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Title

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. 8-10Hz; 10-12Hz) and lower and upper beta (i.e. 12-20Hz; 20-30Hz) 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.

KEYWORDS: EEG, response, preparation, inhibition, alpha, beta.

1.0 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 & Ranganath, 2014; Klimesch, 1999; Klimesch, 2012; Tallon-Baudry & 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 & McNaughton, 2011; Shadli, Glue, McIntosh and McNaughton, 2015; Shadli, Smith, Glue & 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 (7-8Hz; Neo, Thurlow and McNaughton, 2011) but in subsequent studies there has been some degree of variation: 9-10Hz in McNaughton, Swart, Neo, Bates and Glue (2013); 5-9Hz in Shadli, Glue, McIntosh and McNaughton (2015); and 7Hz, coupled with a 10Hz 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, Smith, Glue and McNaughton, 2016).

Similarly, links between EEG and goal conflict, comprising broad increases in EEG theta coherence and power during behavioural goal conflict have been reported in studies where

participants engaged in a continuous monitoring target detection task (Moore, Gale, Morris and Forrester, 2006; Moore, Mills, Marshman and Corr, 2012). The reported theta effects were speculatively linked to increased 'hippocampal – neocortex' interplay during goal conflict resolution (Gray & 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 & 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 - 7Hz (i.e. delta and theta) range in a study conducted by Savostyanov et al. (2009) in which EEG power increased for 800ms 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[1] 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. Holler et al., 2013; Kilavik, Zaepffel, Brovelli, MacKay & Riehle, 2013; Llanos, Rodriguez, Ridrguez-Sabate, Morales & Sabate, 2013; Picazio et al., 2014).

[1] 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.

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 (8-12Hz). Previous research describing 'hippocampal – neocortex' interplay had been based on hippocampal theta derived from lower mammals, which is known to extend up to 10Hz and sometimes possibly as high as 12Hz (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 (8-12Hz). 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 (8-12Hz) 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 & Pfurtscheller, 1997; Manganotti et al., 1998; Pfurtscheller & Berghold, 1989; Leocani, Toro, Manganotti, Zhuang & Hallett, 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, Zaepffel, Brovelli, MacKay & Riechle, 2013; Pfurtscheller, 1981; Stancek & Pfurtscheller, 1996; Leocani, Toro, Manganotti, Zhuang & Hallett, 1997) and well as observed movement (e.g. Babiloni et al., 2016). Characteristically, central beta desynchronisation is initiated approximately 2 seconds 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, Zaepffel, Brovelli, MacKay & Riechle, 2013; Pfurtscheller & 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 & 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. Holler et al., 2013) and this is a neural response on which brain computer interface (BCI) devices often capitalise (e.g. Chaudhary, Birbaumer & Ramos-Murguialday, 2016; Ramos-Murguialday & 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 18-22Hz event related synchronisation (ERS) occurring 0.75s after response termination during self-paced movement. More recently, similar effects have been reported by Espenhahn, Berker, Van Wijk, Rossiter and Ward (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 synnchronisation tends to be dominant over the contralateral sensorimotor region, though can also be displayed over ipsilateral sensorimotor regions (Espenhahn,

Berker, Van Wijk, Rossiter and Ward, 2017; Pfurtscheller, Zaladek & Neuper, 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, Muller-Putz, Pfurtscheller & Neuper, 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 (8-10Hz; 10-12Hz), beta oscillations (12-20Hz; 20-30Hz) 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 and desynchronisation, participants often show a degree of interindividual variability which is often not commented upon. This is, however, an important consideration if effects reported in these studies are used to guide practical applications (e.g. BCI

devices; clinical assessment of human motor system, etc.). Recently, various studies have shown a distributed pattern of response in relation to motor imagery (e.g. Cruse et al., 2011; Goldfine, Victor, Conte, Bardin & Schiff, 2011) in healthy participants and other participants in either locked in states or suffering from other disorders of consciousness. Further, Holler et al. (2013) reported that, in a group of healthy participants, EEG response during rest, motor imagery and actual motor activity differed from the typical oscillatory pattern in some individuals, with a proportion showing synchronisation where desynchronisation was expected during motor imagery. Also, Solis- Escalante, Muller-Putz, Pfurtscheller & Neuper (2012) recently reported data in which a beta post movement recovery was only shown in 80% of their participants for real movement and 60% for imagined movement. Therefore, in the current study we also intend addressing this issue and identifying the degree to which individual participants demonstrate typical or atypical patterns of desynchronisation or synchronisation following response execution or post response recovery respectively.

2.0 Method

As this paper is an extension of earlier work, some sections of the Method have been abbreviated. Specific details about apparatus and physiological and performance data recordings can be found in the Method section of Moore et al. (2012).

2.1 Participants

There were thirty-six participants (7 males) aged 18 to 48 (M: 23.86; SD: 7.51). Due to technical problems (excessively 'noisy' EEG recordings), the EEG data of 2 participants were removed from the final sample (see Section 2.2 for details of rejection criteria). This meant that data from thirty–four participants were entered into statistical analyses. The study was approved by the University of Portsmouth Psychology Department Ethics Committee and all participants gave their informed consent prior to inclusion in the study.

2.2. Procedure and Data Reduction.

Participants sat in a comfortable chair in front of a computer monitor used to present stimuli. Continuous EEG was recorded with a Brain Vision Recorder (version 1.03.0004) from 32 scalp electrodes and collapsed into 12 cortical regions of interest (ROI; see Figure 1 below for details). The ROIs were: left frontal (LF), mid frontal (MF), right frontal (RF), left fronto-central (LFC), mid fronto-central (MFC), right fronto-central (RFC), left centro-parietal (LCP), mid centro-parietal (MCP), right centro-parietal (RCP), left parieto-occipital (LPO), mid parieto-occipital (MPO) and right parieto-occipital (RPO). Afz was used as subject ground; an average reference was applied offline.

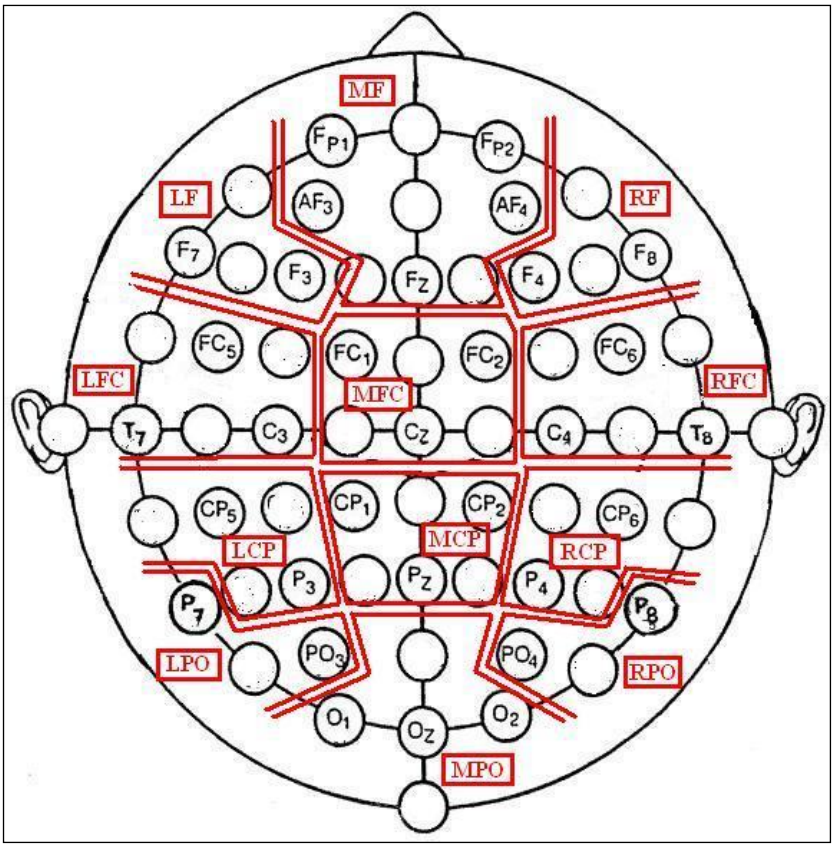


Figure 1. Regions of interest (see text above figure for definition of each region).

