggests that the priming effect was too small to influence motor preparation. However, it is also possible that the rather small effect size observed in the reaction times and error rates likely meant that the LRP became somewhat insensitive to this very small difference. Given the low signal to noise ratio of the LRP, it is likely that small effects will be less noticeable in this component than in others. There were significant differences observed in the amplitude of the no-go N2 component dependent on the prime for no-go trials. Importantly this modulation occurred such that congruent no-go trials showed significantly reduced N2 in comparison to incongruent and neutral primes. This suggests that rather than simply reflecting an increased N2 on congruent trials, the modulation of this component is caused by a priming of the no-go response. Additionally, neutral prime trials did not significantly differ from incongruent prime trials. Similarly, the parietal P300 component showed decreased amplitude for no-go prime trials, but no difference between go and neutral prime trials. Since there were no significant differences in LRP onset this suggests that the go prime was not successful in initiating a motor response, and therefore the N2 component should not be increased in the incongruent no-go condition in comparison to a neutral baseline. It is important to note that an alternative explanation is also possible, namely that the neutral and go prime both primed the go response to an equal degree, thus increasing the amount of conflict/inhibition required in response to a subsequent no-go

target. Given the lack of effect in the LRP and the fact that neutral primes showed a significant behavioural difference from go primes, this seems unlikely. However, since there were behavioural difference between these two conditions it is perhaps somewhat surprising that no ERP effects were evident.

Unlike in the first three experiments there were no significant differences in no-go P3 amplitude between the three no-go conditions in the current experiment. Given the uncertainty regarding the exact functional significance of the two components of the N2/P3 complex a precise interpretation of this is difficult. Some recent evidence suggests that the while the no-go N2 may be more involved in passive monitoring of conflict, the P3 may be involved in inhibition of the response (Dimoska et al., 2006). If this is the case, then the modulation of the no-go N2 in the current experiment may reflect modulation of the amount of conflict induced by the no-go primes. However, there are reasons to believe that when go and no-go response are equally probable, at least part of the N2 component is involved in inhibition (Lavric et al., 2004). A more detailed discussion of the N2/P3 complex in relation to all the experiments in this thesis will be provided in chapter 10.

In addition to the absence of no-go P3 modulation, the current experiment also failed to show any significant early differences related to the nature of the prime. In experiments 1 and 2, as in similar studies exploring the LRP in motor priming (Dehaene et al., 1998; Leuthold & Kopp, 1998) this early prime-related separation was taken as evidence that the unconscious primes could directly initiate frontal inhibition/control mechanisms. Therefore, the lack of such an effect in the current experiment makes it difficult to come to a similar conclusion. Instead it might be the case that priming occurred due to earlier categorisation of the target stimulus for congruent primes in comparison to neutral primes. However, while it is easy to conceive that such an effect may drive the observed difference in reaction times, it is not immediately obvious how early categorisation of a no-go target would reduce the amount of engagement required from frontal inhibition/control mechanisms. However, it is also unclear how the target-related no-go N2

effects may be reduced for congruent trials, without some early engagement of this system.

Analysis of visual ERP effects revealed significant lateralised differences dependent on the prime type, with increased positivity contralateral to the location of the prime. This likely reflects an increased visual response to the lateral prime stimuli. This effect was preserved independent of response mapping, suggesting that it reflected the visual characteristics of the prime rather than a selective process responsive to one type of target. For example, the go arrow might be described as the target, since it requires a response, while a no-go arrow could be described as a non target. Thus any visual or attention effects, such as the N2pc (see Chapter 2) that selectively produces an asymmetry to go primes (and not to no-go or neutral primes) would be reflected in a between participants effect of response mapping. Since no such effect was present these early visual differences likely reflect a basic visual response to the location of the prime stimulus.

Conclusions

The major finding in the current experiment was that unconscious no-go primes were able to facilitate a reduction in no-go N2 in comparison to a neutral baseline. This suggests that rather than reflecting differences in the amount of motor preparation between different prime conditions, the modulation of this component reflects priming of a no-go response. However, the current experiment failed to show significant early modulation of this frontal activity, thus questioning the exact nature of this facilitatory effect.

Chapter 9

Experiment 5 – Unconscious modulation of the no-go N2 and P3 is associated with the degree of behavioural priming.

Introduction

This final experiment aimed to further extend the main findings of the previous experiments while overcoming some of the problems encountered. Experiment 1 found that unconscious masked primes were able to directly initiate ERP components associated with frontal inhibition/control mechanisms. However, this modulation was observed in an experiment that produces a rather unusual negative compatibility effect, such that a congruent prime *impedes* performance. Three further experiments replicated the unconscious modulation of the no-go N2 and P3. However, while the second and third experiments provided further evidence of priming of target-related ERP components a number of problems with the neutral primes in these experiments meant that they could not be fully analysed. While experiment 4 successfully resolved the problems with the neutral primes it produced only very weak priming effects. In addition, there was a complete absence of early prime-related effects in this experiment.

In an attempt to produce a more consistent priming effect the current experiment reverted back to stimuli similar to those employed in experiment 1. However, in order to avoid the complication produced by the mask-induced reversal of the prime effects, a different mask was employed. Instead of using a mask that was constructed from a compound of the two possible targets, a random checkerboard mask was used. Since this mask did not share any physical features in common with the primes or targets, it should produce a positive compatibility effect. Moreover, since the primes were presented in the same location, this should ensure that performance on the prime identification task truly required identification of the nature of the primes, and not simply detection of the primes as in the previous experiment.

Hypotheses

The subliminal primes in the current experiment should produce a positive compatibility effect such that performance is facilitated by a congruent prime and impeded by an incongruent prime. Furthermore, the LRP and the no-go N2/P3 complex should be modulated as a function of the unconscious prime. No-go N2 and P3 amplitude should be reduced for congruent trials in comparison to neutral trials, since the unconscious prime should facilitate processing thus requiring less engagement of frontal control/inhibition mechanisms. Similarly, N2 and P3 amplitude should be increased in response to an incongruent prime in comparison to a neutral prime, since subliminal priming of a go response in a no-go target condition will require a greater subsequent inhibition, to successfully withhold the response. Additionally, the facilitation of the no-go N2/P3 complex and the LRP should be associated with an early separation of these responses dependent on the unconscious primes.

Method

Participants

Twenty one volunteers (four male and 17 female) were recruited by means of poster advertisement. Participants received course credits in exchange for participation in the experiment. All participants were right handed and had normal or corrected to normal vision. The mean age of participants was 22 years and two months, with a range of 18 to 35 years.

Experimental Procedure

All participants completed 14 blocks of the go/no-go task followed by three blocks of the prime identification task in a single experimental session lasting approximately two hours. Each go/no-go block contained 72 trials presented in a random order. The 14 experimental blocks were preceded by two practice blocks of 48 trials. Target stimuli were identical to those in experiment 1 and consisted of either two left pointing (<<) or two right pointing arrows (>>). Participants were required to respond to arrows

pointing in one direction and to refrain from responding to arrows in the opposite direction. Half the participants were instructed to press a button in response to left pointing arrows and half were instructed to respond to right pointing arrows. The response hand was varied from one block to the next. The participants were informed that that they had a time limit of 450 ms to respond to the go stimuli and that they should react as quickly as possible without sacrificing accuracy. Participants were given visual feedback immediately after the 450ms response window for correct responses, incorrect responses and non-response as well as false alarms and incorrect non-responses.

Participants were informed that random chequerboard type patterns would be presented in advance of the stimulus. In addition, masked primes were presented before and after two different checkerboard patterns. Participants were not informed of the presence of the primes. These primes were congruent, incongruent or neutral with respect to the target stimulus. Congruent primes consisted of fainter versions of the same arrows as the target stimuli. Neutral primes consisted of one two arrows pointing in opposite directions (<> and ><). There were an equal number of neutral, congruent and incongruent primes in each block and across the course of the experiment. A different neutral prime was used for each participant, with the choice of prime and the response mapping counterbalanced across participants.

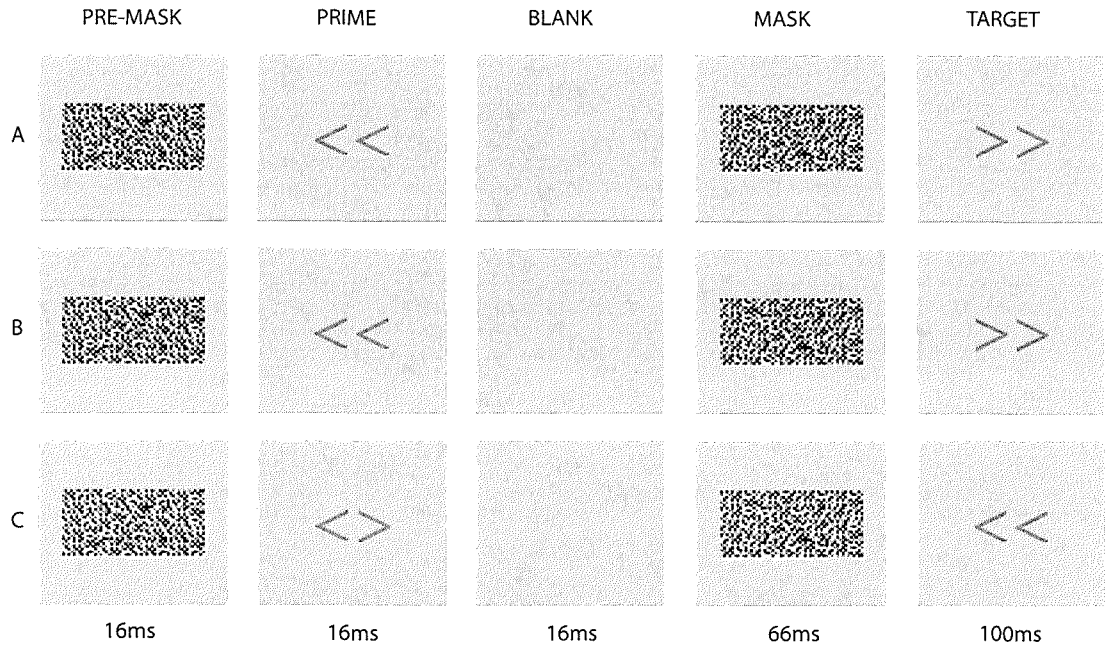

Figure 9.1: Stimuli for experiment 5. A congruent (A), incongruent (B), and neutral (C) trial.

Pre-masks and masks were 6.2cm across and 3.2 cm high. Double arrow prime and target stimuli were 3.2cm across and 1.6cm high. All primes, masks and stimuli were presented at the centre of the display on a grey background. Each trial began with a large central fixation for 200ms which acted as a warning signal to participants that the next trial had begun. A smaller fixation cross was then presented for 600ms. Next, the pre-mask was presented for 16ms followed by the prime for 16ms. After a blank screen for 16ms, the mask (66ms) and then the target stimulus (100ms) were displayed. Following the 450ms response window participants were given visual feedback. Finally a blink pause was presented for 800ms followed by a blank screen for a randomly selected interval between 150ms and 600ms.

As in previous experiments, following the go/no-go task, participants were asked whether they noticed anything other than the checkerboards before the prime, and if so what they saw. Additionally, they were asked if they thought something might have flashed up. They were then shown the exact stimulus sequence in slow motion. After seeing the slowed sequence participants were asked if they recognised having seen any of the primes during the go/no-go task. The prime identification task consisted of one

practice block of 32 trials and three experimental blocks of 96 trials. One half of trials contained a right pointing arrow prime and the other half contained a left pointing arrow prime. No neutral primes were presented in this task and no target stimuli were presented. Participants were informed that they should press the far left button for left arrow primes and the far right button for right pointing primes. Participants had no time limit to respond, and received feedback at the end of each trial. Finally participants were asked to report whether the felt they were able to see the masked primes during the detection task.

Behavioural Results

Awareness of Primes

Table 9.1 shows the participants' responses to the four questions regarding the visibility of the primes. None of the 21 subjects reported having seen anything appear before the diamond shape. Four subjects thought they might have seen a flash, while five thought that they recognised the primes after having seen the slow motion sequence. Only one participant reported having seen the prime in the prime identification task.

Table 9.1: Summary of responses to Questions 1 to 5

	Notice?	What?	Flash?	Recognise?	See?
NO	21	21	17	16	20
YES	0	0	4	5	1

Performance on the forced-choice task ranged from 42% to 56% and averaged 49.5% which was not significantly different from chance ($t(20)=0.7$, $p=0.5$). In addition, d' scores were not significantly different from zero (mean=-0.02, t=-0.5, p=0.62). Mean accuracy for those four subjects detecting a flash was 49%, and 50% for those who claimed to recognise the prime. The one participant who reported to having been able to see the primes in the prime identification task achieved 43% accuracy. These findings suggest that increased subjective awareness was not associated with increased performance on the objective awareness measures.

Priming

Repeated measures ANOVA showed a significant main effect of prime-stimulus congruency on reaction times ($F(2,38)=14.9$, $p<0.001$) for go trials. A significant main effect of accuracy was also observed for go trials ($F(2,38)=4.3$, $p<0.05$) and no-go trials ($F(2,38)=10.3$, $p<0.001$). Subsequent t-tests showed a significant difference between all three prime congruency conditions for go trials (at $p<0.01$). Similarly there were significant differences on accuracy for all pair-wise comparisons (at $p<0.05$) with the exception of the comparison between incongruent no-go and neutral no-go trials.

Table 9.2.1: Mean Reaction times and accuracy (and Standard Deviations) for go trials

	Congruent	Incongruent	Neutral
RT	360 *(30)*	388 *(20)*	373 *(19)*
Acc	0.93 *(0.07)*	0.91 *(0.08)*	0.92 *(0.07)*

Table 9.2.2: Mean accuracy (and Standard Deviations) for no-go trials

	Congruent	Incongruent	Neutral
Acc	0.97 *(0.03)*	0.91 *(0.08)*	0.96 *(0.03)*

Correlations were calculated between priming and prime identification performance to determine whether priming may have been caused by residual awareness on some trials. Raw scores on the forced-choice task as well as d' scores and absolute values of d' were correlated with nine different measures of behavioural priming, reflecting all pair-wise differences for reaction times on go trials and accuracy on go and no-go trials. The only significant correlations were between the difference in reaction times between incongruent and neutral go trials and both d' ($r=-5.6$, $p<0.01$) and prime identification accuracy ($r=-5.6$, $p<0.01$). However, the amount of priming was negatively correlated with each of these measures. These findings confirm that the priming effects were not likely to have been caused by residual awareness of the primes.

EEG Results

Eighteen out of the twenty-one participants were included in the EEG analysis. One participant was excluded due to an equipment failure during the recording. Two further participants were excluded due to excessive blink artefact. ERPs were formed from the remaining eighteen participants with an average of between 115 and 130 trials per condition, with a minimum of 80 trials per condition. There were approximately equal numbers of trials with right (mean = 63 trials) and left (mean = 64 trials) hand responses with a minimum of 38 trials per response hand.

LRP analysis

Figure 9.2 shows the grand average LRP waveforms for the six conditions in the current experiment. ANOVA on the jackknifed LRP onsets for go target trials revealed no significant difference dependent on prime congruency ($p=0.2$). Amplitude analysis was conducted with prime type (go, no-go, neutral) and target type (go, no-go) as repeated measures factors. ANOVA from -100 to 0ms revealed a significant main effect of prime ($F(1.7,29.5)=4.3$, $p<0.05$), with subsequent contrasts showing that go prime trials were significantly more negative than no-go prime trials ($F(1,17)=6.8$, $p<0.05$). Visual inspection of the LRP reveals a large positive deflection for the congruent go condition beginning at around 180ms before target onset. Since this effect occurs prior to prime onset and all conditions were presented unpredictably in a random order and were identical up until prime onset this deflection likely reflects noise. In fact the later part of the LRP for this condition also appears to show increased positive amplitude in comparison to the other two conditions, most notable at around 100ms after stimulus onset. This suggests that perhaps these differences are caused by the entire ERP being shifted slightly upwards. This could be caused by increased negative amplitude in the baseline period.

