



# Group and individual analyses of pre-, peri-, and post-movement related alpha and beta oscillations during a single continuous monitoring task



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## ABSTRACT

Band power linked to lower and upper alpha (i.e. 8–10 Hz; 10–12 Hz) and lower and upper beta (i.e. 12–20 Hz; 20–30 Hz) were examined during response related stages, including anticipation, response execution (RE), response inhibition (RI) and post response recovery (PRR). Group and individual data from 34 participants were considered. The participant's objective was to press a response key immediately following 4 non-repeating, single integer odd digits. These were presented amongst a continuous stream of digits and Xs. Electroencephalogram (EEG) signals were recorded from 32 electrodes (pooled to 12 regions). In the group analyses, participant EEG response was compared to baseline revealing that upper alpha desynchronised during anticipation, RE and RI; lower beta during anticipation and RE; and upper beta just RE. Upper alpha desynchronisation during rapid, unplanned RI is novel. Also, upper alpha and lower/upper beta synchronised during PRR. For upper alpha, we speculate this indexes brief cortical deactivation; for beta we propose this indexes response set maintenance. Lastly, lower alpha fluctuations correlated negatively with RT, indexing neural efficiency. Individual analyses involved calculation of the proportion of individuals displaying the typical RE and PRR trends; these were not reflected by all participants. The former was displayed individually by the largest proportion in upper alpha recorded left fronto-centrally; the latter was most reliably displayed individually in lower beta recorded mid centro-parietally. Therefore, group analyses identified typical alpha and beta synchronisation/desynchronisation trends, whilst individual analyses identified their degree of representation in single participants. Attention is drawn to the clinical relevance of this issue.

## 1. Introduction

The human EEG has frequently been used as an index of neural activity in various experimental contexts. Research has provided vital clues and information in relation to a range of brain processes and associated oscillations (e.g. see Başar, 2012; Hsieh and Ranganath, 2014; Klimesch, 1999; Klimesch, 2012; Tallon-Baudry and Bertrand, 1999 for example reviews). Brain processing relating to ‘goal conflict’ is one specific area in which the human EEG has recently assisted in making in-roads. Studies in this field typically have drawn on Reinforcement Sensitivity Theory (RST; Gray and McNaughton, 2000; McNaughton and Corr, 2004, 2008) which proposes that ‘goal conflict’ is experienced as anxious rumination when the behavioural inhibition system (BIS) acts to resolve approach and avoidance conflicts.

In one set of studies, goal conflict was introduced using an individually calibrated stop signal task (SST) provoking maximal behavioural goal conflict for each participant (i.e. McNaughton, Swart, Neo,

Bates, and Glue, 2013; Neo, Thurlow, and McNaughton, 2011; Shadli, Glue, McIntosh, and McNaughton, 2015; Shadli, Smith, Glue, and McNaughton, 2016). The dominant finding focussed on a right frontal ‘goal conflict specific rhythmicity’ (GCSR) which typically presented itself at electrode F8 during maximum goal conflict. Initially, the GCSR was identified in the theta frequency range (7–8 Hz; Neo, Thurlow and McNaughton, 2011) but in subsequent studies there has been some degree of variation: 9–10 Hz in McNaughton, Swart, Neo, Bates and Glue (2013); 5–9 Hz in Shadli et al. (2015); and 7 Hz, coupled with a 10 Hz left frontal GCSR response in Shadli, Smith, Glue and McNaughton (2016). Otherwise, it was also demonstrated that the GCSR is significantly reduced in participants treated with anxiolytic drugs (relative to placebo) leading to proposals that the GCSR could be used as a specific biomarker of anxiety (McNaughton, Swart, Neo, Bates, and Glue, 2013; Shadli et al., 2016).

Similarly, links between EEG and goal conflict, comprising broad increases in EEG theta coherence and power during behavioural goal

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conflict have been reported in studies where participants engaged in a continuous monitoring target detection task (Moore et al., 2006; Moore et al., 2012). The reported theta effects were speculatively linked to increased ‘hippocampal – neocortex’ interplay during goal conflict resolution (Gray and McNaughton, 2000; Miller, 1989). Further, a step wise discriminant analysis revealed that six EEG variables maximally discriminated participants defined as high BIS or low BIS (using the BIS/BAS scales; Carver and White, 1994) of which four were related to primary goal conflict, two to response execution and five of the six were in the theta frequency range. Otherwise, evidence of a goal conflict effect has been reported in the 1–7 Hz (i.e. delta and theta) range in a study conducted by Savostyanov et al. (2009) in which EEG power increased for 800 ms during a behavioural goal conflict period when a prepotent response was suppressed.

Studies investigating goal conflict, such as those described above, often make use of a self-contained task in which the participant's job is to anticipate and execute (or inhibit) a motor response. By virtue of the tasks used, the studies typically therefore have the potential to capture neural activity linked to response execution (or inhibition), anticipation of response and post response recovery<sup>1</sup> within one self-contained study. However, investigation of EEG oscillations recorded at task stages other than those specifically linked to goal conflict are typically not part of the research agenda. Failure to consider what may be happening at task stages other than those specifically linked to goal conflict is unfortunate, especially when the prominent focus of anticipation and execution of response (or movement) in research concerned with brain rhythms such as the Rolandic mu and central beta EEG are taken into account (e.g. Höller et al., 2013; Kilavik et al., 2013; Llanos et al., 2013; Picazio et al., 2014).