EEG data were analysed offline with Brain Analyser (version 2.0.0.2701). All EEG data were treated with an eye movement reduction algorithm (Gratton & Coles, 1989; Gratton et al., 1983). EEG epochs including data that were greater than '+75µV' or less than '−75µV' were rejected. This

amounted to less than 15% of all epochs for each participant included in the analysis; 2 participants were removed from the analyses as in excess of 15% of their EEG data was outside of this threshold.

Participants monitored a continuous stream of digit sequences containing 4 single integers. Each digit in each digit sequence was presented individually at a rate of 1 digit per second – all digit sequences are shown in Table 1. An X, representing a brief rest period, was displayed after each digit sequence. This was also presented for 1 second. Participants pressed a response key with their right index finger each time a digit sequence was comprised of 4 odd digits (referred to as digit sequence OOOO hereafter); the response key was the left button on a standard computer mouse. They were instructed to press the response key as quickly as possible in response to the final digit in digit sequence OOOO which was considered the *response execution* task stage (see Table 1) – response times were recorded and each participant's mean response speed was calculated. Additionally, the X following the response execution task stage was considered to be the *post response recovery* task stage (see Table 1).

Other digit sequences were: three odd digits followed by one even digit (digit sequence OOOE); two odd digits followed by two even digits (digit sequence OOEE); one odd digit followed by three even digits (digit sequence OEEE); and four even digits (digit sequence EEEE). Forty versions of each type of digit sequence were presented to each participant. The even digit in digit sequence OOOE was the task stage at which conflict between Go/NoGo was experienced most acutely and, hence, was considered the *response inhibition* task stage (see Table 1).

Lower alpha, upper alpha, lower beta and upper beta (8-10Hz, 10-12Hz, 12-20Hz and 20-30Hz respectively) power values were derived for each digit within each digit sequence. This was performed individually for each 1 second epoch (i.e. stimulus presentation duration). The X (signalling a brief 1 second rest) which appeared at the end of each digit sequence was also included in this process. These data were then averaged for each individual electrode (i.e. yielding a waveband specific average for each stimulus for each electrode) and then combined to form aggregated waveband specific data for each ROI. Additionally, we derived ROI specific power values

in the same way for a *baseline* which has commonly been used with this experimental task (e.g. Moore et al. 2006; Moore et al, 2008; Moore et al., 2012). The adopted baseline was the X that was presented subsequent to digit sequence EEEE as this was considered the most neutral stimulus presented to participants (i.e. a stimulus signalling a rest at the end of a period of largely task irrelevant stimuli - see Table 1). Thus, for each waveband, there was an average power value for each of the 20 digit types (which comprised the 5 digit sequences) and the 5 X types (which followed the five digit sequences) for each ROI.

Table 1. Digit sequence types. Examples of digits in each digit sequence are shown in brackets. 'Seq. 5' is the target digit sequence. Participants pressed the response key as soon as they saw the final odd digit in that digit sequence therefore this is the stimulus linked to *response execution*. Other odd digits in digit sequences (especially digit sequences 4 and 5) were reasoned to increase anticipation in relation to response execution. Otherwise, the final even digit in 'Seq. 4' was the stimulus linked to *response inhibition* and the X rest stimulus immediately following 'Seq. 5' (i.e. straight after response execution) was the stimulus linked to the *post response recovery* stage of the task. Lastly, the X rest stimulus following 'Seq. 1' was the *baseline* adopted in the study.

	DIGIT 1	DIGIT 2	DIGIT3 3	DIGIT 4	REST
SEQ. 1 (EEEE)	EVEN (6)	EVEN (2)	EVEN (4)	EVEN (8)	X
SEQ. 2 (OEEE)	ODD (9)	EVEN (4)	EVEN (6)	EVEN (2)	X
SEQ. 3 (OOEE)	ODD (5)	ODD (3)	EVEN (4)	EVEN (6)	X
SEQ. 4 (OOOE)	ODD (9)	ODD (7)	ODD (3)	EVEN (4)	X
SEQ. 5 (OOOO)	ODD (1)	ODD (5)	ODD (7)	ODD (3)	X

2.3 Statistical analyses

Four analyses are described below. For the first two, EEG power data were natural log transformed to achieve a Gaussian distribution in the data (Gasser, Bächer & Möcks, 1982). In those analyses, to control for Type I errors, probability levels in subsequent follow-up analyses (justified by resulting interactions in factorial ANOVAs) were treated with Bonferroni correction (Rosenthal, Rosnow &

Rubin, 2000). In the final 2 analyses described below, untransformed (i.e. not log transformed) data were entered into analyses.

The first analysis considered all alpha and beta EEG power data for each of the digits within each digit sequence in relation to baseline EEG power. This consisted of five factorial ANOVA (i.e. one for each digit sequence) with the factors region (12 levels: see description of ROIs in section 2.2) X stimulus (5 levels: digit positions 1-4 or baseline X) X waveband (4 levels: lower alpha, upper alpha, lower beta, upper beta).

A second analysis was deployed assessing post response recovery in the alpha and beta wavebands. This focused on the response execution stimulus and post response recovery stimulus. This analysis consisted of a factorial ANOVA for the power data with the factors region (12 levels: as described above) X stimulus (2 levels: response execution or post response recovery) X waveband (4 levels: as described above).

Thirdly, we considered the relationship between performance and EEG data. Mean response time (RT) scores to detected targets were considered in these analyses. Firstly, the degree to which each participant followed a predicted trend was quantified (trend value; TV). The TV was characterised by desynchronisation as the digit sequence more closely resembled OOOO (i.e. digit sequence EEEE being associated with the smallest desynchronisation, and digit sequence OOOO the biggest; with digit sequences between these extremes following this relative trend). This follows analyses we deployed in Moore et al. (2008) for lower and upper alpha. Here, we have also included lower and upper beta as we anticipate beta power following a similar respond related trend to the alpha wavebands (after Andrew & Pfurtscheller, 1999; Pfurtscheller, Neuper & Krausz, 2000; Stancák Jr. & Pfurtscheller, 1996). Calculation of the TV is described in the caption text of Table 2. A TV was calculated independently for lower alpha, upper alpha, lower beta and upper beta power data which yielded forty eight trend values per participant (i.e. 12 ROIs x 4 wavebands). These trend values were

then correlated (Pearson) with participant mean RT scores and a Bonferroni correction was applied to the probability levels to control for Type I error.