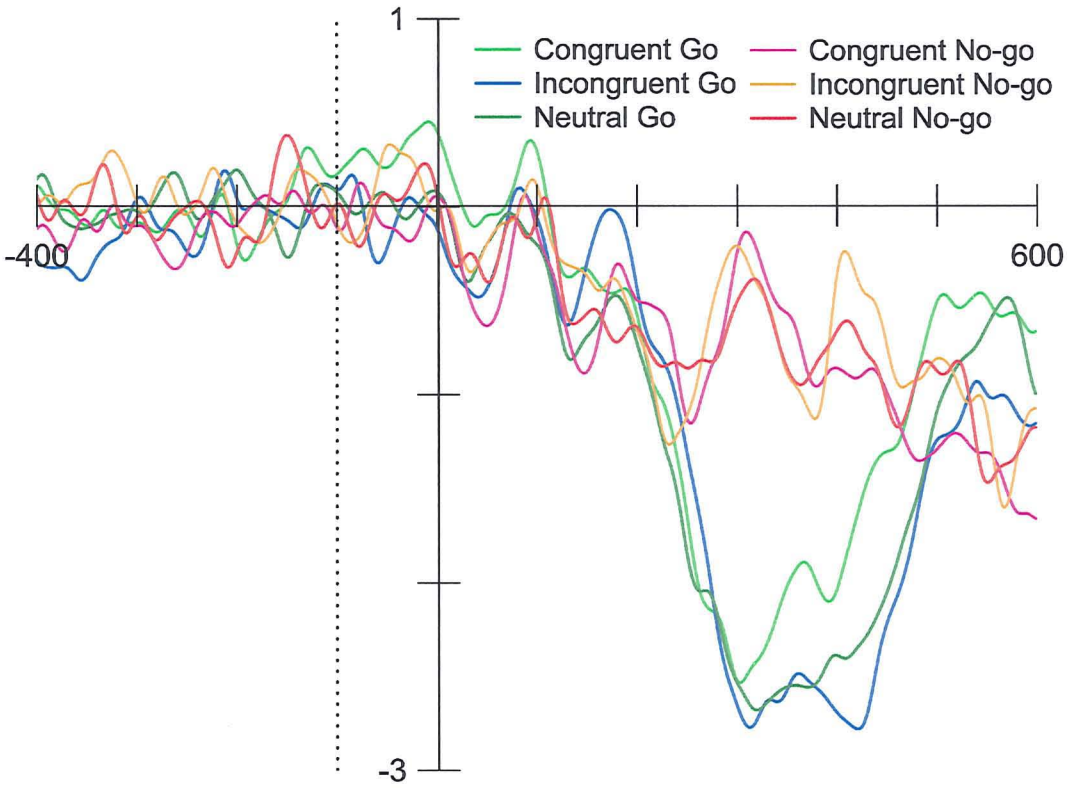

Figure 9.2: Grand average LRP for experiment 5 relative to target onset. Prime onset at -100 signified by dashes line.

Figure 9.3 shows the grand average LRP using the 150 to 100ms pre-stimulus period as a baseline. As expected, this removed the early LRP difference and introduces an extended pre-baseline difference. ANOVA with the re-baselined LRPs confirmed the absence of significant prime-related effects from -100 to 0ms ($F=0.23, p>0.9$). Furthermore, this change in the baseline period makes the early LRP fluctuations more similar between the conditions but also highlights an increased positive deflection at around 150ms for the incongruent go condition and congruent no-go condition. ANOVA from 150 to 230ms revealed a significant main effect of prime congruency ($F(1.6,27.7)=4.4$, $p<0.05$) with a significant linear contrast for prime type ($F(1,17)=5.2$, $p<0.05$) such that no-go primes showed the most positive amplitude and go primes showed the most negative amplitude. There was no significant effect of target and no target x prime interaction in this time window. These findings are in line with the prediction that the unconscious primes could directly initiate motor preparation as indexed by the LRP. ANOVA from 250 to 450ms revealed a significant main effect of

target ($F(1,17)=68$, $p<0.001$), reflecting the increased amplitude of the LRP for go target trials.

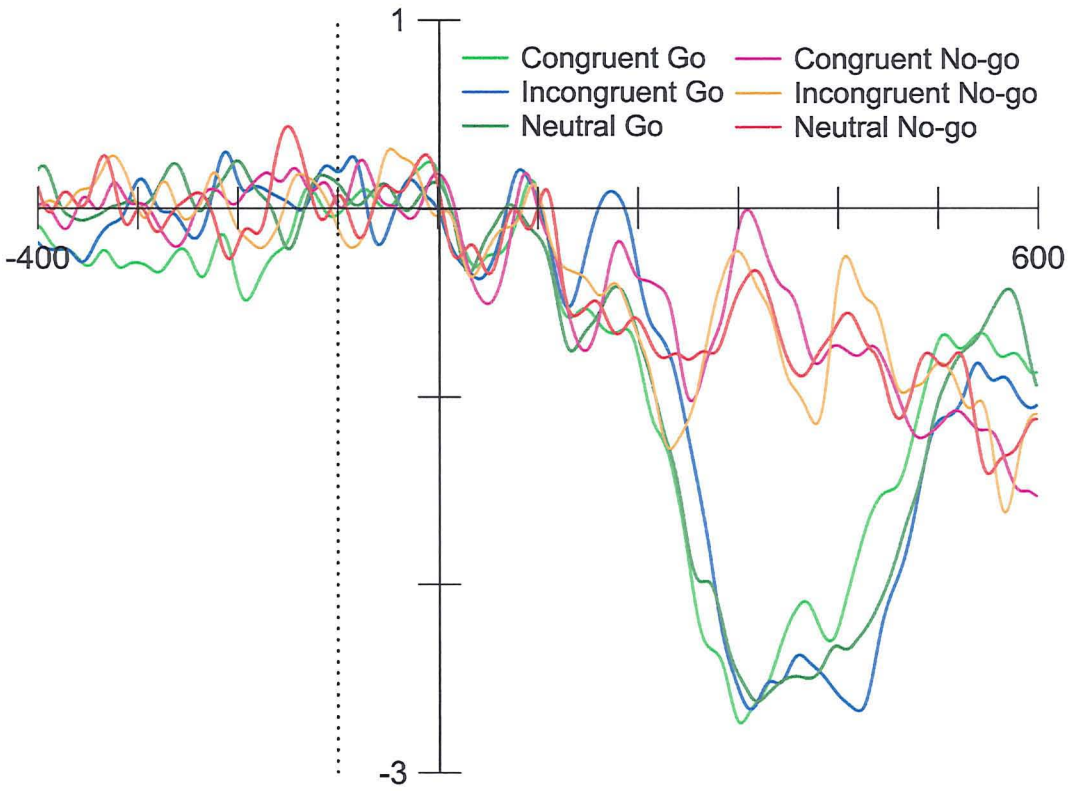

Figure 9.3: Grand average LRP for experiment 5 with a -150 to -100 baseline period.

Frontal no-go N2 and P3 analysis

Figure 9.4 shows the no-go difference waveforms at electrode Fz. Two separate one-way ANOVAs were conducted at electrode Fz for the three no-go difference waveforms to explore the amplitude of the no-go N2 and P3. The N2 time window (250ms to 350ms) was centred on the peak of the no-go N2. Similarly the P3 time window (375ms to 475ms) was selected to capture any differences in average amplitude of the no-go P3. Each ANOVA included prime congruency as a repeated measures factor (congruent, incongruent and neutral).

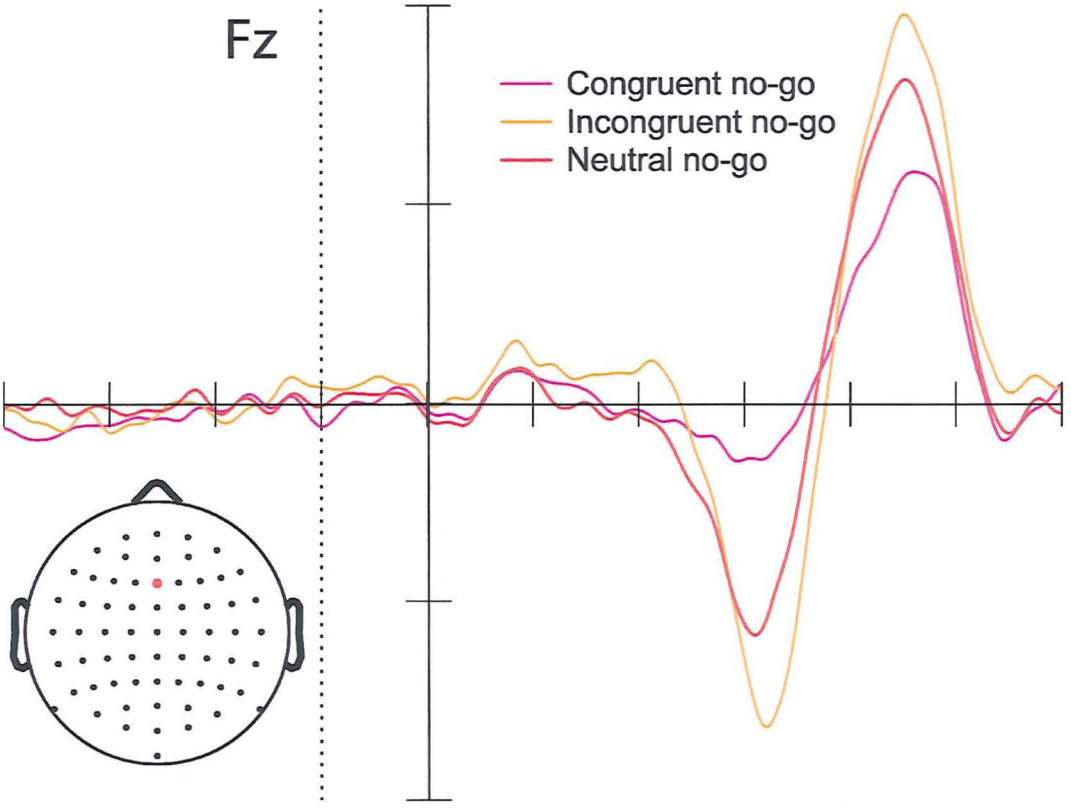

Figure 9.4: Difference ERP waveforms for the three no-go conditions at electrode Fz, with respect to target onset.

In the N2 time window there was a significant main effect of prime congruency ($F(1.4,23.1)=12.5$, $p<0.001$). Follow up t-tests confirmed that incongruent no-go trials (m=-5.3; std=4.8) showed significantly more negative amplitude than congruent no-go trials (m=-1.1; std=3.5; $t(17)=3.8$, $p<0.005$). Congruent no-go trials also showed significantly reduced N2 amplitude in comparison to neutral no-go trials (m=-4.2; std=3.7; $t(17)=3.7$, $p<0.005$). Neutral no-go N2 amplitude did not significantly differ from incongruent no-go N2 ($t(17)=0.43$, $p=0.68$).

In the P3 time window there was a significant main effect of prime congruency ($F(1.3,22.4)=5.7$, $p<0.05$). Follow up t-tests confirmed that no-go P3 average amplitude was significantly reduced for congruent no-go trials (m=4; std=3) in comparison to both neutral no-go trials (m=5.9; std=4.2; $t(17)=2.3$, $p<0.05$), and incongruent no-go trials (m=6.5; std=4.5; $t(15)=2.02$, $p=0.059$). Neutral no-go P3 amplitude did not differ from incongruent no-go P3 amplitude ($t(17)=1.4$, $p=0.19$).

Correlations were calculated between prime identification performance and the amount of N2 and P3 priming, defined by the three pair-wise differences between the three conditions in each time window. The only significant correlation was observed between d' performance and the difference between congruent and neutral N2 amplitude ($r=-0.51$, $p<0.05$) with a non significant trend also observed for raw scores on the forced-choice task ($r=-0.44$, $p=0.65$). Once again the negative nature of these correlations suggests that as prime identification performance increased, the amount of priming of the N2 amplitude decreased, suggesting that priming was not caused by residual awareness of the primes.

Interestingly, there was also a significant correlation between N2 and P3 priming and the behavioural priming. Table 9.3 shows the correlations between behavioural and ERP priming effects. It is clear that there is a widespread positive correlation particularly between no-go N2 priming and behavioural priming. For P3 amplitude, it is also of note that the congruent versus neutral comparison showed the most significant correlations with behavioural priming, including being the only EEG marker to significantly correlate with the congruent versus neutral accuracy comparison for no-go trials.

Table 9.3: Correlations between behavioural priming and ERP priming effects

		Reaction Times			Accuracy					
		Go Trials			Go Trials			No-go Trials		
		CI	CN	NI	CI	CN	NI	CI	CN	NI
N2	CI	0.88***	0.73***	0.8***	0.7***	0.43	0.59*	0.83***	0.41	0.79***
	CN	0.81***	0.47*	0.88***	0.59**	0.36	0.5*	0.58*	0.35	0.53*
	NI	0.51*	0.71***	0.26	0.49*	0.31	0.41	0.76***	0.27	0.75***
P3	CI	0.46	0.23	0.53*	0.3	0.37	0.15	0.11	0.38	0.01
	CN	0.52*	0.25	0.6**	0.46	0.48*	0.26	0.18	0.55*	0.04
	NI	0.001	0.02	0.23	0.23	0.13	0.2	0.1	0.23	0.05

***p<0.001 ; **p<0.01 ; *p<0.05 ; CI=Congruent vs. Incongruent ; CN=Congruent vs. Neutral ; NI=Neutral vs. Incongruent

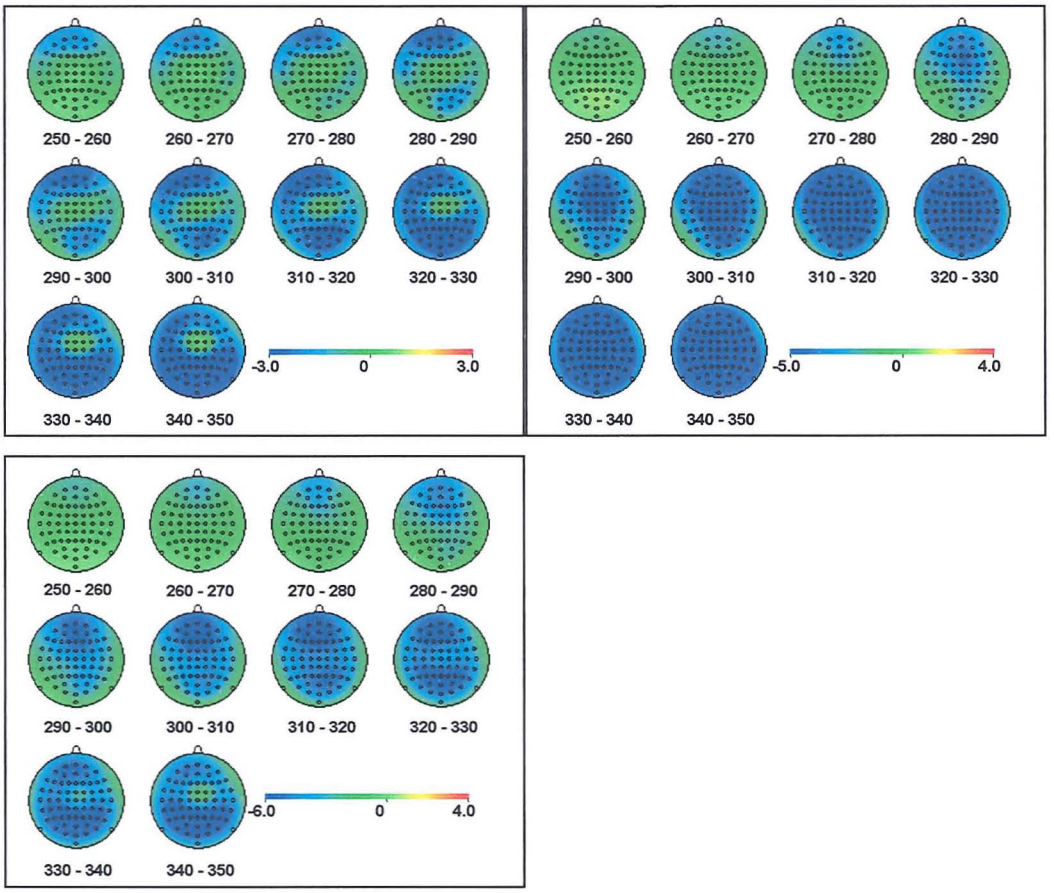

Figure 9.5: Scalp distribution of no-go N2 for congruent (top left), incongruent (top right) and neutral (bottom left) no-go trials.