In Moore et al. (2008), we revisited the EEG data recorded in Moore et al. (2006). However, on that occasion, we focussed on EEG alpha (8–12 Hz). Previous research describing ‘hippocampal – neocortex’ interplay had been based on hippocampal theta derived from lower mammals, which is known to extend up to 10 Hz and sometimes possibly as high as 12 Hz (Vanderwolf, 1969). Therefore, initially we set out to identify whether the goal conflict effects identified in Moore et al. (2006) extended into the alpha range of frequencies (8–12 Hz). Whilst we found this not to be the case, the reanalysis did reveal a variety of findings associated with preparation and execution of movement. For instance, centrally located lower alpha coherence increased during motor activity (i.e. response execution and response inhibition). Also, widespread upper alpha coherence showed an increase during the same task stages. Broad alpha power (8–12 Hz) globally desynchronised during motor response indicating (at least in part) a classic Rolandic mu rhythm power response. Overall, these effects showed good consistency with previously reported investigations of traditional alpha and Rolandic mu oscillations during preparation for and execution of movement (e.g. Andrew and Pfurtscheller, 1997; Manganotti et al., 1998; Pfurtscheller and Berghold, 1989; Leocani et al., 1997). Additionally, novel findings were reported including an alpha coherence profile proposed to reflect a working memory network activated during response execution and an EEG trend linked to neural efficiency, in which a progressive alpha desynchronisation trend (provoked by incremental increases in anticipation) was linked to faster response times.

The data reported in Moore et al. (2008) therefore provided both replication of previous findings in relation to traditional alpha and the Rolandic mu rhythm alongside novel results. However, as a study focussing on anticipation and execution of response, failure to consider EEG in the beta range of frequencies was a key omission, since this waveband is known to also have a close link with movement and

preparing for movement. For instance, in previous research, beta has shown evidence of desynchronisation of the central beta rhythm prior to and during movement. This effect was first described over 60 years ago by Jasper and Penfield (1949) and has been reported on numerous occasions since for actual movement (e.g. Kilavik et al., 2013; Pfurtscheller, 1981; Stancák and Pfurtscheller, 1996; Leocani et al., 1997) and well as observed movement (e.g. Babiloni et al., 2016). Characteristically, central beta desynchronisation is initiated approximately 2 s before overt movement, has a contralateral dominance (though becoming bilaterally symmetrical just before movement) and is most apparent in electrodes placed close to sensorimotor regions (Kilavik et al., 2013; Pfurtscheller and Lopes da Silva, 1999). In terms of topography, central beta response presents itself slightly anterior to the central Rolandic mu rhythm and occupies the pre-Rolandic motor area (compared to the Rolandic mu rhythm which occupies the post Rolandic motor region) (Pfurtscheller and Lopes da Silva, 1999). Further research has shown that beta oscillations also follow this trend during imagination of movement as well as actual movement (e.g. Höller et al., 2013) and this is a neural response on which brain computer interface (BCI) devices often capitalise (e.g. Chaudhary et al., 2016; Ramos-Murguialday and Birbaumer, 2015). Data have also been reported by Babiloni et al. (2016) which suggest a role for anterior beta oscillations (and alpha) as part of a human mirror neuron system differentiating one's own moves compared to moves of someone else that one observes.

EEG beta oscillations have also been shown to react after movement – this is characterised by rapid synchronisation immediately after response. For example, in one study Leocani et al. (1997) reported evidence of 18–22 Hz event related synchronisation (ERS) occurring 0.75 s after response termination during self-paced movement. More recently, similar effects have been reported by Espenhahn et al. (2017) in which post movement beta synchronisation showed prominence slightly anterior to the central midline in 6 healthy participants; it was also reported that the EEG index remained relatively consistent when test retest analyses were performed over a number of EEG sessions taking place over several weeks. In terms of location, post movement beta synchronisation tends to be dominant over the contralateral sensorimotor region, though can also be displayed over ipsilateral sensorimotor regions (Espenhahn et al., 2017; Pfurtscheller et al., 1998). Additionally, this post movement beta synchronisation is also present during imagination of performing a movement and so also potentially has utility in BCI applications (e.g. Solis-Escalante et al., 2012).

### 1.1. The current study

In the current study, we aim to follow up Moore et al. (2008) only this time, as well as EEG alpha (8–10 Hz; 10–12 Hz), beta oscillations (12–20 Hz; 20–30 Hz) and post movement neural activity will be included in the analyses. Here, we focus on EEG power and hope to gain further information about the electrocortical signature linked to preparing for movement, executing movement and recovering from movement during the versatile response task used in Moore et al. (2012). Data recorded in Moore et al. (2012), which was primarily a study investigating EEG correlates of goal conflict, will be reanalysed.

Concerning hypotheses, we predict that alpha power will desynchronise at task stages in which motor response is deployed and this will be particularly evident at regions of interest close to post Rolandic motor regions contralateral to the movement. It is anticipated that beta EEG power will show a similar trend though, in terms of topography, these effects will be strongest at pre-Rolandic motor area (i.e. anterior to those predicted for alpha desynchronisation). One other primary prediction which can be made for beta is that there will be a post-movement synchronisation of beta power in the contralateral (and possibly ipsilateral) sensorimotor region.

Additionally, one other novel aspect will be addressed in this study. Although, research concerning Rolandic mu and movement related beta oscillations present a relatively consistent account of synchronisation

<sup>1</sup> We use the term ‘post response recovery’ in the current study in place of the more typically used term ‘post-movement beta rebound’ since post response recovery is more specific to the experimental task used here and also not exclusively describing beta.

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