Table 2. Calculation of trend value (TV). For each participant, EEG power data were summed for each digit sequence and were correlated (Kendall) with the predicted rank. This yielded a single integer between 1 and –1 expressing the degree to which each individual participant followed the expected trends - the TV. 1 and -1 indicated a perfect positive and perfect negative relation to the predicted trend respectively.

Predicted Rank	Lower alpha	Upper alpha	Lower beta	Upper beta
1	OOOO	OOOO	OOOO	OOOO
2	OOOE	OOOE	OOOE	OOOE
3	OOEE	OOEE	OOEE	OOEE
4	OEEE	OEEE	OEEE	OEEE
5	EEEE	EEEE	EEEE	EEEE

The aim of the final analysis which we applied was to determine the degree to which participant EEG complied with expected desynchronisation or synchronisation during response execution or post response recovery respectively. For these analyses, the ROI specific alpha and beta (i.e. 8-10Hz, 10-12Hz, 12-20Hz and 20-30Hz) power values for each individual participant were considered for baseline, response execution and post response recovery. The percentage of participants who either: (a) showed a 15% reduction in EEG power for response execution relative to baseline; or (b) a 15% increase in EEG power for post response recovery relative to response execution, was calculated in relation to each region of interest[2].

3.0 Results

3.1Task performance

[2] A decrease or increase in EEG power of 15% was selected since it was reasoned that this equates to a clear decipherable EEG desynchronisation or synchronisation according to the task stage, in contrast to a random and coincidental small fluctuation in EEG power.

Performance data were reported in Moore et al. (2012) – those data will not be re-reported here but, in summary, they showed evidence that all participants complied with the task with very few errors. Additionally, there was nothing to suggest response speeds were abnormally long or short (Mean: 480.25ms; SD: 59.82).

3.2 Upper alpha desynchronisation is provoked by anticipation, response execution and response inhibition; beta desynchronisation is provoked by anticipation and response execution

When making selections for follow up analyses, interactions uncovered in the superordinate ANOVAs (i.e. those described in section 2.3) were not considered to be meaningful if they did not collectively involve at least the factors 'waveband' and 'stimulus'. The former confirmed that the interaction was waveband specific and the latter that it was related to the significance of individual stimuli within a digit sequence. The initial analyses showed that for digit sequence OOOE and OOOO there were interactions of the waveband and stimulus factors ($F(12, 396) = 4.03$, $p < 0.001$, EPS: 0.587; and $F(12, 396) = 5.85$, $p < 0.001$, EPS: 0.571, respectively). However, as none of these interactions were modulated by the region factor, follow up analyses considered mean power levels across all ROIs.

The follow up analyses revealed an upper alpha stimulus main effect for digit sequence OOOE ($F(4, 132) = 12.67$, $p < 0.001$, EPS: 0.710), and upper alpha, lower beta and upper beta stimulus main effects for digit sequence OOOO ($F(4, 132) = 31.60$, $p < 0.001$, EPS: 0.817; $F(4, 132) = 19.150$, $p < 0.001$, EPS: 0.572; and $F(4, 132) = 5.74$, $p < 0.01$, EPS: 0.730 respectively). Follow up analyses of these stimulus main effects are displayed in Figure 2.

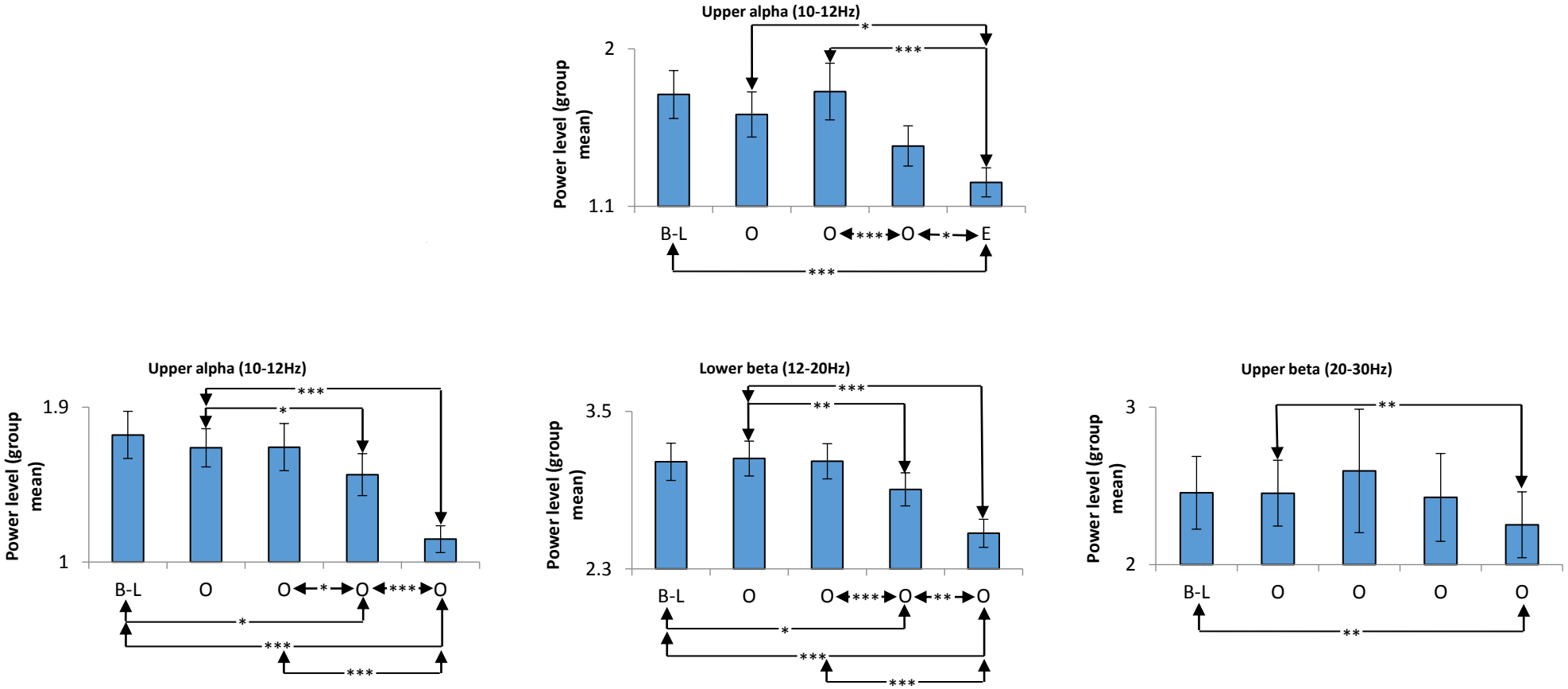


Figure 2. Mean upper alpha (10-12Hz) power associated with digit sequence OOOE (top row); mean upper alpha power associated with digit sequence OOOO (bottom row, far left); mean lower beta (12-20Hz) power associated with digit sequence OOOO (bottom row, middle) and mean upper beta power associated with digit sequence OOOO (bottom row, far right) (+/- standard errors). B-L = baseline. The arrows show significant differences between digits within each digit sequence. * = $p<0.05$; ** = $p<0.01$; *** = $p<0.001$. (N=34).