Figure 9.5 shows the topographic distribution of the no-go N2 for the three no-go conditions. The distribution of the N2 shows a similar topography to previous experiments, with an early frontal negativity followed by a second parietal negativity. The no-go P3 also shows a frontocentral maximum (figure 9.6)

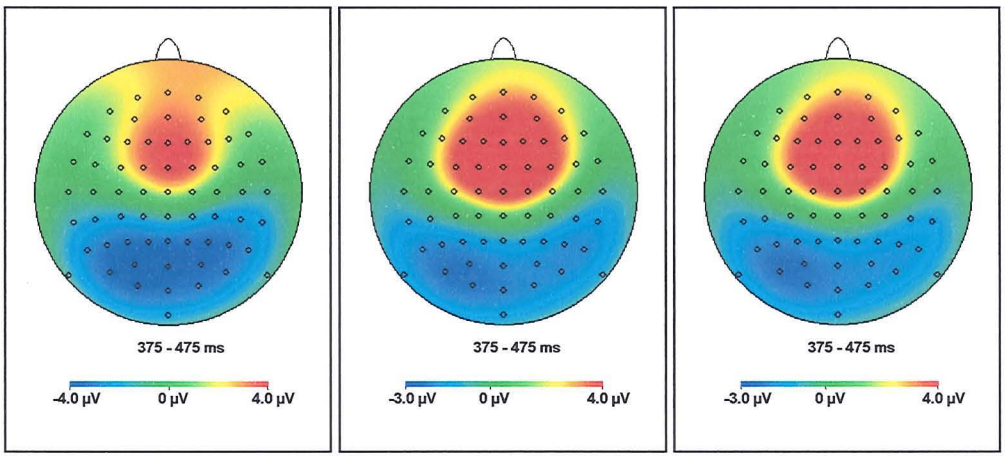

Figure 9.6: Scalp distribution of no-go P3 for congruent (top left), incongruent (top right) and neutral (bottom left) no-go trials.

To explore the possibility that the unconscious primes were able to directly elicit an early frontal no-go N2, grand average ERPs for all six conditions were explored at electrode Fz. Figure 9.7 shows the ERP waveforms for each condition at Fz. The target-related no-go N2 and P3 are clearly visible beginning around 250 after stimulus onset. Although no clear early separations are evident visual inspection reveals that from around 160 to 210ms after stimulus onset incongruent no-go and congruent go trials are the most positive, while incongruent go trials are the most negative. ANOVA in this time window with prime type (go, no-go, neutral) and target type (go, no-go) as repeated measures factors revealed a non-significant trend towards a main effect of prime ($F(1.5,25.4)=2.8$, $p=0.09$), with the subsequent contrast between go and no-go prime trials just reaching significance ($F(1,17)=4.4$, $p=0.05$). These results suggests that, although small, there were some early effects at electrode Fz that where entirely dependent on the nature of the unconscious prime.

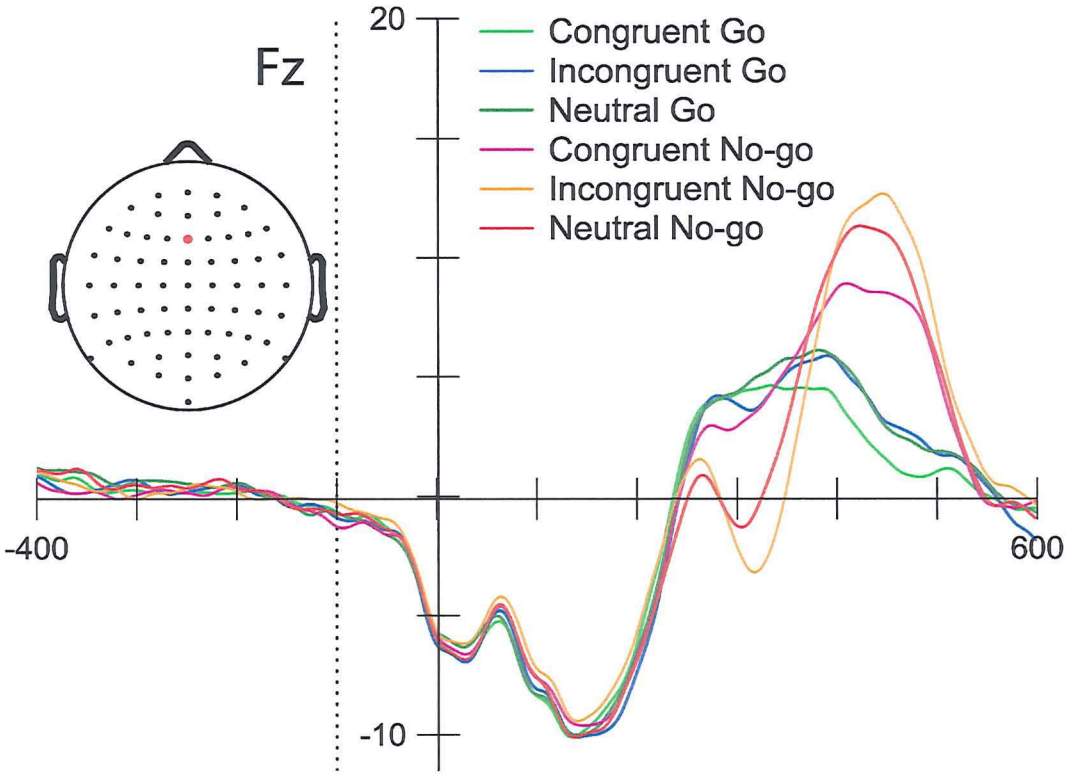

Figure 9.7: ERP waveforms at electrode Fz.

Early visual ERP effects

Figure 9.8 shows the grand average ERPs at electrode Oz for all six conditions. Repeated measure ANOVA with prime type (go, no-go and neutral) and target type (go, no-go) revealed no main effect of target, no main effect of prime and no two-way interaction for P1 (0 to 20ms) amplitude. Similarly in the N1 time window (50 to 80ms), there was no significant effects. In addition, the comparison between congruent trials (no-go prime no-go target and go prime go target) and incongruent trials (no-go prime go target and go prime no-go target) was not significant ($F(1,17=2.6, p=0.13)$).

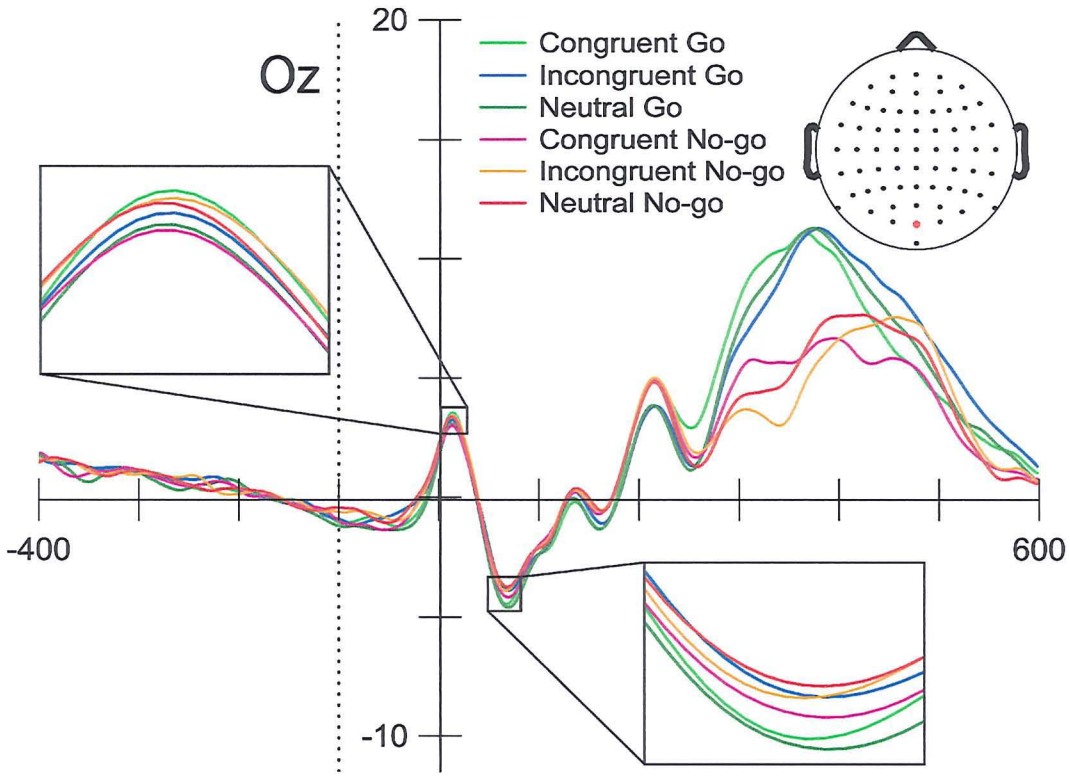

Figure 9.8: ERP waveforms at electrode Oz.

Figure 9.9 shows the difference waveforms at lateral occipital and occipito-parietal electrodes dependent of the physical stimuli. Visual inspection reveals that there appears to be no modulation of lateralised visual ERPs in response to the primes. ANOVA was conducted with prime type (left, right, neutral) and target type as repeated measures factors and response mapping an independent factor. ANOVA in the P1 time window revealed no significant effects. In the N1 time window there was a significant main effect of prime ($F(2,31.9)=5.2$, $p<0.05$). Subsequent contrasts revealed that ERP difference amplitudes were significantly reduced for neutral prime trials in comparison to left and right prime trials ($p<0.05$). There was no significant difference between left and right prime trials ($F=0.8$). There was no significant interaction between prime and prime type and response mapping. Additionally, there were no significant early prime-related effects for the PO7-PO8 difference. From around 180ms the lateralised ERP effects for both electrode pairs appeared to reflect target-related differences with increased activity contralateral to the direction of the target stimulus. This was confirmed by a main effect of target at occipital ($F(1,16=21.5$, $p<0.001$) and occipito-parietal electrodes ($F(1,16=18.1$, $p<0.001$).

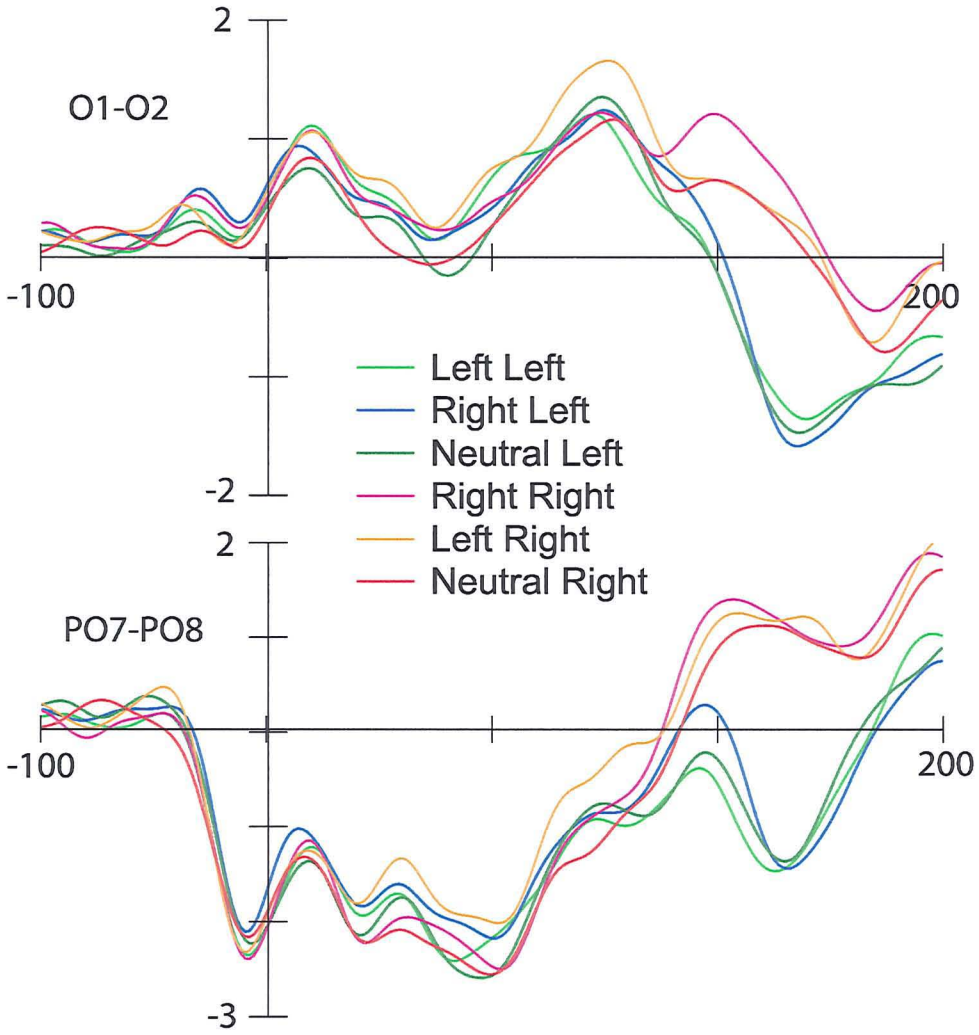

Figure 9.9: ERP difference waveforms for electrodes O1/O2 and PO7/PO8.

Go/no-go Differences

Figure 9.10 shows the scalp distribution of the ERP waveforms for experiment 5. Similar to the previous four experiments a frontal no-go N2 and P3 is clearly evident, maximally distributed over frontocentral electrodes. In addition, a parietal P300 modulation is also evident. Amplitude analysis of go/no-go differences was conducted using a five-way repeated measures ANOVA with prime type (go,no-go,neutral), target type (go,no-go), hemisphere (left,right), anterior-posterior (Fp, F, FC, C, P, and O) and time (120-180, 180-350, 350-550) as within-subjects factors. The early time window (120-180ms after target onset; 220-280ms after prime onset) was selected to explore any early differences in the ERPs associated with the unconscious primes. The second and third time windows were centred on the no-go N2 and no-go P3 respectively.