The data presented in Figure 2 show that for upper alpha, there was a significant desynchronisation during both response inhibition and response execution. Additionally, there was a significant upper alpha desynchronisation in response to odd digits provoking anticipation throughout both digit sequence OOOO and also digit sequence OOOE. Similarly, the Figure also shows evidence of this same desynchronisation trend in relation to response execution and anticipation (but not response inhibition) for lower and upper beta. However, the low number of significant effects for upper beta linked to this trend, suggest that it is more strongly characteristic of upper alpha than upper beta. Lower beta power, on the other hand shows a strong connection to this trend.

3.3 Power synchronisation immediately following movement is widespread for upper alpha, lower beta and upper beta

The second analysis (assessing post response recovery), revealed an interaction of the region, waveband and stimulus factors ($F(33, 1089) = 2.66$, $p < 0.001$, EPS: 0.423). Follow up analyses of these effects revealed a main effect of stimulus for upper alpha, lower beta and upper beta ($F(1, 33) = 43.20$, $p < 0.001$), ($F(1, 33) = 62.948$, $p < 0.001$) and ($F(1, 33) = 74.30$, $p < 0.001$ respectively) and also a stimulus x region interaction for those 3 wavebands ($F(11, 363) = 2.66$, $p < 0.001$, EPS: 0.423), ($F(11, 363) = 6.82$, $p < 0.001$, EPS: 0.661), ($F(11, 363) = 12.88$, $p < 0.001$, EPS: 0.594) respectively).

Regarding the stimulus main effects for upper alpha, lower beta and upper beta, it was always the case that mean (i.e. scalp-wide) EEG synchronised during the post response recovery period following response execution: for upper alpha - response execution = 1.133μV (SD: 0.078) and post response recovery = 1.537μV (SD: 0.113); for lower beta - response execution = 2.571 μV (SD: 0.107) and post response recovery = 3.276μV (SD: 0.121); and for upper beta - response execution = 2.253μV (SD: 0.209) and post response recovery = 2.703 μV (SD: 0.244).

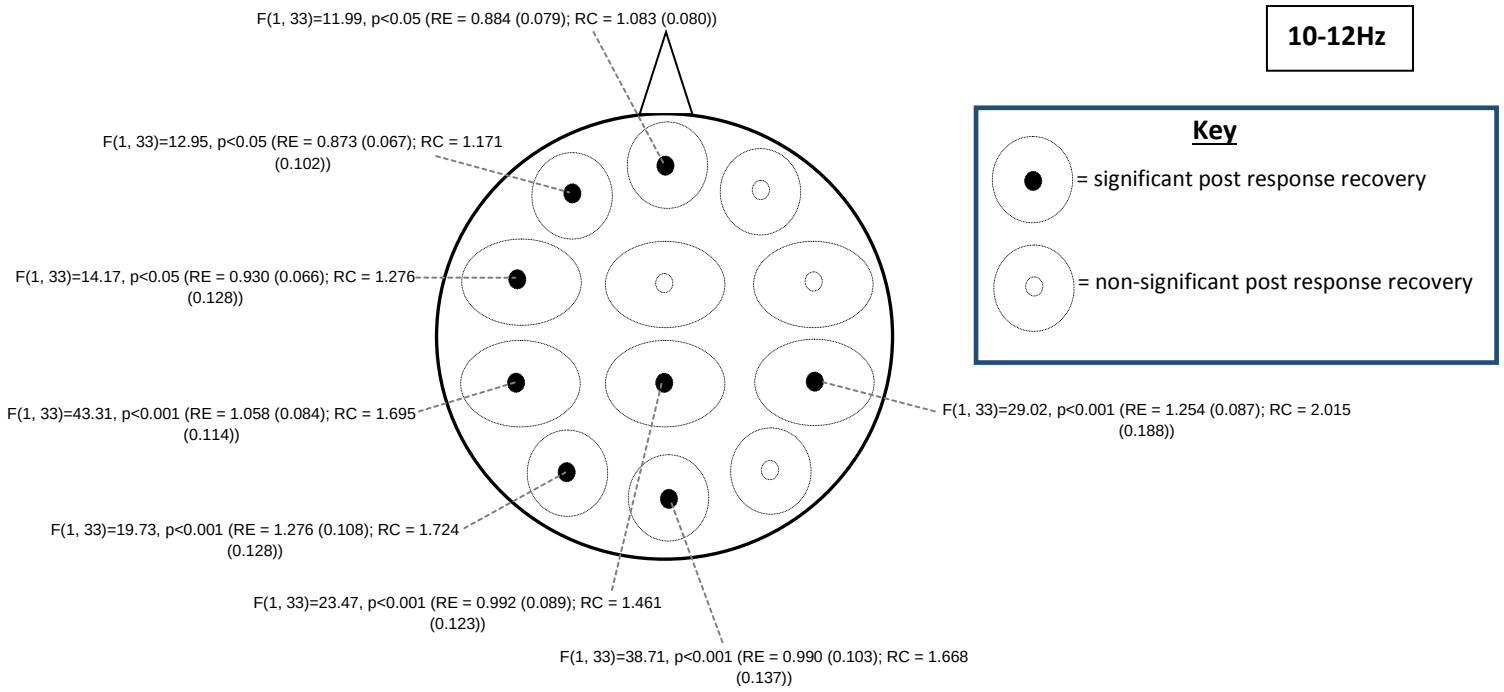
When the stimulus x region interactions mentioned above were further investigated, effects at an abundance of ROIs were revealed. In each case, these were characterised by EEG

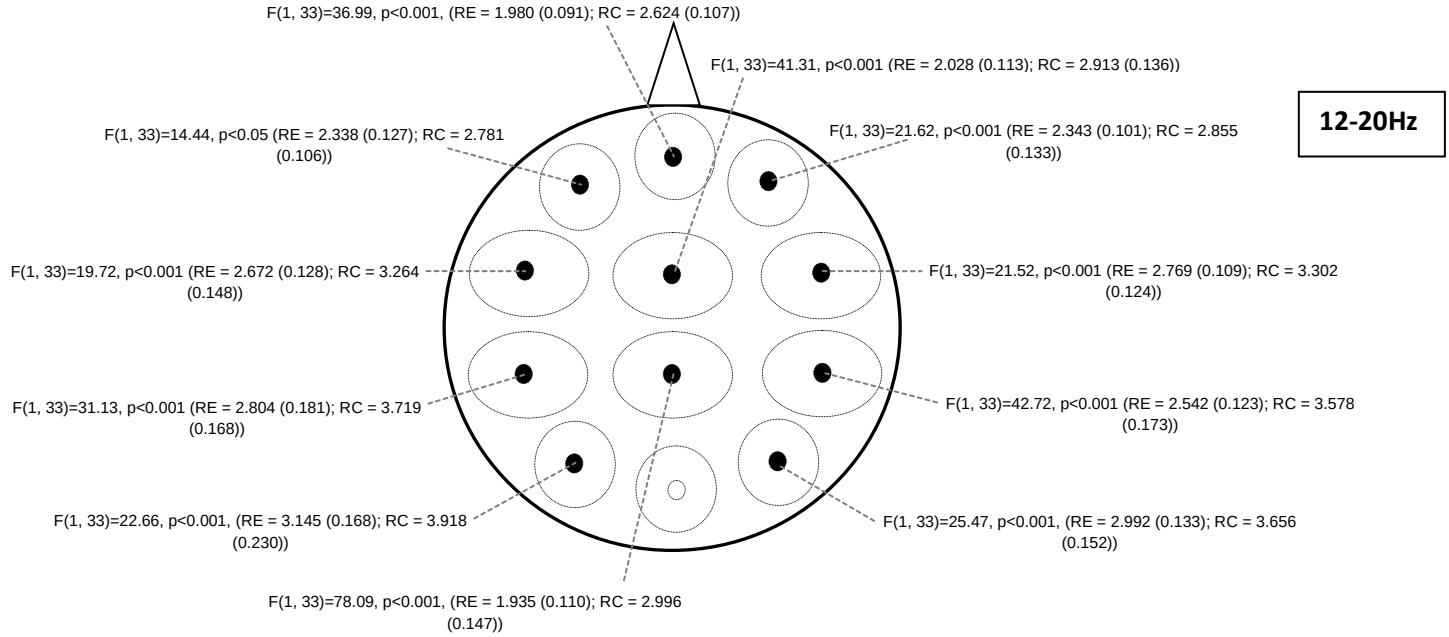
synchronisation immediately following response execution (i.e. during post response recovery). These region specific effects were revealed in each of the 3 wavebands under test (i.e. those where the stimulus x region effects reached significance). Figure 3 depicts specific details of these region specific, post response recovery synchronisation effects.

Scrutiny of Figure 3 shows that for upper alpha, the power increase linked to recovery tended to show a left hemisphere bias. For lower beta on the other hand, the effect seemed to be global as it reached significance at 11 out of 12 ROIs, whilst for upper beta there seemed to be a more centro-posterior bias with just one of the 3 frontal regions linked to a significant effect.

3.4 Mean RT is related to left centro-parietal lower alpha power

The third analysis, which described the relationship between TV (for lower alpha, upper alpha, lower beta and upper beta) and mean RT, only returned one significant result. This was in relation to 8-10Hz EEG recorded at the left centro-parietal ROI. Though this was the only correlation to reach significance from 48 independent Pearson correlations, it retained significance *after* Bonferroni correction, $r(34), = -.58$, $p < .05$. Specifically, this finding suggests that participants whose lower alpha EEG more closely displayed the predicted lower alpha trend (see Table 2) had faster mean RT scores. This shows that changes in neural activity (represented in lower alpha EEG), prior to response, predict speed of response.





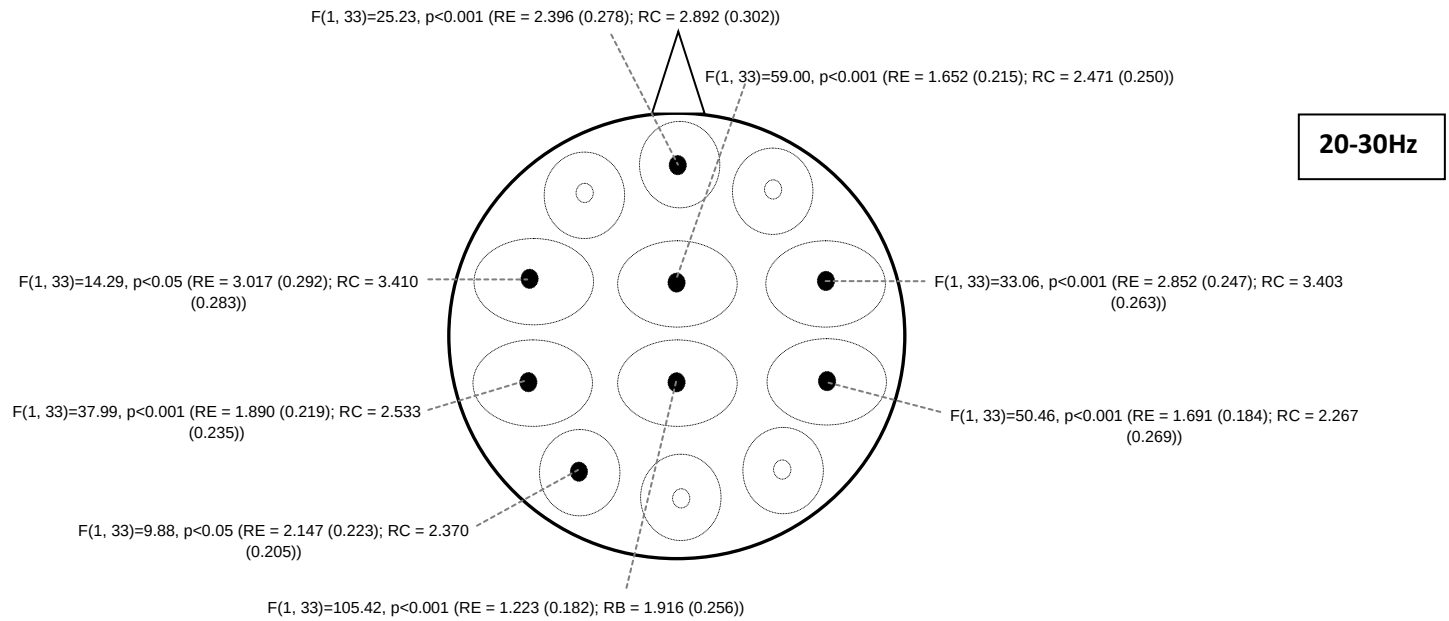


Figure 3. Region specific effects for EEG power of post movement recovery. Regions of interest (ROIs) where there was a significant EEG synchronisation during post response recovery (relative to response execution) are displayed with a black centre at each ROI. In all cases where there was a significant effect, post response recovery *always* provoked EEG synchronisation compared to response execution. Means and standard errors for response execution (RE) and post response recovery (RC) for each region are reported (standard errors shown in brackets) (N=34).

3.5 Interindividual variation in trends associated with response execution and post response recovery

Lastly the degree to which individual participants complied with typical desynchronisation and synchronisation trends linked to response execution and post movement recovery respectively was considered. The data reported in Table 3 show the percentage of the whole sample who followed the typical trends. For each ROI in each waveband, where the number of participants displaying the expected trend was greater than 75% of the whole sample (i.e. more than 25 participants out of 34), this is indicated in bold, underlined italicised text and shaded.

The data in the Table show that greater than 75% of the participants showed a (>15%) *reduction* in 10-12Hz EEG power (i.e. desynchronisation) for response execution relative to baseline at the LFC, MFC, MCP and RCP regions of interest. Additionally, greater than 75% of the participants showed a (>15%) *increase* in EEG power (i.e. synchronisation) for post response recovery relative to response execution at the MCP regions of interest for 10-12Hz, 12-20Hz and 20-30Hz along with the MFC region of interest for 20-30Hz.

Table 3. Percentage of individual participants showing a typical response execution or post response recovery trend (desynchronisation and synchronisation respectively) according to a criterion 15% reduction or increase in EEG power for each ROI in each waveband (N=34).