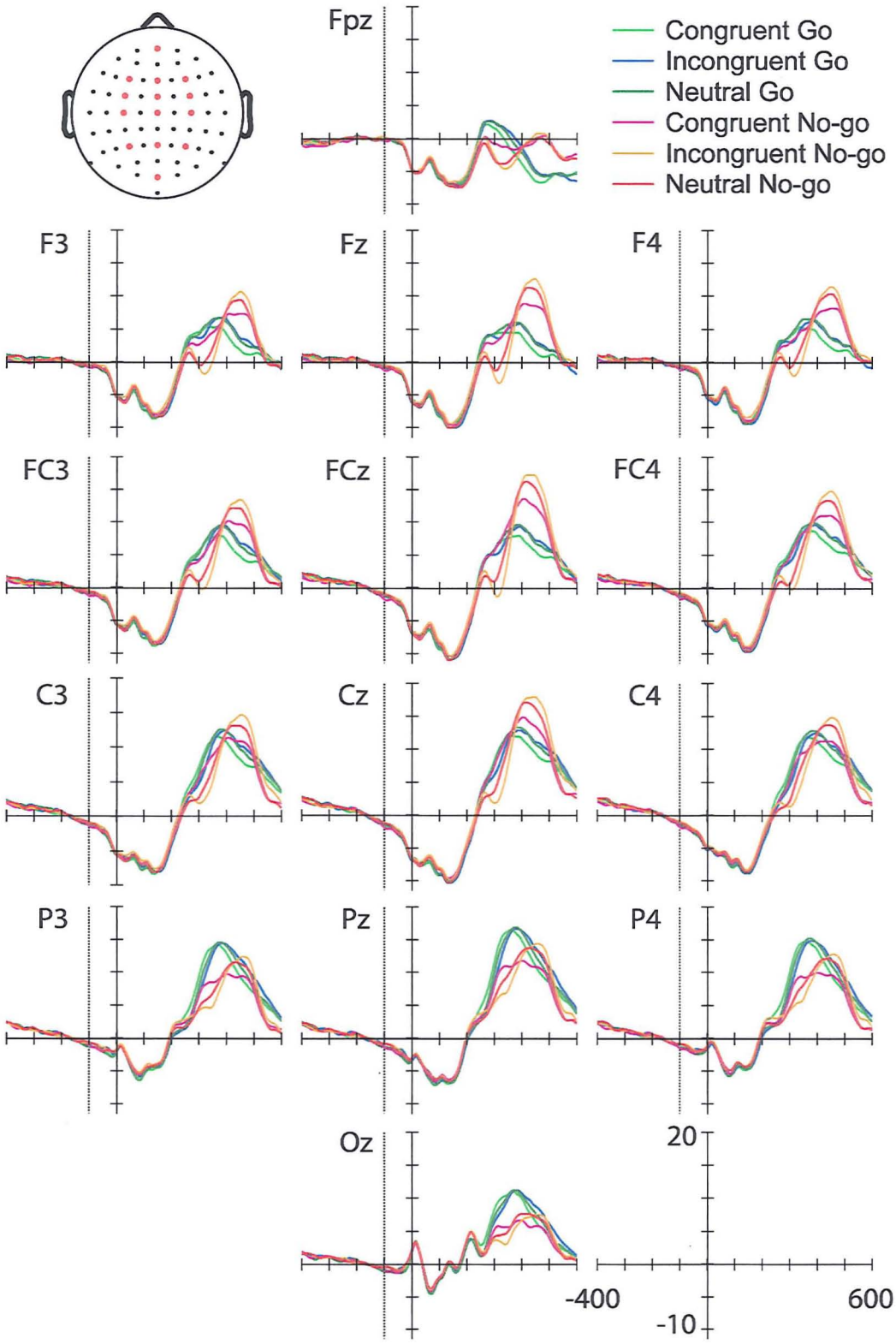

Figure 9.10: Grand average ERP waveforms for experiment 5.

The initial five-way ANOVA showed no main effects of hemisphere and no significant interactions involving hemisphere and target or prime type.

Therefore, further analysis was conducted on the six midline electrodes only (Fpz, Fz, FCz, Cz, Pz, and Oz). Both the ANOVA at lateral electrode sites ($F(4,76)=9$, $p<0.001$), and over the central electrode sites ($F(3.3,62.6)=10.3$, $p<0.001$) showed a significant four-way interaction between prime type, target type, time and anterior-posterior electrode location. This suggests that a different relationship between prime, target and anterior-posterior was evident for each time window. Therefore, further analysis explored the three way interactions between these variables separately for each time window.

In the first time window there were no significant effects involving either prime type or target type. In the second time window there was a significant main effect of target ($F(1,19)=17$, $p<0.001$) as well as significant prime x target ($F(1.3,25.3)=9.2$, $p<0.01$) and prime x anterior-posterior ($F(3.4,64.7)=5.4$, $p<0.01$) interactions. In addition, there was a significant prime x target x anterior-posterior x time interaction ($F(3.2,60.3)=7.7$, $p<0.001$). Figure 9.11 shows the average ERP amplitude in the middle time window for go and no-go trials separately. For go trials (left panel) the prime-related effects appear largest at posterior electrodes, while for no-go trials (right panel) they appear larger at anterior sites. Paired t-tests confirmed that for go target trials the only significant amplitude differences occurred at electrode Pz, between go and no-go prime conditions (at $p<0.001$). The same comparison was also marginally significant at Oz ($p<0.005$) with the neutral versus go prime conditions also approaching significance ($P<0.005$) at these two electrodes.

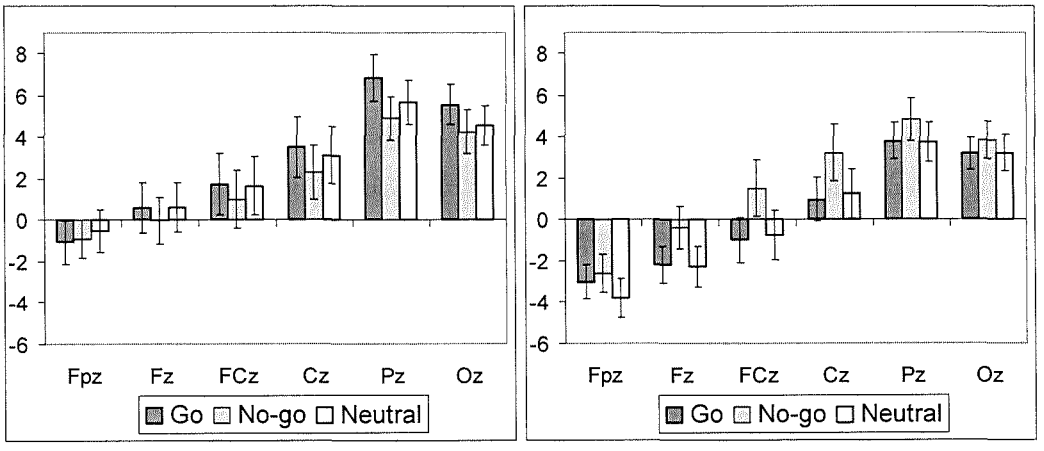

Figure 9.11: Average amplitude for midline electrodes in the middle time window dependent on prime type for go target trials (left panel) and no-go target trials (right panel)

For no-go target trials there was a significant difference (at $p<0.001$) between neutral prime trials and no-go prime trials at electrode FCz and Cz. This comparison also approached significance ($p<0.005$) at Fz and Fpz, with the comparison between congruent and incongruent trials also marginally significant ($p<0.005$) at Fz, FCz and Cz. These results confirm that the modulation of no-go target trials was maximal over frontocentral electrodes, and thus likely reflects modulation of a frontal no-go N2. Similarly the parietal modulation of go target trials likely represents modulation of P300 for these trials.

In the third time window there was a significant main effect of target ($F(1,19)=13.4$, $p<0.01$) as well as significant interactions between all pairs of factors and a 3-way interaction between prime type, target type and anterior-posterior electrode location ($F(3.4,64.7)=5.4$, $p<0.01$). Figure 9.12 shows the average amplitude dependent on target type along the midline electrodes for the late time window. At frontal electrodes no-go trials show greater amplitude in comparison to go trials, with the direction of this effect reversed over electrodes Pz and Oz. Contrasts revealed that there were significant (at $p<0.001$) differences between go and no-go target trials at each of the six electrode locations. This modulation reflects the anteriorisation of the P300 on no-go trials to cause the frontocentral no-go P3.

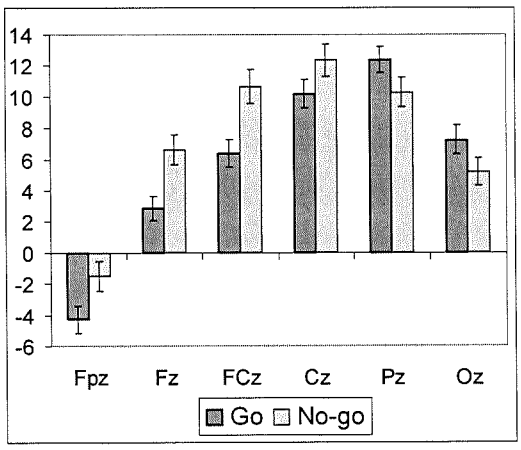

Figure 9.12: Average amplitude for midline electrodes in the late time window dependent on target type

Figure 9.13 shows that average amplitude in the late time window dependent on prime type for go and no-go trials respectively. For go target trials (left panel) there appears to be a small but fairly widespread effect of greater negative amplitude for go prime trials with a greater positivity for no-go prime trials also emerging at posterior electrodes. T-tests showed that the only significant differences (at $p<0.001$) were observed between neutral prime trials and go prime trials at electrodes Fz and FCz. For no-go target trials (right panel) go prime trials appeared to show greater positive amplitude with no-go prime trials showing the most negative amplitude. However, there were no significant pair-wise effects for no-go trials in this time window.

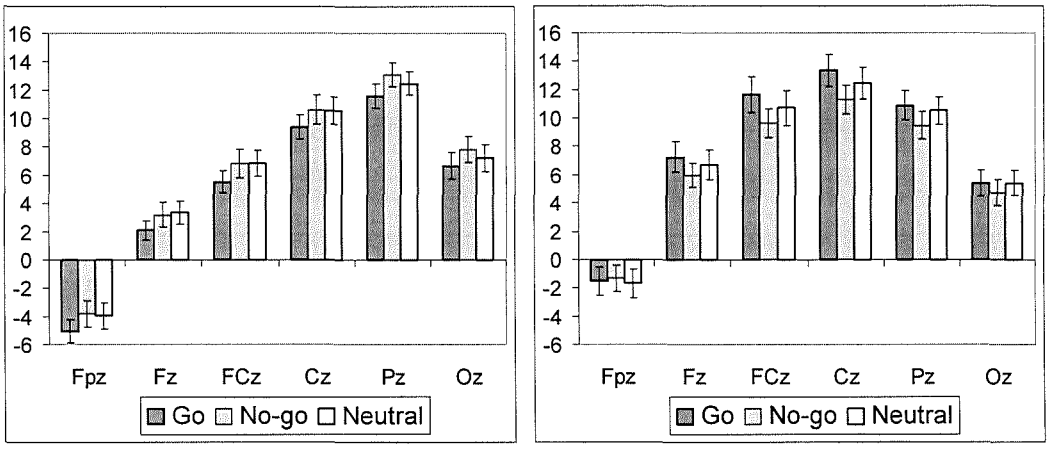

Figure 9.13: Average amplitude for midline electrodes in the late time window dependent on prime type for go target trials (left panel) and no-go target trials (right panel)

Priming and ERP go/no-go differences

Further analysis was conducted to explore the relationship between the amount of behavioural priming and the ERP effects. The current experiment observed a number of significant correlations between the behavioural priming and no-go N2 and P3 priming, which were not evident in the previous experiments. One possible reason for this is that there was a particularly large variation in the amount of priming observed in the current experiment. Table 9.3 shows the response congruency effect (RCE; congruent minus incongruent reaction times) for the 18 participants in the current experiment. It is evident that while there are some participants that show a large RCE, there are also many participants who showed very little priming. Participants were subsequently divided into large and small priming groups by taking a

median split. These groups had mean RCE of 55.19ms and 4.4ms respectively. Importantly, these two groups did not differ in terms of their performance on the prime identification task ($t(16)=1.4$, $p=0.19$), suggesting that the differences in the magnitude of the priming effects were not caused by greater awareness of the primes in the primed group.

Table 9.3: Response congruency effects for each participant

80	10	43	0	5	75	5	15	47	76	4	76	48	26	3	26	6	3

Figure 9.14 shows grand average ERPs for the primed and un-primed groups separately. Visual inspection reveals that, in line with the behavioural effects, the amount of ERP priming appears to be different between the two groups. While the ERP priming effects in the lower panel (behaviourally un-primed participants) are restricted to a reduced N2 for congruent no-go trials, there are much more widespread ERP effects for the participants that showed large behavioural priming (top panel). Both the N2 and P3 amplitude appear to be modulated for this group, with an early modulation dependent on prime type also evident.

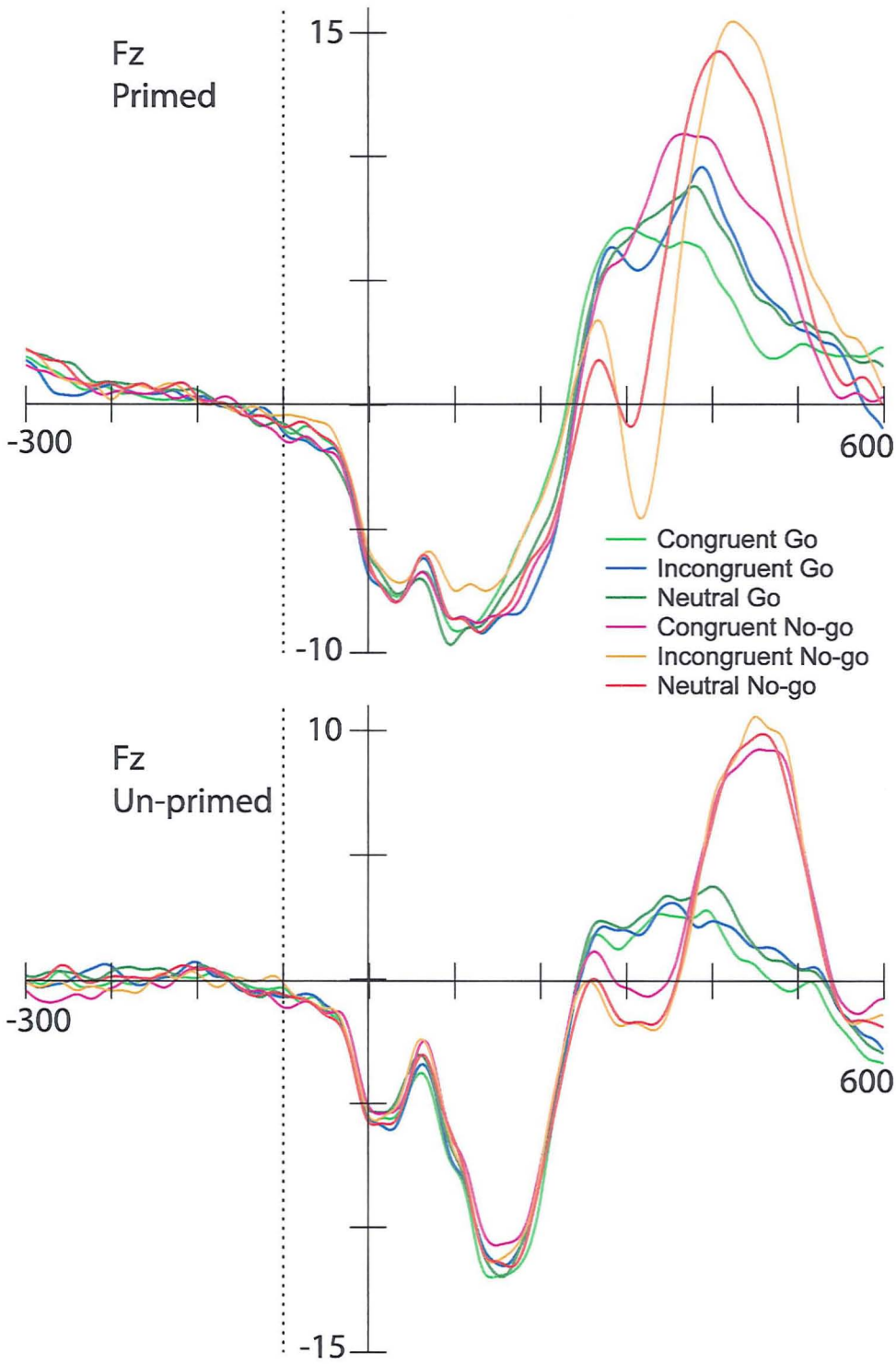

Figure 9.14: ERP grand average at electrode Fz for those participants showing behavioural priming (top) and those participants not showing behavioural priming (bottom).