	8-10Hz		**10-12Hz**		**12-20Hz**		**20-30Hz**	
ROI	**BL > RE**	**PRR>RE**	**BL > RE**	**PRR>RE**	**BL > RE**	**PRR>RE**	**BL > RE**	**PRR>RE**
LF	47.06	29.41	70.59	52.94	44.12	52.94	35.29	35.29
MF	44.12	29.41	58.82	55.88	58.82	70.59	52.94	64.71
RF	41.18	38.24	50.00	44.12	47.06	52.94	38.24	50.00
LFC	32.35	41.18	***88.24***	58.82	52.94	61.76	29.41	55.88
MFC	41.18	20.59	***76.47***	70.59	58.82	67.65	55.88	***82.35***
RFC	52.94	44.12	67.65	55.88	41.18	61.76	26.47	67.65
LCP	61.76	44.12	73.53	67.65	58.82	67.65	47.06	64.71
MCP	50.00	41.18	***79.41***	***79.41***	61.76	**85.29**	70.59	***88.24***
RCP	47.06	32.35	***76.47***	70.59	61.76	70.59	50.00	67.65
LPO	41.18	41.18	67.65	58.82	38.24	50.00	20.59	44.12
MPO	47.06	38.24	58.82	50.00	29.41	38.24	26.47	29.41
RPO	47.06	32.35	50.00	55.88	44.12	50.00	35.29	50.00

4.0 Discussion

There were four main findings in this study. First, mean upper alpha, lower beta and upper beta desynchronised for anticipation and response execution. This desynchronisation trend also extended to response inhibition for upper alpha. Next, synchronisation of upper alpha, lower beta and upper beta was widespread and region specific immediately following response execution (i.e. post response recovery). Third, left centro-parietal lower alpha negatively correlated with mean response time. Fourth, upper alpha desynchronisation during response execution was demonstrated over 75% of the sample individually at fronto-central and centro-parietal ROIs; additionally, post response recovery synchronisation was demonstrated by over 75% of the sample individually at the mid centro-parietal ROI for upper alpha, lower beta and upper beta and the mid fronto-central ROI for upper beta.

4.1 Alpha

4.1.1 A profile of Rolandic mu driven EEG fluctuations during response execution extending into response inhibition

Upper alpha desynchronised with respect to every other digit in digit sequence OOOO as response was executed. This was also the case for digit sequence OOOE where the stimulus provoking the majority of significant effects was response inhibition. The former of these effects is mainly consistent with classic desynchronisation of Rolandic mu circuitry during movement (Andrew & Pfurtscheller, 1997; Babiloni et al., 2016; Holler et al., 2013; Manganotti et al., 1998; Pfurtscheller & Berghold, 1989; Leocani, Toro, Manganotti, Zhuang & Hallett, 1997; Rappelsberger, Pfurtscheller, & Filz, 1994).

However, it is important to note that the effects reported here reflect *mean* upper alpha EEG activity from all of the ROIs, rather than just those above the traditional Rolandic mu regions.

The conservative data analysis approach adopted in this study (in which data from a full scalp topography were considered rather than selective regions close to somatosensory cortex) did not justify follow up analyses at individual ROIs (see Section 3.2 for details). Hence, follow up analyses were applied to mean upper alpha EEG activity. Therefore, fluctuations linked to classic Rolandic mu desynchronisation (i.e. close to somatosensory cortex) during anticipation, response execution or response inhibition (see below), could be conflated with upper alpha EEG activity recorded more generally across the scalp. Therefore, despite a consistent pattern of response, these findings should be treated with caution as an index of classic Rolandic mu activity per se. However, it should also be noted that it is not uncommon to see distributed patterns of alpha desynchronisation during movement (i.e. beginning centrally and extending to frontal and parietal regions) where a broad topography of electrodes has been sampled (e.g. Alegre, Gurtubay, Labarga, Itiate, Malnda & Artieda, 2004).

The latter of the effects (i.e. upper alpha desynchronisation during response inhibition) is puzzling as Rolandic mu rhythm desynchroinisation is typically not linked to response inhibition (or inhibition of movement). More generally, alpha synchronisation during response inhibition would typically be predicted. For instance, Pfurtscheller, Stancak and Neuper (1996a) suggested that when alpha EEG synchronises at specific electrodes sites, it is because the related cortical regions are in a state of idling (i.e. inactive). As support for their view, they cite results from studies focussing on the Rolandic mu activity during tasks which did not require movement (e.g. Brechet & Lecasble, 1965; Koshino & Niedermeyer, 1975; Pfurtscheller & Klimesch, 1992). Typically, these studies report enhanced (synchronised) Rolandic mu rhythm activity during non-movement task stages. This was interpreted by Pfurtscheller et al. (1996a) as evidence of cortical idling of specific regions during periods when their input is not required.

Similarly, synchronisation would be predicted by the 'inhibition-timing hypothesis' (Klimesch, 2012; Klimesch, Sauseng and Hanslmayr, 2007). This view proposed that inactive brain

regions are inhibited (through alpha synchronisation) whilst regions released from inhibition are indexed by alpha desynchronisation. The process follows specific temporal organisation and supports a role for alpha in accessing a broad range of stored information (termed a "Knowledge System" by Klimesch, 2012). Klimesch et al. (2007) linked this to movement and proposed that alpha associated with brain areas linked to movement (i.e. the Rolandic mu regions) will synchronise during inhibition. One study is cited to support their view (Hummel, Andres, Altenmüller, Dichgans & Gerloff, 2002), in which synchronisation of EEG in the upper alpha range was found in a task condition where participants were told to inhibit sequential finger movements; this was in contrast to a task condition in which participants freely performed the finger movements which were accompanied by alpha desynchronisation.

The data in the current study challenge aspects of the views of Pfurtscheller et al. (1996a) and also Klimesch et al. (2007). However, differences between the task used in the current study and those cited as evidence by Pfurtscheller et al. (1996a) and Klimesch et al. (2007) may shed light on the discrepancy. For instance, in the current study participants were on the verge of response execution just before response inhibition was signalled; in this sense, response inhibition was rapid and largely unprepared. However, in the case of Hummel et al. (2002) (cited by Klimesch et al., 2007), participants knew, at the outset, response inhibition was required, meaning it was less rapidly deployed and also was consciously prepared. Additionally, in the studies cited by Pfurtscheller et al. (1996a), alpha synchronisation was recorded from sites unlikely to be active due to the nature of the task (i.e. from the hand area of the motor cortex during a reading task or the during foot movement) so, similar to the Hummel et al. (2002) study, inhibition was prepared.

Therefore, the degree to which inhibition is rapidly deployed and prepared could be relevant. For instance, in this study, where response inhibition is rapid and largely unprepared upper alpha desynchronised exactly as if movement had been deployed. However, if inhibition is less rapid and consciously prepared, the alpha synchronises above relevant brain regions (i.e. in a manner

consistent with Pfurtscheller et al.'s, 1996a, Klimesch et al.'s, 2007, and Klimesch's, 2012, viewpoints). This proposal does not necessarily undermine the cortical idling view proposed by Pfurtscheller et al. (1996a) or Klimesch et al.'s (2007) and Klimesch's (2012) inhibition timing view, but the data reported here suggest that the specific dynamics of response inhibition are important and such an anomaly would need to be accounted for in both perspectives.