Repeated measures ANOVA was conducted in the same three time windows as those above with priming added as a between groups factor. In an early time window there was a significant prime x priming group interaction

(F(1.9,31)=3.63, p<0.05). Separate ANOVA was conducted in this time window for primed and un-primed groups to explore this interaction in more detail. For the un-primed group there was no significant main effect of prime and no prime by anterior-posterior interaction. In the primed group there was no significant main effect of prime but there was a near significant prime x posterior interaction (F(2.8,22.5)=2.6, p=0.08). This finding suggests that the significant prime x priming group interaction reflects a difference in the amount of prime-related modulation in the ERPs, with a likely interaction with electrode site. Further analysis was conducted only on primed trials to explore this early prime-related modulation. Figure 9.15 shows the average amplitude for the three prime types in the early time window for primed participants. It is evident that the prime-related differences are largely driven by less negative amplitude for go trials in comparison to no-go trials, especially over frontal and central electrodes. In addition, no-go trials show consistently more negative amplitude than neutral trials at anterior electrode sites. Contrasts revealed significant differences (at p<0.05) between go and no-go primes at electrodes Fpz, Fz, and FCz, peaking at electrode Fz.

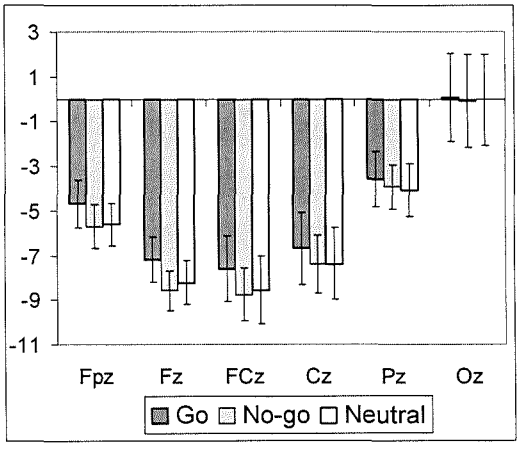

Figure 9.15: Average amplitude in the early time window dependent on prime type for primed participants.

In the second time window there was a significant prime x target x anterior-posterior x priming group interaction (F(3.8,60.1)=3.9, p<0.01). This suggests that the priming of ERP components in this time window was different between the two groups. Separate ANOVA for each group confirmed that the only effect to approach significance for the un-primed group was a main effect of target (F(1,8)=4.2, p=0.07), whereas for the

primed group there was a significant main effect of target ($F(1,8)=11.2$, $p<0.01$) as well as a prime x target interaction ($F(1.2,9.2)=10$, $p<0.01$) and a target x prime x anterior-posterior interaction ($F(2.9,22.9)=9.7$, $p<0.001$). Although the main effect of target failed to reach significance for the not-primed condition it is important to note that the target x priming group interaction was not significant. Therefore, the magnitude of the target-related effects in the second time window were not different between these two groups. However, the significant interaction 4-way interaction suggests that priming of ERP components in this time window was different in the two groups. The presence of target x prime and target x prime x anterior-posterior interactions for the primed conditions suggests that the priming effects were larger in this group. Figure 9.16 shows ERP amplitude dependent on prime type for go and no-go target separately for the primed participants and un-primed participants. Comparing the top panels with the bottom panels reveals that priming of both go and no-go response is much greater for primed trials. As for the analysis with all participants together, no-go priming was maximal at frontal electrodes while go priming was maximal at parietal electrodes. Paired t-tests for go targets confirmed significantly increased amplitude at Oz and Pz for go prime trials in comparison to both neutral and no-go prime trials for primed participants only (at $p<0.005$). Similarly no-go target modulation only occurred for the primed group, where no-go prime trials were more negative than go and neutral prime trials at electrodes Fz, FCz and Cz (at $p<0.01$).

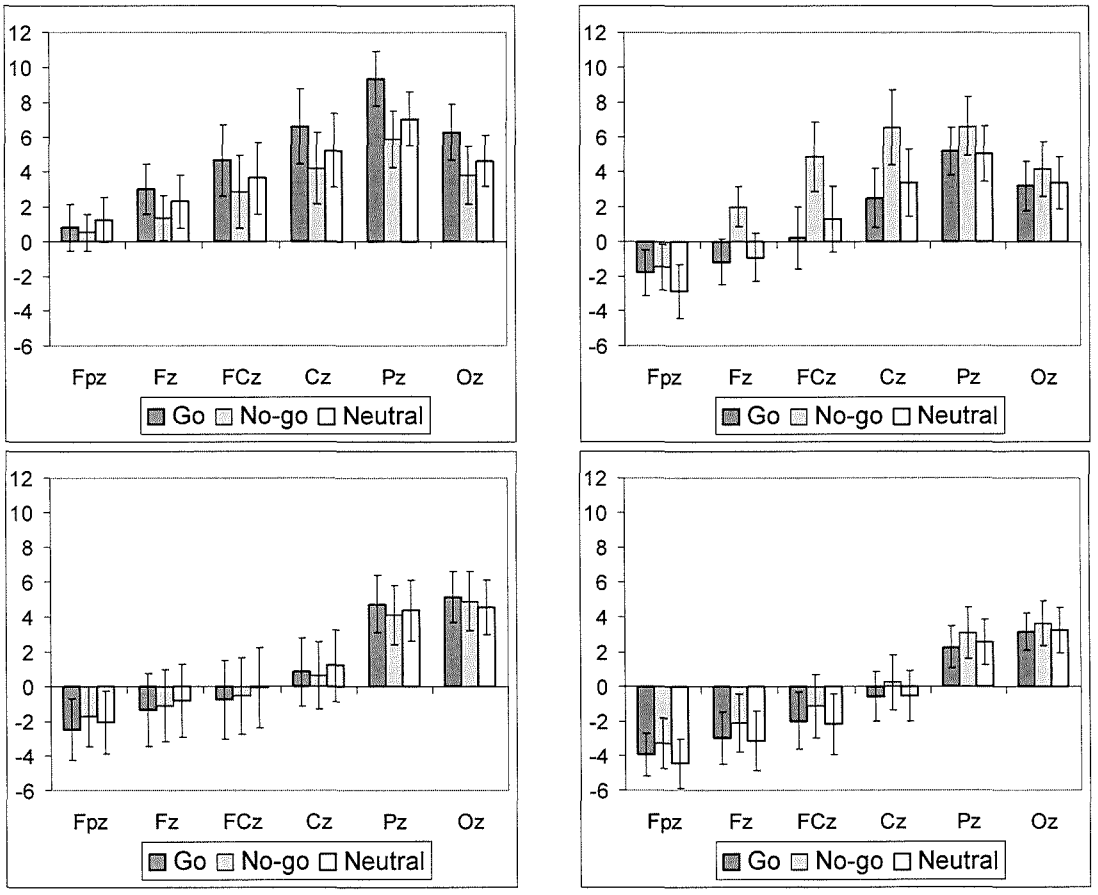

Figure 9.16: Average amplitude in the middle time window dependent on prime type for go target trials (left panels) and no-go target trials (right panels) and for primed participants (upper panels) and un-primed participant (lower panels).

In the third time window there was a significant target x prime x priming group interaction and a near significant target x prime x anterior-posterior x priming group interaction. Once again these results suggest a difference in the magnitude of ERP priming effects for the primed and un-primed groups. Subsequent analysis confirmed that these effects were similar in nature to those observed in the middle time window with significant prime x target ($F(1.8,14.5)=26$, $p<0.001$) and prime x target x anterior-posterior ($F(2.9,23.5)=5.5$, $p<0.01$) interactions for the primed group only. This reflects increased priming of the frontal no-go P3 and parietal P300 components for primed participants only.

In summary, the between participants analysis separating those participants with a large behavioural effect from those with a small or no behavioural effect confirmed the relationship between behavioural priming and ERP

priming observed in this and previous experiments. While this effect is not unsurprising, it is important to show that the ERP priming effects are genuinely caused by the experimental manipulation. The observed correlation between N2/P3 priming reported in an early section already pointed towards this relationship. However, the additional finding of increased early frontal modulation dependent on prime type for the primed participants provides further evidence that these frontal mechanisms are directly initiated by the unconscious primes, and that they are related to the degree to which the unconscious primes influence behaviour. Importantly these effects were unrelated to visibility of the primes, as measured by performance in the prime identification task.

Interestingly reaction times on neutral go trials were significantly faster ($t(16)=2.4$, $p>0.05$) for the primed group (m=364, std=15) than the un-primed groups (m=378; std=13.5), suggesting perhaps that the primed group were generally more focused on the task. This improved reaction time did not simply reflect a speed/accuracy trade of for these participants, since they also showed marginally greater accuracy for these trials (0.96 versus 0.92; $t(16)=2$;$p=0.076$). This explanation is supported by the observation that the pre-stimulus negativity appears somewhat larger for primed participants than un-primed participants (see figure 9.14), suggesting that anticipation of the upcoming stimulus and the need to respond was greater for the primed participants. However, ANOVA at electrode Fz from -300 to -250ms showed that ERP amplitude for primed participants (m=2.9, std=2.6) was not significantly more positive than ERP amplitude un-primed participants (m=1.4, std=2.6; $F(1,16)=2.8$,$p=0.11$).

Discussion

The current experiment aimed to show unconscious modulation of frontal inhibition-related ERP components in a paradigm that produces positive compatibility effects. While the experiment replicated the modulation of the no-go N2 and P3 components observed in the previous experiments, initial analysis of all eighteen subjects together revealed only modest early frontal

ERP separations. However, subsequent analysis showed that, not only was such modulation present for a subset of participants, but that both this early modulation and later target-related ERP modulation was associated with participants' behavioural performance.

Behavioural analysis revealed that the stimuli utilised in the current experiment were successful in producing a positive compatibility effect. Reaction times were significantly reduced for congruent go trials, and significantly increased for incongruent go trials in comparison to the neutral baseline. Importantly, the magnitude of this effect was more comparable to that observed in experiment 1, than in experiments 2 to 4, with an average RCE of twenty-eight milliseconds. This suggests that reverting back to the type of stimuli used in experiment 1 was successful in increasing the behavioural priming effect. There are a number of possible reasons for this increased behavioural priming effect. Firstly, only in experiments 1 and 5 were the primes identical to the targets. In all other experiments the primes were somewhat modified versions of the targets and were often presented in a different location to the target. Therefore, if participants set up a stimulus-response mapping for particular stimuli, it is likely that the closer the prime is to the original S-R mapping the more it will influence behaviour. Additionally, in experiments 2 and 3 the stimulus response mapping was much more complicated, and involved combining information about objects in different spatial locations in order to produce an appropriate response. This increased complexity might serve to reduce the influence of the unconscious primes, since they will also have a less clear S-R mapping. This issue will be discussed in more detail in chapter 10 with reference to the general mechanisms of subliminal priming. Finally, as discussed in the previous chapter, the primes in experiment 4 were likely below the threshold for prime detection, likely reducing their efficacy in priming the no-go response.

Despite this increased behavioural priming effect in the current experiment, it still failed to show ERP effects of a comparable magnitude to those observed in experiment 1. LRP analysis did reveal an early modulation dependent on prime type with no-go prime trials showing increased positivity and go prime

trials showing increased negativity immediately prior to the rising bank of the LRP. However, this effect was only significant when the LRP was baselined to 150 to 100ms prior to stimulus onset. This was conducted due the suspected presence of noise in the pre-stimulus LRP for the congruent go condition. These pre-stimulus differences could not be the result of any stimulus related effects since the trial order was random in each block for each participant. As discussed in chapter 2, the LRP has a particularly low signal to noise ratio. In the current experiment the LRP was formed from an average of 60 responses per hand, with sometimes as little as 40 responses. Although, ideally, LRPs would be constructed from a larger number of trials this was not possible in the current experiment (or in experiments 2 to 4) if all conditions were to be included in a single experimental session. Since the LRPs in the current experiment appear to be rather noisy, the result of prime-related effects must be treated with some caution.

Visual ERP effects were also significantly reduced in comparison to previous experiments. Analysis at electrode Oz revealed no significant effects of prime or target in the early visual ERPs. Similarly only relatively modest lateralised visual ERPs were observed in response to the subliminal primes in the current experiment. Furthermore, these effects were limited to electrode the O1-O2 comparison and were not present at for the PO7-PO8 difference. In addition there were no significant interactions evident with response mapping. These findings suggest that the small lateralised occipital ERP effect reflected a visual rather than attentional modulation dependent on the functional significance of the target such as the N2pc (see chapter 2). Since the precise stimulus response mappings were counterbalanced across participants these lateralised ERPs were formed dependent on the physical stimuli and therefore contained a mixture of different possible responses. This meant, for example, that left prime left target ERPs were calculate by combining congruent go trials for those participants who were asked to respond to a left pointing arrow, and congruent no-go trials for those participants who were asked to respond to a right pointing arrow. Therefore any modulation of these lateralised ERP effects related to the more functionally salient stimulus (the go stimulus)

should result in an interaction with response mapping. Therefore it is likely that these effects reflect either modulation of early visual ERP components or attentional modulation that is independent of the functional significance of the targets. Later target-related ERP asymmetries were also evident, with increased amplitude contralateral to the target stimulus. Once again this likely reflects low level visual/attention related effects of the target stimulus.

As in all previous experiments, the amplitude of the no-go N2 and P3 were modulated as a function of the unconscious prime. In the current experiment, this was evident both in the comparison between no-go primes and neutral primes and the comparison between go primes and neutral primes. This suggests that the modulation of the no-go N2 and P3 is not just an effect of increased engagement of frontal inhibition/control mechanisms in response to a primed motor response. More specifically, the finding that the N2 and P3 are reduced in amplitude for congruent no-go trials in comparison to neutral no-go trials suggests that the no-go response is facilitated by unconscious no-go primes in comparison to a neutral baseline. While this effect was limited to the no-go N2 in the previous experiment, it was evident on both N2 and P3 components in the current experiment. Strikingly, the no-go N2 appears to be almost completely abolished in the congruent no-go condition in the current experiment. As in all previous experiments there was again a difference in the modulation of go and no-go target trials, with no-go trials showing a frontal modulation of the no-go N2 and P3 and go target trials showing modulation of the parietal P300.

In addition to these prime-related effects in response to the target stimuli, a small early separation was also evident that was entirely dependent on prime type. However, this separation was not statistically reliable. An early prime-related effect would signify the direct engagement of frontal inhibition/control mechanisms by the unconscious primes. Inspired by the finding of a significant correlation between behavioural priming and ERP priming in the current experiment, further analysis was conducted to explore how the ERP priming effects differed between those participants that showed a large behavioural priming effect and those who showed little or no effect.

Inspection of the RCEs for each participants revealed that while some participants produced particularly large priming effects, in others the subliminal primes had little or no effect. ERP waveforms were formed separately for these two groups with ERP analysis repeated adding piming group as a between participants factor. This analysis revealed significant interactions between prime type and priming group in the first time window and between prime type, target type and priming group in the second and third time windows. These findings further confirmed that the ERP priming effects were strongly associated with the behavioural priming effects. In particular, the finding that the degree of frontal prime-related ERP modulation in the early time window was significantly greater for the primed group strongly suggests that the subliminal primes in this group were able to directly influence frontal control mechanisms, leading to facilitation of behavioural performance. This facilitation is further manifested in the increased modulation of N2 and P3 amplitude for this group, showing that the subliminal primes not only facilitated this early separation, but that this was associated with an increased later priming effect.

Importantly, this modulation of ERP and behavioural priming appears to be independent of awareness of the primes. If a number of participants had some residual awareness of the primes, this would likely increase the magnitude of the priming effect. However, there was no significant difference between the primed and un-primed groups on the prime identification task, suggesting that both groups were completely unaware of the masked primes and that the variation in priming was not due to differences in visibility of the primes between participants. An alternative explanation for this differential priming effect is that increased priming was associated with improved performance on the task. This is supported by the finding that primed participants showed significantly faster reaction times than un-primed participants, as well as marginally increased accuracy. Similarly the observation that the pre-stimulus CNV type activity appears to be slightly higher for the primed group (although not significant) suggests that that they were in a heightened state of anticipation for imminent stimulus presentation (cf. Luck, 2005). If these participants are more focused on

making a fast response and more attentively looking to identify the stimulus as quickly and accurately as possible, it seems likely that they will be more influenced by an arrow prime, even if this prime is presented below the threshold of awareness.

Another important observation in this final analysis is that the no-go P3 appears to onset earlier for the congruent no-go condition. In the combined ERPs presented earlier in the chapter this looked more like a straightforward N2 amplitude modulation. While the spatial overlap of these components make it impossible to distinguish which of these two explanations is the correct one, it is important to note that the apparent abolition of the no-go N2 might be masked by a somewhat earlier P3. It is perhaps somewhat surprising that the normal no-go N2 was completely abolished in the congruent no-go condition, especially considering the absence of early prime-related effects in the initial analysis – if no frontal control/inhibition mechanisms are engaged then how is the response inhibited? If, as suggested, the no-go P3 onsets somewhat earlier for the congruent no-go condition, this would remove any remaining effect of a no-go N2 in this time window. These findings highlight the difficulty in separating spatially overlapping ERP components. A similar problem is evident in the earlier frontocentral prime-related separation. As discussed in earlier chapters, negativity for no-go primes in this time window is the opposite of what would be predicted by motor priming alone, since the readiness potential is a negative wave and thus an increased negativity should be present for go trials. Therefore, the increased motor activation for go trials in this time window will reduce the predicted separation, and in some cases may mask it completely. These overlapping motor components likely explain why the early N2 modulation appears somewhat less sensitive than the later N2/P3 modulation. However the presence of this effect in a number of experiments, despite this complication, strongly supports the claim that frontal inhibition/control mechanisms can be directly initiated by a subliminal prime.

Finally, the current experiment also provides some new insight into the functional significance of the no-go N2 and P3. While previous studies have

failed to show modulation of these components in a predicted manner when the degree of engagement of frontal inhibition/control mechanisms are manipulated (Smith et al., 2007), the current series of experiments shows that both the N2 and P3 are modulated when inhibition/conflict is increased or decreased by subliminal primes. The association of this modulation with behavioural priming in the current experiment strongly supports the claim that the N2/P3 complex reflects engagement of frontal inhibition and control mechanisms. The no-go behavioural priming effect, signified by the difference between a congruent no-go and a neutral no-go trial, was significantly associated with the same contrast in P3 amplitude modulation and not N2 amplitude modulation. While this suggests that perhaps the P3 is directly involved in no-go priming, the widespread correlations observed with the no-go N2 suggest that this component is also important in priming of go and no-go responses in the go/no-go task. In addition, as discussed above, the spatial overlap between these components makes it difficult to completely disentangle them.

Conclusions

The current experiment provides perhaps the strongest evidence that unconscious masked primes were able to directly initiate frontal inhibition/control mechanisms. Modulation of the target-related N2 and P3 was observed such that congruent no-go trials showed significantly reduced N2/P3 responses in comparison to a neutral baseline, while incongruent no-go N2/P3 amplitude was increased in comparison to neutral trials. This finding confirms that modulation of the no-go N2 and P3 includes facilitation of the no-go response and not just interference caused by an unconscious go response. Furthermore, the current experiment showed that both this later target-related modulation and early prime-related separation were significantly associated with behavioural performance, confirming that facilitation of the no-go N2 is associated with priming of the no-go response.

Chapter 10

General Discussion

Outline

This chapter will present a general discussion of the research conducted in this thesis. The first part of this chapter will include a brief review of the aims and objectives of the current investigation. The second part will be organised into sections dependent on the different results obtained for the five experiments. This will include a brief reminder of relevant previous research and a summary of the results obtained in each of the five experiments and their relation to previous results. The final part of the chapter will address the importance of the current findings in relation to the literature outlined in the first three chapters as well as presenting a critical review and presenting possible avenues for future research.

Background to current research

The first three chapters of this thesis outlined a number of experiments that have shown a strong association between consciousness and frontal inhibitory/control mechanisms. This association was evident from Libet's early work exploring the role of consciousness in preparing a voluntary motor act as well as in more recent experiments which measured brain activity in response to conscious and unconscious conflicts (cf. Mayr, 2004). Libet et al. (1983) found that while the brain appears to begin preparation for a voluntary action some 300ms before conscious awareness of the decision to move, consciousness may still have a role in either vetoing or allowing the continuation of the action.

A similar conclusion is also evident in the parallel research area of subliminal priming. Leuthold and Kopp (1998) showed that motor responses can be initiated in response to a subliminal stimulus, a finding which has been supported by a number of other research groups (Dehaene et al., 1998; Eimer & Schlaghecken, 1998). While motor preparation has been shown to

be initiated by an unconscious stimulus, the conflict induced by two different response alternatives was not sufficient to induce frontal cognitive control mechanisms. Dehaene et al. (2003) showed that while anterior cingulate cortex (ACC) activity was increased in response to incongruent primes that were consciously visible, unconscious primes did not produce the same ACC activation. Similarly, Praamstra and Seiss (2005) showed that response conflict produced by the negative compatibility effect (NCE) was not mediated by frontal control mechanisms, as indexed by the absence of a frontal N2 ERP component in response to such unconscious conflict. Mayr (2004) reviews a number of other studies exploring ACC activation in response to conscious and unconscious conflicts, and concludes that ACC-related control processes are closely associated with conscious but not unconscious conflict.

While it is clear that there is a strong association between consciousness and activity in ACC, related to conflict monitoring and behavioural control, it is not clear from previous research whether voluntary inhibition of a motor response can occur in response to an unconscious stimulus. This is an important consideration with regard to Libet's (1985) suggestion that the function of consciousness is to allow or prevent motor behaviour from being executed. If, like preparation of a motor response, inhibition of an imminent response can be initiated unconsciously then this casts doubt over the role of consciousness in guiding behaviour. Eimer and Schlaghecken (2003), inspired by the findings that response conflict is strongly associated with consciousness suggest that:

"*endogenous* inhibition is voluntary, optional, and is presumably mediated in prefrontal cortex. Since endogenous inhibition depends on the conscious detection of task-relevant signals, it is not available when stimuli are presented subliminally". Eimer and Schlaghecken (2003; page 8).

Eimer and Schlaghecken (2003) have suggested that this type of inhibition can only occur in response to a conscious stimulus, despite the absence of direct evidence in support of this claim. The current research directly

explored this claim and aimed to determine whether inhibition of an imminent response can be initiated by a stimulus that is not available to consciousness. In a series of experiments, participants were asked to complete a go/no-go task, where they were required to make a response to a go stimulus and withhold their response to a no-go stimulus. Unbeknownst to the participants, unconscious masked primes were presented in advance of the target stimulus. These primes were congruent, incongruent or neutral with respect to the target stimulus. If the unconscious masked primes are able to influence the go/no-go decision then a congruent go prime should produce smaller reaction times, while a congruent no-go trial should produce improved accuracy in comparison to neutral and incongruent go trials.

In addition to these behavioural priming effects, the research also investigated the influence of unconscious primes on ERP markers of the no-go response. As outlined in chapter 2, no-go trials in the go/no-go task have been associated with an N2/P3 complex, such that no-go trials show an increased negativity over frontocentral electrodes beginning around 200ms after stimulus onset. Immediately following this negativity, no-go trials exhibit an increased positivity over frontocentral electrodes, termed the no-go P3. While the exact functional significance of each of these components is still under intense debate, the N2 has been associated both with active inhibition of a motor response as well as monitoring of response conflict (Falkenstein, 2006). Similarly, while the no-go P3 consistently appears following no-go trials, it normally occurs too late to be actively involved in inhibition of the response (Falkenstein et al., 1999), leading some people to suggest that it reflects the outcome of the inhibitory process measured in the primary motor cortex (Dimoska et al., 2006). Despite this uncertainty, there is a consensus that these no-go ERP components together reflect frontal inhibition/control mechanisms partly originating from the ACC, with another likely source in the pre-frontal cortex (Falkenstein, 2006).

Previous reports exploring the modulation of these ERP components have focused on their role of frontal control functions in mediating response conflict, such as that induced in a choice reaction time task (Dehaene et al.,

2003; Leuthold & Kopp, 1998). In this task, these ERP correlates of control functions are selectively elicited on incongruent trials on which a response conflict is present. Since these components are often absent when response conflict is unconscious (Dehaene et al., 2003; Mayr, 2004), it is often assumed that these frontal control processes can not be engaged unconsciously.

The current research used a somewhat different approach by employing a task where these mechanisms are engaged in order to enable inhibition of an imminent motor response. Presenting unconscious primes in advance of the go/no-go target stimuli would allow exploration of the degree of engagement of these mechanisms dependent on the nature of the unconscious information. For example, on an incongruent no-go trial, a greater engagement of frontal control mechanisms should be required to overcome the primed motor response. In contrast, on congruent no-go trials, no-go N2 and P3 amplitude should be reduced, reflecting the fact that the unconscious primes were able to facilitate inhibition of the response.

Recording ERP correlates of the no-go response allowed a more detailed investigation of the mechanisms by which the unconscious primes might exert their influence on behaviour, compared to using purely behavioural measures. For example, if behavioural results show that the subliminal primes influenced performance on the go/no-go task this could be due to a number of different reasons. Firstly, since there is much evidence that motor preparation can be initiated unconsciously, the go/no-go effects might be mediated within the motor cortex, without the need to elicit frontal control mechanisms. Alternatively, reaction times and error rates may be affected via a simple perceptual priming effect such that participants are quicker to classify the target stimulus when the preceding prime is congruent (this is discussed in more detail below). Finally, the unconscious primes could exert their influence by directly initiating the no-go response. The use of ERP measures of go/no-go differences allowed disentangling of these different possibilities. Leuthold and Kopp (1998) used ERPs in a similar way to show that unconscious primes could directly program a motor response. They

showed that the LRP produced an initial activation dependent on the subliminal prime, suggesting that despite being unconscious the masked primes were able directly specify a hand specific motor response. In the current series of experiments ERP activity over frontocentral electrodes was utilised in a similar manner to determine if unconscious primes were able to directly initiate frontal inhibition/control mechanisms. If these processes are directly elicited by the primes, then an early ERP separation should be evident over frontocentral electrodes that is entirely dependent on the prime information. More specifically, if the unconscious primes are able to directly elicit the frontal no-go N2 then an early negativity should be observed following a no-go prime and not following a neutral prime or a go prime.

Overview of experimental results

This section provides an overview of the results from the five experiments reported in this thesis. Experiment 1 explored ERP responses to unconscious primes in a go/no-go version of Eimer and Schlaghecken's (1998) masked priming experiment. This particular paradigm was known to produce a negative compatibility effect such that incongruent primes facilitate responses and congruent primes impede responses. Much of the aim of the subsequent experiments was to replicate the findings of this first experiment in a situation that produced a positive compatibility effect. Although each experiment showed some behavioural priming effects, there were a number of complications with the neutral primes in some experiments, and a number of effects observed in experiment 1 were not consistently replicated. The following section provides a summary of the results. Rather than reviewing the results one experiment at a time, this section will deal with each of the main results observed in these experiments, on each occasion comparing these effects across experiments and relating this to previous research.

Awareness of primes

In each experiment visibility of the masked primes was assessed using both subjective and objective measures. Participants were asked a number of

questions regarding the visibility of the primes. In addition, they completed a prime identification task which required them to report on each trial, which of two possible prime stimuli had been presented. In experiments 4 and 5 prime identification performance was not significantly different from chance. In experiment 1, despite being subjectively unaware of the masked primes a number of participants performed significantly above chance. Similarly, in experiments 2 and 3 performance on the prime identification task was significantly above zero. In each of these experiments, to ensure that any effects of the masked primes were unconscious each of the major effects was correlated with participants' performance in the prime identification task. If some residual awareness of the primes was evident on the prime identification task, then any priming effects could simply be due to this residual awareness. In this case, performance of the visibility task should correlate with the amount of priming in the go/no-go task, since the greater degree to which the participant could see the prime the greater the priming effect is likely to be. Finally, to ensure that the priming effects were still present when prime identification was at chance level, analysis was repeated excluding those participants showing slightly elevated performance on the prime identification task. In each experiment where there was evidence of possible residual awareness of the primes, each of these calculations showed no association with the amount of priming observed in the go/no-go task, confirming that the priming of responses was independent of awareness of the primes.

Behavioural results

As predicted, experiment 1 produced a negative compatibility effect with congruent go trials showing significantly increased reaction times in comparison to neutral and incongruent trials. In addition, accuracy was greatest for incongruent trials and smallest for congruent trials. The response congruency effect (RCE) between congruent and incongruent go trials in this experiment was large at -58ms. In each of the following experiments a positive compatibility (PCE) effect was observed. In experiment 2, however, there was no significant difference between incongruent go and neutral go reaction times, or congruent no-go and neutral

no-go error rates. These findings suggest that in this experiment the neutral primes grouped together with the no-go primes. Since the task required participants to respond to a diamond on one side of the screen, a possible explanation suggested for this effect was that participants selectively attended to the side of the screen where the diamond stimulus would appear. Since neutral and incongruent primes only differed on the side opposite to where the diamond was presented, this suggested attentional focus meant that neutral and no-go primes were effectively identical. The RCE between congruent go and incongruent go trials was smaller at 19ms.

Experiment 3 aimed to overcome the problems with the neutral primes that was found in experiment 2, and also reverted to a task where the participants were required to press either a left or right button in response to specific go stimuli and withhold a response to no-go stimuli. This allowed exploration of trials with response competition (incongruent go trials) alongside trials with a no-go prime (no-go go condition). All pair-wise comparisons were significantly different from one another for reaction times, suggesting that the neutral primes were producing neutral priming effects, such that they did not appear to facilitate or interfere with go or no-go responses. The RCE between congruent go and incongruent go trials was 27ms and between congruent go and no-go go trials was 22ms. However, further analysis revealed that while some neutral trials appeared to prime a right hand response, other neutral trials primed a left hand response. More specifically, when a neutral prime appeared on the left it acted as a left-go prime, while a neutral prime on the right acted as a right-go prime. This meant that neutral primes effectively acted as go primes, most likely because they contained diagonal features that were present in go stimuli. When averaging the different types of neutral trials together, the overall reaction times were significantly different from both congruent and incongruent trials, giving the impression that they were genuinely neutral. Due to this confound with neutral trials, they were excluded from further analysis.

Experiment 4 reverted back to a simple go/no-go task where participants responded to a central arrow pointing either left or right. This experiment

produced a positive priming effect, with congruent go trials showing significantly reduced reaction times in comparison to neutral go trials, while incongruent trials showed slowest reaction times. All pair-wise comparisons for both reaction times and accuracy were significant. However, the RCE for this experiment was extremely small (just 7 ms), suggesting that the primes had only a small effect on go/no-go behaviour. A likely explanation for this small priming effect is that the masked primes were presented either side of a central location, and therefore in order to successfully identify the prime participants simply had to detect its presence or absence on one side or the other. Since the prime detection threshold is lower than the prime identification threshold, the primes were significantly weaker in this experiment. The finding that even at this strict objective threshold, go/no-go priming was maintained suggests that the effect is truly unconscious. Experiment 5 reverted back to similar stimuli to those utilised in experiment 1, using different masks to ensure a positive rather than negative compatibility effect. This experiment produced the greatest response congruency effect of all the experiments except experiment 1 with a congruent versus incongruent reaction time difference of 28ms. In addition to this large reaction time RCE, error rates followed a positive compatibility effect for both go and no-go target trials.

In summary, all five experiments produced significant modulation of behavioural responses as a function of the primes, suggesting that despite being presented below the threshold of awareness they were able to influence the go/no-go decision. It is interesting to note that there was a great deal of variation in the RCE across the five experiments. The greatest RCE was observed in experiments 1 and 5. One possible explanation for the increased RCE in those experiments is that the primes and targets were perceptually identical. If participants have built a stimulus-response association for a specific target, and are looking out for that target in the visual presentation, it is likely that the closer the prime to the target stimulus, the more likely it is to activate the stimulus-response association. Moderate RCEs were observed in experiments 3 and 4. In these experiments, the primes were small versions of the targets, and therefore differed somewhat

in their precise shape. The finding that a modest RCE was observed in these experiments shows that the priming of no-go responses is not restricted to identical primes. Finally, the small but significant RCE observed in experiment 4 was attributed to the fact that in this experiment primes were presented either side of fixation. This meant that the primes needed to be particularly weak in order to be below the objective threshold for prime identification, since identification could be achieved by simply looking out for a flash either side of fixation, making the prime identification task more like a prime detection task.