4.1.2 Post movement alpha synchronisation may reflect prepared response withdrawal

Upper alpha power synchronisation during the rest period (i.e. immediately after response execution) showed a distinct left hemisphere bias. These data reflect a post response recovery and the left sided bias may have been the expected outcome considering the right handed movement when participants executed motor response. Post response recovery is typically associated with the beta waveband so this was not something for which we had formed a specific hypothesis. However, this finding for upper alpha is not altogether surprising as centrally recorded upper alpha and beta often show similar trends during movement (e.g. Pfurtscheller, Stančak & Edlinger, 1997).

Also, alpha resynchronisation after extreme task related desynchronisation is a phenomenon which has been previously reported (e.g. Woertz, Pfurtscheller, & Klimesch, 2004) and data supporting the idea that alpha synchronisation and desynchronisation reflects activation and deactivation of underlying sensorimotor regions has also been previously reported (Neuper, Wortz & Pfurtscheller, 2006). The anterior and central brain regions likely to have been involved in the planning and anticipatory phases leading up to response execution (i.e. the prefrontal cortex, the supplementary motor area, the premotor cortex, the primary motor cortex) reflect the pattern of significant results reported here. In this sense, alpha synchronisation reflects brief, deactivation of these regions during rest.

Further, an alpha synchronisation response would be expected when movement is consciously inhibited following both Pfurtscheller et al.'s (1996a), Klimesch et al.'s (2007) and

Klimesch's (2012) views outlined previously. The synchronisation of ROIs close to brain regions associated with planning and executing movements were explained above; those close to the occipital cortex may reflect a brief suspension in visual processing during the short rest period when the X is presented.

4.1.3 Neural efficiency indexed in mean RT is reflected in EEG alpha

There was a localised relation between lower alpha EEG power and RT. Specifically, participants displaying the lower alpha EEG trend detailed in Table 2 were also those recorded as having the faster mean RT scores. This effect was localised to the left centro-parietal ROI. Of all of the ROIs considered, when the contralateral organisation of the primary motor cortex is taken into account, EEG data recorded at the left centro-parietal ROI would logically have been expected to be the most likely to show such a relation with RT scores.

These data provide evidence of functional links between physiology and measurable behaviour and can be considered alongside other studies indexing efficiency of neural processing. For instance, Haier, Siegel, Tang, Abel and Buchsbaum (1992) reported that magnitude of glucose metabolic rate change at a number of brain regions during learning of a 'tetris' computer game positively related to intelligence scores. Similarly, handwriting quality in children has been linked to progressive activation of the right inferior frontal gyrus (Giminez et al., 2014). Additionally, other EEG studies have been reported previously which show similar relationships between performance data and physiology (e.g. Bablioni et al., 2010; Doppelmayr, Klimesch, Hődlmoser, Sauseng & Gruber, 2005; Micheloyannis et al., 2006) and which also can all be considered to be evidence of neural efficiency.

4.2 Beta

4.2.1 Lower beta EEG power acts as a sensitive index of the central beta rhythm during anticipation and response execution

As expected, lower beta desynchronised significantly during motor response. This finding is in line with results reported in a number of studies reporting similar findings for beta (i.e. Babiloni et al., 2016; Höller et al., 2013; Kilavik, Zaepffel, Brovelli, MacKay & Riechle, 2013; Pfurtscheller, 1981; Muller, Neuper, Rupp, Keinrath, Gerner & Pfurtscheller, 2003; Stancek & Pfurtscheller, 1996; Leocani, Toro, Manganotti, Zhuang & Hallett, 1997) many of which define beta in a range which overlaps with the lower beta definition used in the current study (e.g. Muller, Neuper, Rupp, Keinrath, Gerner & Pfurtscheller, 2003; Stancek & Pfurtscheller, 1996). Therefore, it can be tentively proposed that these data reflect commonly reported classic central beta rhythm blocking during motor activity.

Additionally, lower beta desynchronised as stimuli indicating approaching response execution (i.e. odd digits within a digit sequence prior to response execution) were presented, showing that lower beta power also reacted to anticipation as well as response execution. This lower beta power trend mainly matches the upper alpha trend discussed above (section 4.1.1) demonstrating that beta activity (particularly linked to the lower beta) and upper alpha follow comparable anticipation and response execution related trends. In relation to lower beta, desynchronisation linked to anticipation is not new and has been reported several times before (e.g. Alegre, Gurtubay, Labarga, Iriate, Malanda & Artieda, 2003; Pfurtscheller & lopes Da Silva, 1999; Stancak & Pfurtscheller, 1996). However, we believe this study is novel in demonstrating the progressive and graduated nature of the lower beta's anticipation response over repeated stimuli leading up to response execution.

For instance, in their review of beta activity during movement, Kilavik et al. (2013) described typically studied task epochs as pre-cue, post-cue, pre-Go, movement and hold/relax. Drawing on range of data and studies, they produced a representation of expected sensorimotor recorded beta activity across these stages (see their Figure 3). For instance, at the initial stage (pre-cue), they described a synchronised beta response leading to a progressive pattern of desynchronisation

(reaching a minimum during movement) from the post-cue stage right through to pre-Go and movement stages. Following the movement stage, they described a rapid synchronisation following movement end. The lower beta data reported here generally map onto this model leading up to response execution, though the task used in the current study effectively included 3 pre-Go phases (within digit sequence OOOO).

Upper beta showed a different profile of response. Whilst upper beta also desynchronised during response execution, it was not sensitive to anticipation in the same way as lower beta. Therefore, this indicates that lower beta acts as a more finely tuned index of preparation of a pre-potent response (and desynchronisation of the beta rhythm) than upper beta. Therefore, whilst lower beta is sensitive to anticipation and response execution, upper beta is only responsive to response execution.

However, it is important to point out that, as well as the alpha data reported in Section 4.1.1, results linked to these lower and upper beta data are also based on *mean* response rather than region specific response. Therefore, as beta activity recorded from the central ROIs has been conflated with beta response from other ROIs, any conclusions derived implicating the central beta rhythm should be treated with caution.

4.2.2 *Post movement EEG beta – maintaining the response set and status quo*

Strong evidence was also found of post movement beta synchronisation for both upper and lower beta. This finding is supportive of the many other studies which have shown that EEG beta quickly synchronises following movement (e.g. Juriewicz, Gaertz, Bostan & Cheyne, 2006; Pfurtscheller, Stancak & Neuper, 1996b; Pfurtscheller, Stancak & Edlinger, 1997; Stancak & Pfurtscheller, 1996). However, the locations of the effects are surprising as we have recorded significant beta synchronisation at a broad range of regions of interest; typically studies relating to this issue report post movement beta synchronisation as having a distinct pre-central topography. However, it is not

unusual for studies to report distributed beta effects linked to movement when EEG is sampled broadly across the scalp (e.g. Chung et al., 2017; Höller et al, 2013; Picazio et al.,2014). Therefore, given the apparent role of beta oscillations in motor activity, it is actually surprising that previous studies investigating post response recovery (i.e. synchronisation) of beta oscillations have not often reported effects more broadly than central brain regions.