LRP

Experiment 1 showed a clear early separation of LRP waveforms dependent on the unconscious primes, with a direct activation of LRP following a go prime quickly replaced by LRP activation associated with a go prime-mask effect, replicating the effects of Eimer and Schlaghecken (1998). This effect of direct specification of a particular hand response was replicated in experiments 2 and 5 which showed increased LRP activation in response to a go prime. In experiment 3, the LRP became contaminated by confounding, lateralised visual effects, consequently the effect of the unconscious primes on this component could not be explored. In experiment 4, there were no consistent effects of the unconscious primes on LRP activation. Nevertheless, despite these problems with experiments 3 and 4, the clear results from the other three experiments show that motor responses can be initiated unconsciously.

No-go N2 and P3

All five experiments showed some modulation of the no-go N2/P3 complex in response to the unconscious primes. In contrast to previous research exploring the N2 in response conflict situations (Dehaene et al., 2003; Mayr, 2004; Praamstra & Seiss, 2005), this shows that modulation of frontal inhibition/control mechanisms in response to unconscious masked primes in the go/no-go task is a highly replicable effect. It is important to note that in experiment 1 the predicted modulations of no-go N2 and P3 components by the prime were the opposite of those predicted in experiments 2 to 5. Since

experiment 1 produced a negative compatibility effect, unconscious facilitation of frontal inhibition/control mechanisms should result in a reduced N2 and P3 for the incongruent no-go condition. In line with this prediction, N2 and P3 amplitude was significantly increased for congruent no-go trials, and decreased for incongruent no-go trials. Moreover, an early frontal negativity was evident for incongruent no-go and congruent go trials. This negativity appeared to reflect an early N2 in response to a no-go prime-mask effect. Prior to this negativity related to the prime-mask effect, activity at frontal electrodes was more positive for go primes in comparison to neutral and no-go primes for both go and no-go target trials. Importantly, both these early modulations were entirely dependent on the nature of the prime stimulus, suggesting that they reflect N2-related activity directly elicited by the unconscious primes. Moreover, the topography of these effects, with a frontocentral maximum suggests that they reflect modulation of the no-go N2 component. This finding is analogous to Leuthold and Kopp's (1998) LRP experiment which showed direct specification of a motor response by an unconscious prime, suggesting that like a motor response, inhibition of an imminent response can also be initiated unconsciously. However, given the rather unusual nature of the direction of the priming effect using these precise stimuli – namely an NCE rather than a PCE – the following experiments aimed to replicate this effect in an experiment that produced positive compatibility.

While experiments 2 to 5 replicated the modulation of later target-related N2 and P3 components, only experiments 2 and 5 showed any significant early prime-related modulation. In each of experiments 2 to 5 target-related N2 amplitude was significantly reduced for congruent no-go trials in comparison to incongruent no-go N2 amplitude. Experiments 4 and 5 also showed a significant decrease in target-related N2 amplitude for congruent no-go trials in comparison to neutral no-go trials. However, comparisons between congruent and neutral trials were not possible in experiments 2 and 3 due to potential confounds with the neutral primes in these experiments. In experiment 5 there was also significant modulation of the no-go P3, with congruent no-go trials revealing significantly reduced amplitude and

incongruent no-go trials showing increased amplitude in comparison to neutral no-go trials. This modulation of target-related N2 and P3 components is important as it shows a facilitatory effect for congruent trials and not simply an interference effect for incongruent trials. Since it is well known that unconscious primes can directly initiate a motor response it is perhaps not surprising that N2 amplitude is increased following a go prime, since the go response will become partially activated and will require greater engagement of frontal control mechanisms to be successfully withheld. However, it is also noteworthy that such modulation of N2 was not evident in response conflict tasks where the source of the conflict remains unconscious. In particular both Leuthold and Kopp (1998) and Praamstra and Seiss (2005) found that although a subliminal prime was able to directly specify a specific hand response, there was no genuine N2 effect following an incongruent trial where presumably the response specified by the unconscious prime would need to be inhibited before the alternative response was specified. If the modulation of the N2/P3 in the current experiment was simply the result of increased motor preparation for go primes, then a similar N2 should have been elicited in these previous experiments. In any case, the additional finding of a facilitatory effect of congruent primes shows that like the go response, the no-go response can also be unconsciously primed. More specifically, since neither the neutral prime nor the no-go prime should produce an initial activation of a motor response, the difference in N2 and P3 amplitude between these two conditions indicates that the reduction in amplitude of these components is caused by priming of the no-go response.

In experiments 2 and 5 the additional finding of early frontal negativities related to no-go primes supports the assumption that the no-go N2 was elicited in response to direct unconscious engagement of frontal control mechanisms. In experiment 2, neutral prime trials showed significantly reduced amplitude at frontocentral electrodes in comparison to go prime trials. In addition, the comparison between no-go primes and go primes showed a near significant trend, with no-go prime trials showing more negative amplitude than go prime trials. A significant early modulation of

frontocentral ERP activity dependent on prime type was also observed in experiment 5, with no-go primes showing significantly more negative amplitude in comparison to go primes. Furthermore, this effect was found to be dependent on the degree to which participants' responses were affected by the unconscious primes. Participants were split into one group with a large priming effect and another group with little or no priming effect. A significant interaction between prime type and priming group was then observed, such that participants with a large priming effect also showed a greater degree of frontocentral ERP modulation dependent on the nature of the unconscious prime. Importantly, this effect was independent of performance on the prime identification task, suggesting that the group differences were not driven by differences in the visibility of the primes. The finding that these frontal negativities were associated with behavioural priming provides strong evidence that the successful recruitment of frontal control mechanisms by the unconscious primes was present on these trials, and to a greater degree in the primed participants.

The failure to find early prime-related differences in experiments 3 and 4 means that it is not possible to conclude that engagement of frontal control mechanisms was directly elicited by the primes in these experiments. Although target-related N2 and P3 components did vary as a function of the unconscious prime, the finding of early visual effects, in particular in experiment 3, suggests that perhaps the modulation of target-related N2 and P3 components was the result of perceptual priming in experiments 3 and 4. This possibility will be discussed in more detail later in a later section of this chapter exploring the locus of the priming effects. Overall, the current set of experiments show highly replicable effects of modulation of frontal no-go N2 and P3 ERP components in response to subliminal primes. This is in contrast to previous reports that these components are not affected by unconscious primes (Dehaene et al., 2003; Mayr, 2004).

P300

In addition to the frontal no-go N2 and P3, no-go trials showed decreased parietal P300 amplitude. This large positive deflection maximal over parietal

electrodes is perhaps the most widely investigated ERP effect, possibly due to its extremely large amplitude and its appearance in a number of different tasks (Coles, 1989; Verleger, 1997; Verleger et al., 2005). It is most commonly studied in the oddball paradigm, where participants are required to look out for infrequent targets (oddballs) in a sequence containing mainly non-target distractors (see Potts, 2004; Verleger, 1997 for comprehensive reviews). The parietal P300 is typically larger in response to the infrequent targets. This has led a number of researchers to conclude that this component reflects the updating of contextual information related to the nature of the stimulus. Since the P300 reliably distinguishes targets from non-targets it is widely thought that it peaks after the completion of stimulus evaluation when the stimulus has been successfully identified. A number of reports also suggest that while it is sensitive to manipulations of stimulus discriminability, it is insensitive to situations involving response conflict (Coles, 1989). However, more recently P300 has been strongly linked with decision making processes, in particular the transition from stimulus-related to motor-related processes (Verleger et al., 2005). In contrast to the traditional view of P300, this theory states that this component is affected both by response and stimulus manipulations. Verleger et al. (2005) suggest that the P300 indexes the point at which a decision is reached about an upcoming stimulus, and is associated with the transition from stimulus evaluation to response processing.

In the current research, increased P300 amplitude was observed in response to go trials in each of the five experiments. Importantly, modulation of ERPs dependent on prime type showed a functional dissociation between go and no-go trials. For no-go trials prime-related modulation was maximal at frontocentral electrodes while for go trials prime-related modulation was maximal at parietal electrodes. This dissociation of frontal no-go related priming and parietal go related priming is important to consider with respect to Leuthold and Kopp's (1998) finding of a parietal N2 for incongruent go trials, which they interpreted as reflecting response conflict between left and right hand responses. Leuthold and Kopp (1998) asked participants to respond with one hand when a stimulus above fixation was flanked by

horizontal bars, and with the other hand when a stimulus below fixation was flanked by horizontal bars. They found that subliminal primes were successful in directly eliciting the motor response as measured by the LRP. In addition, they found a parietal negativity for incongruent trials in comparison to congruent trials peaking around 400ms after stimulus onset. They interpreted this as a parietal N2, reflecting conflict between the primed response and the target. This finding prompted Eimer and Schlaghecken (2003) to suggest that while a frontal N2 is evident only in response to conscious conflict, a parietal N2 might reflect a similar process in response to unconscious conflict.

Inspection of the topographic distribution of the N2 component in the current research revealed that although the no-go N2 showed an initial frontal distribution, this was then replaced by a parietal negativity. However, the functional dissociation between the no-go N2 and the parietal P300 modulation described above confirms that modulation of no-go trials was present over anterior electrodes while modulation of go trials was present over posterior electrodes. Furthermore, the temporal dissociation evident in the scalp topographies – with an earlier onset for the frontal modulation in comparison to the parietal modulation – provides further evidence that these two aspects evident in the topographic maps were functionally distinct. More precisely, while the frontal effects reflected modulation of the no-go N2, the posterior effects reflected modulation of the P300. This functional distinction between these two components may give some insight into the effects observed by Leuthold and Kopp (1998). The latency and topography of their posterior N2 is consistent with the observed modulation of P300 trials in the current experiments, perhaps suggesting that the parietal modulation observed in their experiment was a P300 modulation and not an N2 modulation. This suggestion is further supported by the presence of a similar parietal negativity observed on incongruent go trials in the current experiment 3. In this experiment, participants were required to respond with one hand to one stimulus configuration and the opposite hand to another stimulus configuration. They were required to make a no-go response to a third stimulus set. Importantly, a similar dissociation was observed between

the parietal and frontal prime-related modulations in this experiment such that incongruent go trials – which were functionally identical to the same condition in Leuthold and Kopp (1998) – showed a parietal negativity in comparison to congruent go trials, while no-go go trials showed frontal modulation.

LRP and go/no-go differences

Praamstra and Seiss (2005) showed that a pseudo N2 was evident on congruent no-go trails caused by averaging together left and right hand responses. Since the first experiment in this thesis utilised a similar paradigm, namely one which produced a negative compatibility effect, it was important to consider the influence of overlapping motor potentials in this experiment. Generating ERP separately for left and right hand responses revealed that the same pattern of activity was evident for each hand over each hemisphere, confirming that the no-go N2 observed in response to congruent trials in experiment 1 was not an artefact generated from lateralised movement related activity. This issue was also explored in experiments 2 and 3. In each of these experiments, behavioural and EEG effects followed a positive compatibility effect. Thorough inspection of ERP waveforms generated separately for each response hand revealed that early frontal go/no-go effects were in the opposite direction to motor related effects. More specifically, since both the RP and LRP are negative potentials, the presence of a go prime should produce an increased negativity if the early separation were dependent on motor activation. However, in experiments 2 and 5 where early separations were evident, they were in the opposite direction, with increased negativity for no-go trials in comparison to go trials. Therefore, rather than being caused by increased motor activation, the early separations in these experiments are evident despite overlapping motor related activity which would work in the opposite direction. It is important to note that increased motor activation produced by the primes, could work to increase the amplitude of the no-go N2 on incongruent go trials. However, the latency of these N2 effects, beginning around 250ms after target onset, rules out the possibility that they were generated by overlapping motor potentials, which were consistently evident

only in the first 150ms after stimulus onset. These findings confirm that in each experiment the go/no-go differences were not caused by overlapping motor potentials and thus they more likely reflect true modulation of the no-go N2.

Visual ERP effects

In each experiment visual ERP effects were explored both in relation to each condition (such as congruent versus incongruent go conditions) at electrode Oz and in relation to the physical stimuli (such as left versus right sided diamonds) at electrodes PO7/PO8 and O1/O2. Since in each experiment the stimuli were either presented at lateral location, or were somewhat asymmetrical (arrows), these lateralised visual effects were explored to determine if the visual response was augmented over electrodes contralateral to primes and targets. In each experiment (except experiment 3) the visual stimuli were counterbalanced either within or between participants such that while one stimulus configuration coded for a go response for some participants (or in one experimental session), the same stimulus coded for a no-go response for other participants. Therefore, lateralised ERP effects were explored in relation to the physical stimuli and not their functional significance, since any lateralised effects related to the physical characteristics of the stimuli would cancel out when the counterbalancing was averaged together. Response mapping was also included as a factor in the analysis to explore whether any visual asymmetries were also dependent on the functional significance of the target. Any such modulation would likely reflect an N2pc component which is thought to reflect attention to and identification of target-related aspects of the stimulus (Kiss et al., 2007).

Each experiment showed some modulation of early visual ERP effects in response to the unconscious primes. In experiment 1 there were no significant effects on P1 and N1 amplitude, although a significant later effect was observed at electrode Oz with go primes showing significantly increased amplitude. In addition, significant lateralised visual ERP effects were observed with increased P1 and N1 amplitude contralateral to the direction of

the prime arrow. Similar effects were observed in experiment 2 and 3, with a significant effect of prime congruency on N1 and significant lateralisation of N1, with increased amplitude contralateral to the side of a diamond stimulus. Experiment 4 showed no modulation of visual ERP effects, but did show small increased lateralised ERP components contralateral to the side of the prime. Similarly experiment 5 showed no effects at electrode Oz, but did reveal some lateralised ERP components. In summary, experiments 1 to 3 showed both prime-related effects at Oz and lateralised visual ERP effects, while experiment 4 showed only the lateralised effects.

Locus of priming effects

The presence of early visual ERP effects is important in trying to determine the locus of the priming effects in the current experiments. This was particularly important in those experiments where no early prime-related frontal negativities were observed in response to the subliminal prime. The presence of visual ERP effects in these experiments would point to the possibility that the prime-related effects on the target N2 and P3 were caused by visual or attentional priming. Since experiments 3 and 4 both failed to show such early prime-related modulation, visual ERP analysis in these conditions is particularly important.

As described above, experiment 3 showed significant lateralised visual effects as well as a significant effect at electrode Oz such that congruent go trials showed a significantly increased N1 component. In this experiment participants were instructed to press a left button in response to a left diamond and a right button in response to a right diamond. The presence of early lateralised visual effects suggests that the visual system successfully coded the location of the target stimulus (the diamond). The further finding of an increased N1 at electrode Oz for congruent go trials suggests that the unconscious primes were able to direct attention to the location of the target stimulus. Previous research has found that visual P1 and N1 components are subject to modulation by attention, such that they are increased when attending to the relevant visual information (Clark & Hillyard, 1996). Therefore, the finding that the visual N1 component was significantly

increased for congruent go trials suggests that a go prime was able to direct attention to its location, which meant that when the target was presented in this same location, it showed an increased visual response. Such an interpretation could mean that the modulation of target-related go/no-go differences was caused by modulation of attention by the unconscious primes.

However, it is not clear how such attentional modulation might cause the N2 and P3 effects observed in experiment 3. If attention is directed toward the location of the upcoming stimulus by the prime then this would likely speed up reaction times for congruent go trials and reduce reaction times to incongruent go trials. This would provide an adequate explanation of priming on go trials without the need to postulate direct unconscious initiation of action. However, if attention was directed towards a go prime, then responses to incongruent no-go primes should be quicker (as measured by the N2) than responses to congruent no-go primes, since on incongruent trials attention would be directed to the go prime, which would then be replaced by a no-go target. In fact the exact opposite was observed, with congruent no-go trials showing evidence of early engagement of frontal inhibition/control mechanisms. Interestingly, the increased amplitude of the visual N1 component at Oz was only present for the congruent go, with congruent no-go, incongruent no-go and incongruent go all showing similar amplitude. This suggests that any modulation of attention was limited to congruent go trials. Therefore, the priming of the no-go N2 on no-go trials was unlikely to be caused by such an attentional modulation, suggesting that the early onset of the N2 on congruent no-go primes reflects direct engagement of frontal inhibition/control mechanisms on these trials. However, the failure to find a similar early N2 for incongruent go trials is still problematic for this interpretation.

Experiment 4 showed significant modulation of target-related N2 and P3 components dependent on the unconscious primes, but did not show any early prime-related effects at frontocentral electrodes. Visual ERP analysis revealed no significant effects on N1 or P1 amplitude. In addition, only a

modest lateralised effect was evident with a significant separation based on prime type from 20 to 50ms after target onset. The absence of early prime-related frontal ERP effects in experiment 4 again meant that it was not possible to conclusively state that the unconscious primes were able to directly initiate inhibition/control mechanisms. However, the absence of any congruency related ERP effects at electrode Oz also rules out the possibility that the target-related effects were simply caused by modulation of attention by the unconscious primes. Moreover, the fact that the targets were presented in a central location in this experiment, with the primes at lateral locations, means that priming of attention toward the prime, even if it did occur, should not influence target processing. The absence of an interaction between prime type and response mapping for the early lateralised effects also suggests that they reflected low level visual processing of the lateral primes and not modulation of spatial attention. These observations strongly suggest that target-related N2 modulations in this experiment were not caused by perceptual/attentional priming, but rather that the no-go response was unconsciously primed. Nevertheless, the failure to show early prime-related effects means that it is not possible to conclude that, in this experiment, the unconscious primes directly activated the no-go response.

Experiments 1, 2, and 5 all showed significant early prime-related frontal modulation, making interpretation of the locus of priming in these experiments more straightforward. Despite the presence of early visual effects in these experiments, the finding that no-go prime trials showed increased negativity over frontocentral electrodes suggests that the unconscious primes were able to directly initiate the no-go response. The fact that in each of these experiments the early separations at frontal electrodes was entirely dependent on prime type (rather than prime congruency) means that these effects could not have been caused by attentional modulation for congruent trials. For example, if congruent primes were successful in alerting attention to the location of the upcoming target, then an earlier N2 would have been observed only for congruent no-go primes and not incongruent go primes. The presence of an early N2 and LRP entirely determined by prime type thus suggests that in each of these

experiments the prime directly modulated both initial preparation and inhibition of the motor response.

The visual effects in these experiments likely reflect unconscious detection of visual features of the targets. Similar effects have recently been described by Del Cul, Baillet and Dehaene (2007), who show that early visual responses differentiate subliminal primes. Kiesel, Kunde and Hoffmann (2007) outline a likely mechanism for unconscious priming effects, which includes an early identification of relevant features of the prime. They suggest that unconscious priming effects are caused by activation of action triggers defined by the particular set of target stimuli. This account allows for the presence of perceptual facilitation, without direct perceptual priming. The crucial step in allowing the prime to activate the appropriate response is its classification as an adequate action trigger. Although this activation is more likely to occur when the primes are also used as target stimuli (as in the current experiments) this account can also be extended to account for unseen primes. For example, Kunde, Kiesel and Hoffmann (2003) used similar stimuli to those employed by Dehaene et al. (1998) and showed that when participants were asked to respond to numbers above and below 5 with different hands priming extended to the unseen numbers 2 and 3 only when 1 and 4 were used as the target stimuli. When 3 and 4 were used as targets then primes 1 and 2 exerted no priming effect. Additionally Kiesel et al. (2007) suggest that priming of unseen prime words that are semantically associated with target words only occurs if the response set is large enough for participants not to easily remember individual exemplars and thus mistakenly include certain items as action triggers. For example, they suggest that once knife, mug and cup have been included as targets, it seems plausible that spoon would also become an active trigger since participants are likely to set up a response set for crockery items. While the debate surrounding the plausibility of truly semantic priming effects is not directly relevant for the work presented in this thesis, the model outlined by Kiesel et al. (2007) provides a good account of the likely mechanisms for the priming effects in the current research.

Conclusions

Implications of current research

The research described in this thesis shows that, like preparation for a motor act, inhibition of an impending action can also be initiated unconsciously. This finding has important implications for the role of consciousness in the control of action. As described above, there is a great deal of research associating frontal inhibition/control mechanisms with consciousness. For example, Dehaene et al. (2003) and Praamstra and Seiss (2005) have shown using both fMRI and EEG that these mechanisms normally engaged in resolving response conflict, are not activated when the conflict is unconscious. In addition, Libet (1985) suggested that while voluntary acts are initiated in the brain prior to conscious awareness of the decision to act, consciousness may retain the ability to veto the action. A similar position was outlined by Eimer and Schlaghecken (1998) who suggested that inhibition of a motor response, such as that in the go/no-go task can only occur in response to a conscious stimulus. This has led to the continued popularity of Libet's suggestion of conscious "free wont" that consciousness acts to veto the performance of unwanted actions. While a number of objections have been made to this possibility on theoretical grounds (cf. Velmans, 2003), up to now there has been no direct evidence in support of, or against this assumption. The current research provides empirical evidence against the assumption that inhibition of an imminent motor response can only occur consciously. This has important implications for conscious free will, suggesting that when interacting with our external world, decisions regarding our actions can be arrived at prior to consciousness of those decisions.

It is important to note that the current experiments are somewhat different to Libet's et al.'s (1983) experiment exploring voluntary action. In those experiments participants were asked to flex their wrist whenever they felt the urge to do so, and to retrospectively report the time at which they decided to move. Libet et al. (1983) found that the brain began to prepare the movement, as measured by the readiness potential, some 300 ms before the

time at which participants consciously decided to act. In an attempt to explore the suggestion that consciousness was required to veto an action Libet et al. (1983) explored the readiness potential in situations where participants pre-prepared a response and then decided on some trials to veto that action at the last moment. Libet et al. (1983) found that on these veto trials, the readiness potential was very similar to that observed in the act condition. However, it is important to note that Libet provides no statistical analysis of the difference in RP between the veto condition and the condition where an action was performed. In addition, he did not record EEG activity from any other electrodes, such as frontal electrodes where any correlates of the veto are likely to occur. Since on freely initiated and freely vetoed trials, no external event would occur with which to time lock the ERP, it is difficult to explore the veto in such a paradigm. The current research therefore explored the potential of unconscious primes to initiate the ERP correlates of withholding an impending action. Importantly, in each of the experiments described in this thesis, participants were responding to external stimuli rather than making responses at the time of their choice. However, as outlined in chapter 1 there is reason to believe that although different to freely initiated actions, responses to external stimuli are more accurate representations of our typical interaction with the external world. Despite this, it is important to note that while the current set of results is relevant to the question of whether consciousness is required to withhold an impending action, the experimental paradigm is different to the original one employed by Libet et al. (1983).

Given the conclusions from the current research that both preparation and inhibition of a motor act can be initiated without consciousness, it is important to consider what the role of consciousness might be. Gomes (1998) has suggested that even if both the initiation of action and a veto have unconscious brain correlates, consciousness may still have a role in controlling behaviour since it might not be possible for a veto to occur without the initial action having entered consciousness. Consistent with this position, there is evidence to suggest that trial to trial adaptation of behaviour does not occur in response to an unconscious stimulus. Kunde (2003) explored

sequential modulation of conflict induced by subliminal primes. Participants were required to make a left handed response to left pointing arrows and a right handed response to right pointing arrows. Kunde (2003) was particularly interested in the effect of the subliminal primes on trial to trial modulation of behaviour. He found that when the primes were conscious, trial to trial effects were evident such that the RCE was reduced on trials where the previous trial was incongruent. This finding shows that detection of response conflict on one trial was able to trigger control mechanisms to reduce the effect of this conflict on the subsequent trial. Interestingly, trial to trial modulation was not evident when the primes were unconscious. This was despite the fact that the subliminal primes still produced a significant response congruency effect, indicating that they were modulating behaviour within a single trial. Thus Kunde's (2003) results suggest that in order to flexibly adapt ones behaviour in response to previous events, one must be conscious of those events. In the research outlined in this thesis, all the behavioural modulation was present within a single trial. Although both preparation and inhibition of a response were found to be directly initiated by the subliminal primes, the original decision to act was likely a conscious one. Since participants were required to make a speeded reaction to a go stimulus, their default mode was likely to be ready to press the button. This pre-potent readiness to respond was set up consciously in response to the experimental instructions in the task. Therefore, even with the current finding that inhibition of a motor act can occur unconsciously, in order for this inhibition to occur participants were first required to make a conscious decision to prepare to press a button on each trial. Therefore, the research described in this thesis is not inconsistent with the theory that consciousness is required for cognitive control mechanisms to allow flexible adaptive control of behaviour. Instead it directly assesses Libet's (1985) claim that consciousness is required to veto an impending action, showing that contrary to his claim, inhibition of an imminent response can be initiated unconsciously.

Directions for future research

There are a number of ways in which future research might be able to explore the generality of the current findings to different situations. For example, in the current research although both go and no-go responses were found to be directly initiated by subliminal primes, responses were ultimately triggered by conscious stimuli. Without the presentation of the target stimuli, the partial activation of go and no-go stimuli would likely have remained below the threshold to produce overt behaviour. An interesting way to explore this unconscious activation would be to present unconscious primes in advance of neutral stimuli that code neither for a go nor a no-go response. Such an experiment was recently described by Kiesel, Wagener, Kunde, Hoffmann, Fallgatter and Stocker (2005), who showed that participants' responses on such free choice trials were influenced by subliminal stimuli. They explored this modulation in an experiment where participants were primed to act either with their left or right hand, showing that the subliminal primes could bias responses on free choice trials such that 60 percent of responses were compatible with the prime. In addition to this effect on the decision of which button to press in this experiment, Kiesel et al. (2005) found that even when participants acted in the opposite direction to the primes, their reaction times were significantly slower due to an initial unconscious activation of the primed response hand. An interesting variation on this experiment would be to explore if such modulation of free choice trials is evident in a go/no-go task, as this would show that a free decision about whether or not to continue with an impending action can be influenced unconsciously.

Another interesting extension of the current research might be to see if overcoming a pre-potent left or right hand response is influenced unconsciously. In a sense, the important aspect of Libet's conception of a veto is that participants need to change their mind about an imminent action. This may result in inhibition of the action altogether, or selection of an alternative action. While there is much research exploring the role of subliminal primes in choice reaction time tasks (Dehaene, 1998; Eimer & Schlaghecken, 1998; Leuthold & Kopp, 1998), these experiments have an

equal probability of a stimulus requiring a left hand response and a right hand response. One way to build a predominant response hand would be to increase the proportion of trials in which a response with that hand was required. Presenting subliminal primes in such a task would allow exploration of whether unconscious primes are able to influence a decision to overcome a predominant response with an alternative response, and whether this process is mediated by frontal control/inhibition mechanisms.

Similarly, to explore the locus of the priming of no-go response in more detail, it would be interesting to see if early prime-related effects were evident in response to subliminal primes that were only categorically or semantically related to targets, and were not visually related. This could be explored using a variation of the task employed by Dehaene et al. (1998) by asking participants to make a go response to numbers above five and a no-go response to numbers below five. By presenting only the numbers 1, 4, 6 and 9 as targets, it would be possible to explore if the numbers 2, 3, 7 and 8 were able to prime the go/no-go decision. This would further confirm that priming of a no-go response can be directly elicited by an unconscious prime and not only via perceptual or attentional priming.

New analysis techniques may also provide ways to extend the research described in this thesis. For example, as new time-frequency and single trial measures of EEG activity are developed, researchers are closing in on the neural correlates for consciousness. Some candidates for these neural correlates have included synchronous high frequency oscillatory EEG activity (Melloni et al., 2007) as well as measures of neural complexity in the EEG signal (Burgess, Rehman, & Williams, 2003). As analysis techniques of EEG data improve and the precise neural correlates of consciousness are discovered it may be possible to explore unconscious behaviour without the need to present subliminal stimuli in advance of target stimuli which participants are required to respond to. For instance it might be possible to simply ask participants to make responses dependent on events in the external world, determine when they became conscious of these events using EEG markers of conscious awareness, and then compare this to when

they began preparing responses to the events. Although such an experiment would still require responses to external stimuli it would allow more direct examination of whether such responses are made before conscious awareness of the events leading to the decisions.

In addition, although the poor signal to noise ratio of the LRP makes it impossible to determine motor readiness in single trial EEG, recent analysis techniques including independent components analysis have led to the possibility of extracting ERP components on single trials associated with preparation and inhibition of a motor response. One candidate for such an EEG component is the sensorimotor mu (12-15 Hertz) rhythm, which correlates with preparation and inhibition of motor responses (Chatrain, Peterson, & Lazarte, 1959). The ability to reliably detect this component on single trials is currently being utilised in the exploration of possible human-computer interface systems which allow direct control of external events by imagining movements with the left or right hand (Pfurtscheller, Brunner, Schlogl, & Lopes da Silva, 2006). This rhythmic EEG activity could also be utilised to detect instances where participants began to prepare an action but did not follow through with the act itself. This would be directly analogous to the veto situation described by Libet et al. (1983), who mentioned that a number of participants reported after the experiment that they occasionally prepared an action but then withheld it at the final moment. Such trials could not be picked up with conventional ERP analysis due to the low signal to noise ratio of the RP and LRP, but the successful detection of the sensorimotor mu on individual trials suggests that this technique might allow direct examination of such trials by retrospectively classifying acts that were prepared but not subsequently performed.

New analysis techniques may also help to disentangle some of the difficulties observed in the current research with overlapping ERP components. This was evident both in terms of the overlapping motor related activity described above as well as for the no-go P3 components. Since the no-go N2 and P3 components immediately follow one another it is difficult to determine whether a reduced N2 reflects modulation of the N2 itself, or overlap from an

early onsetting P3. In addition to the N2/P3 complex, ACC activity has been reliably associated with phase resetting of theta oscillations (5-7 Hz; Wang, Ulbert, Schomer, Marinkovic & Halgren, 2005). Since, as described above motor activity is typically associated with the higher frequency mu rhythm, as well as very high frequency gamma activity (Gonzalez et al., 2006), time frequency analysis of the current data could provide a way to separate control mechanisms from motor activation-related mechanisms, since although their ERP correlates overlap in the time domain, their EEG correlates may be separable in the frequency domain. Furthermore, Hanslmayr, Pastotter, Bauml, Gruber, Wimber and Klimesch (2008) recently showed that increased phase coupling between ACC and pre-frontal cortex (PFC) was associated with resolution of conflict in a Stroop task. They suggest that while theta amplitude emanating from ACC is associated with the detection of conflict, synchronous activity in ACC and PFC may be associated with engagement of control mechanisms in resolving the conflict detected by the ACC mechanism. Experiments exploring the role of such modulation on a within and between trial basis may allow exploration of the hypothesis outlined above, that while consciousness is not required to simply inhibit an action it may be involved in allowing flexible adaptive processing of a changing environment.

Summary

This thesis aimed to explore whether the decision to withhold an impending motor action can be initiated unconsciously. This issue has important implications for our understanding of the nature of conscious free will, in particular in regard to Libet's (1985) suggestion that while consciousness is not required to begin preparation for action, it may be required to veto an impending action. In addition, a number of experiments utilising masked priming have suggested that subliminal primes do not engage frontal control mechanisms (Dehaene et al., 1998, Eimer & Schalghecken, 1998, Praamstra & Seiss, 2005). The research in this thesis aimed to clarify whether this assertion was also true for inhibition in the go/no-go task. Five experiments were conducted to explore if the no-go N2 and P3 components could be

modulated by an unconscious prime. In each experiment there were significant modulations of target-related N2 and P3 components as a function of the unconscious prime. Moreover, in three of the experiments, significant early separations at frontocentral electrodes pointed to the possibility that the subliminal primes were able to directly engage the frontal inhibition/control mechanisms indexed by the no-go N2. These findings suggest that like preparation for action, the decision to withhold an action can be initiated unconsciously.

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