In terms of the significance of beta synchronisation, in their review of EEG beta activity, Engel and Fries (2010) draw on the results of a range of studies in which beta EEG has provided the focus. They present a view that beta relating to motor activity on one hand, and beta relating to non-motor activity on the other, perform two related roles. Specifically, they propose that when engaged in a state where no transitions are expected (i.e. during some ongoing, coordinated activity), enhanced beta oscillations promote and maintain the existing motor set while acting to specifically compromise neural processing of new movements that may be detrimental to performance. Various studies are offered as evidence for this position but, perhaps, the most convincing are those which show the effect of beta oscillations in patients with Parkinson's disease (e.g. Kűhn et al., 2008; Wingeier et al., 2006). Specifically, these studies show that high levels of beta activity can be reduced in Parkinson's patients (using deep brain electrical stimulation for instance) which, in turn, improves movement capability.

Therefore, in relation to our data, beta synchronisation which emerges just after motor response during the brief delay period could be actively preventing neural processing of new or distracting movements which could compromise the participant's performance in the ongoing activity in which they are engaged. Such a mechanism would help to protect the existing motor set whilst making it more impervious to distraction in the process. We would suggest that this interpretation could possibly be applied to the regions showing beta enhancement which are closest to the sensorimotor cortex.

Similarly, Engel and Fries (2010) suggest that, on a cognitive level, beta activity is enhanced when the current 'status quo' (i.e. the current cognitive set) takes priority over new signals which may be deemed distractive. They suggest that in a top down, endogenous situation (i.e. where performance is determined in terms of adherence to internally held parameters, rules and conditions) enhanced beta activity would be expected in delay periods where the cognitive set has to be maintained during a delay. This is in contrast to a bottom up situation (i.e. where task performance is largely stimulus driven) where beta activity would not be expected to increase in a delay period. Various studies which show increased beta activity in relation to top down processing relative to bottom up (e.g. Buschman & Miller, 2007; 2009) are offered by Engel and Fries (2010) as evidence for this position.

In terms of the data we have reported, it could be the case the cognitive set linked to the experimental task is being maintained whilst the participant is anticipating the next stage of the task. In this sense, our data support the account offered by Engel and Fries (2010). Finally, it is also worth adding that Engel and Fries's (2010) account of the functional significance of enhanced beta activity is an attractive extension to other views which see beta synchronisation as simple cortical idling (i.e. Pfurtscheller et al. , 1996b), since Engel and Fries's (2010) account deals with beta synchronisation at both a motor and cognitive level.

4.3 Considerations concerning individual desynchronisation and synchronisation response

The individual analysis revealed upper alpha as the waveband in which the greatest number of participants showed a typical desynchronisation trend during response execution. In fact, upper alpha was the only waveband in which this desynchronisation trend was demonstrated by greater than 75% of the participants. In relation to brain regions, the ROIs where this was reflected were typically close to central cortical regions (i.e. close to the somatosensory region), with the left fronto-central ROI (i.e. contralateral to the right hand response) resulting in the highest percentage magnitude overall. There was one unexpected ROI which also displayed the expected trend in upper

alpha for over 75% of the participants that was located in one of the ipsilateral ROIs (right centro-parietal) which, whilst consistent with a post somatosensory region linked to Rolandic mu oscillations, would not have been expected due to its ipsilateral location. However, this could simply be a function of ipsilateral desynchronisation of the Rolandic mu rhythm which has been previously reported (Derambure, Defebvre, Bourriez, Cassim, Guieu, 1999; Storm van Leeuwen, Arntz, Spoelstra & Wieneke, 1976).

In relation to the typical synchronisation pattern displayed during post response recovery, EEG recorded from midline central ROIs produced evidence of this typical response in greater than 75% of the participants in lower beta, upper beta and upper alpha. However, this was strongest in lower beta and upper beta recorded from mid centro-parietal brain regions for both, followed by the mid fronto-central brain region for upper beta only. In terms of expectation, beta synchronisation is most often linked to post response recovery and, hence, it is no surprise that these are the wavebands (i.e. lower and upper beta) in which the greatest number of participants displayed the typical trend. However, the mid centro-parietal region is a little surprising as previous research has identified post response recovery beta oscillations anterior to the midline (e.g. Pfurtscheller & lopes Da Silva, 1999) , reflecting the pre-Rolandic basis of central beta response. The finding that greater than 75% of the participants displayed the typical trend for post response recovery in the mid fronto-central region too, is more consistent with the pre-Rolandic mu basis of the central beta rhythm however.

This interindividual level analysis was conducted following other studies that have shown variability in relation to the degree to which individuals follow typical patterns of response (i.e. Holler et al., 2013; Solis-Escalante et al., 2012). The current data support previous studies by showing that there are a proportion of participants who do not display typical desynchronisation or synchonisation trends. This issue has particular relevance for clinical uses such as BCI applications. Additionally, considering its high test-retest reliability (Espenhahn et al., 2017) beta oscillations

relating to movement have a high potential to act as an index of motor system function and dysfunction in clinical settings.

In this sense then, the data presented here concerning the degree to which individuals display the typical trends would, along with other studies showing a potential lack of response in some participants, act as caution during interpretation of any motor dysfunction diagnosis based on expression of either a typical desynchronisation or synchronisation trend. However, on a more positive note, the data presented here provide hints at cortical regions which provide the best opportunity to find the typical response if it is displayed by a patient under test.

4.4 Conclusion

Alpha and beta synchronisation and desynchronisation have been studied during a single experimental task containing three of the most commonly studied aspects of movement related behaviour: pre-movement (preparation), peri-movement (during) and post-movement (recovery). Additionally, the degree to which individual participants show typical response patterns associated with response execution and post response recovery has been identified. Data have been reported which replicate previous findings along with novel findings which build on previous studies in this research area.

Alpha power followed a trend which we have related to classic Rolandic mu rhythm desychronisation during response execution, and also shows a post response recovery trend (indexed through synchronisation) consistent with previous accounts of alpha's role in neutral dynamics (e.g. Pfurtscheller et al., 1996a; Klimesch et al., 2007; Klimesch's, 2012). However, a novel trend for upper alpha was also reported where it was found that rapid and largely unplanned response inhibition leads to a desynchronisation response similar to that expected for response execution – this presents a challenge to these accounts. A further finding was reported for alpha (which replicated Moore et al., 2008) that demonstrated a link between neural efficiency and

response. Specifically, participants whose lower alpha desynchronised in alignment with anticipatory odd digits also had the fastest RT scores.

Oscillations in the beta frequency range primarily reacted as expected, namely desynchronisation during preparation for response and response execution. Lower beta was revealed as a more sensitive movement index than upper beta since it was responsive to anticipation and response execution, compared to upper beta which was only responsive to response execution. The strongest effect for the beta was found for post response recovery which showed a wide topographical distribution for both lower and upper beta. Following Engel and Fries (2010), we speculated that post response recovery synchronisation played a dual role of preventing primary motor cortex processing of new or distracting movements and maintaining the cognitive set linked to the experimental task in distributed brain regions whilst the participant is anticipating the next stage of the task.

Finally, the interindividual analysis adds further support to the variable nature with which typical alpha and beta desynchronisation and synchronisation responses are reproduced on an individual level. This is an important issue concerning clinical applications of these brain rhythms in relation to movement, especially where studies based on pooled data act as the main source of reference. However, the broad topography which has been considered in this analysis provides useful clues about the best site from which to identify typical response in either of these wavebands during clinical applications.

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