Title: Do movement-related beta oscillations change following stroke?

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Contribution: HER designed, acquired and analysed data and wrote manuscript, MHB helped design and acquire MEG and MRI, NSW helped design the study and co-wrote manuscript.

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Running head: Movement-related beta oscillations following stroke

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Abstract:

Stroke is the most common cause of physical disability in the world today. Whilst the key element of rehabilitative therapy is training, there is currently much interest in approaches which ‘prime’ the primary motor cortex to be more excitable, thereby increasing the likelihood of experience-dependent plasticity. Cortical oscillations reflect the balance of excitation and inhibition, itself a key determinant of the potential for experience-dependent plasticity. In the motor system, beta band oscillations are important and are thought to maintain the resting sensorimotor state. Here we examine motor cortex beta oscillations during rest and unimanual movement in a group of stroke patients and healthy controls using magnetoencephalography. Movement-related beta desynchronization (MRBD) in contralateral primary motor cortex was found to be significantly reduced in patients compared to controls. Within the patient group, smaller MRBD was seen in those with more motor impairment. We speculate that impaired modulation of beta oscillations during affected hand grip is detrimental to motor control highlighting this as a potential therapeutic target in neurorehabilitation.
**Introduction:**

Stroke is the most common cause of physical disability in the world today. Whilst the key element of rehabilitative therapy is training, there is currently much interest in approaches which ‘prime’ the primary motor cortex (M1) to be more excitable, thereby increasing the likelihood of experience-dependent plasticity (Hagemann et al. 1998). Examples of this include non-invasive brain stimulation (Hummel & Cohen 2006; Ayache et al. 2012), active-passive bimanual movement therapy (APBT) (Stinear & Byblow 2004) or pharmacological interventions (Molina-Luna et al. 2009; Chollet et al. 2011). Understanding the mechanisms of these interventions and which stroke subtypes they work best in will help in delivering them to appropriate patients.

Currently, changes to M1 excitability can be assessed using transcranial magnetic stimulation (TMS) which measures the effect of experimental ‘priming’ interventions on motor evoked potentials (MEPs) in the affected limb. One disadvantage of TMS is that it cannot be used in patients with absent MEPs in the paretic arm. Furthermore, the technique is most commonly performed at rest which does not provide information about task-related changes in cortical excitability.

Previous studies exploring task-related activity after stroke have predominantly used fMRI. These studies have shown that patients with greater impairment tend to have more widespread activity across the motor network (including contralesional motor cortex) (Ward 2011; Rehme et al. 2012). One potential problem in using fMRI after stroke is that it relies on intact neurovascular coupling to generate the BOLD signal (Blicher et al. 2012). An alternative method of examining motor cortex activity that does not rely on neurovascular coupling is magnetoencephalography (MEG). MEG is an excellent technique for exploring the oscillatory dynamics in the cortex in more detail (Lopes da Silva 2013). Beta oscillations (15-30Hz) are present during rest in the primary motor cortex (Engel & Fries 2010). Just prior to a movement, the strength of beta oscillations decreases and then returns to a level above baseline following movement (Pfurtscheller & Lopes da Silva 1999). Movement-related beta power decrease (MRBD) has been linked to M1 excitability in recent EEG-TMS studies (Aono et
al. 2013; Takemi et al. 2013) and both the strength and frequency of oscillations are influenced by
levels of the inhibitory neurotransmitter GABA (Muthukumaraswamy et al. 2009; Hall et al. 2010;
Muthukumaraswamy et al. 2012). These results suggest that MEG might be a useful tool for studying
the balance between inhibition and excitation in the human cortex.

Changes in beta-band oscillations have been seen in a number of settings. For example, enhanced
beta oscillations have been seen as part of the aging process (Rossiter et al. 2014). Patients with
Parkinson’s disease exhibit abnormal beta oscillations in both basal ganglia (Kühn et al. 2004) and
motor cortex (Heida et al. 2014) that they are unable to suppress when trying to initiate a
movement. These findings have led to the suggestion that this abnormal beta-band activity is
pathological and results in abnormal persistence of some sensorimotor states and therefore
impairment of flexible motor control (Engel & Fries 2010). In stroke, previous studies have explored
oscillatory parameters at rest and during tactile stimulation (F Tecchio et al. 2007; Franca Tecchio et
al. 2007; Laaksonen et al. 2012) but currently there is little information on how these oscillations
change during movement of affected limbs following stroke. It appears that beta oscillations may
play a role in the pathology of diseases affecting movement and are therefore worthy of exploration
in stroke.

In this study, we investigated cortical oscillatory signals at rest and during movement of the affected
hand in stroke patients with a range of impairments and at different times after stroke. In stroke
patients and older healthy control subjects, fMRI studies have shown task-related activation in both
ipsilesional and contralesional primary motor cortex during unilateral hand movement (Ward 2011;
Rehme et al. 2012). Bilateral MRBD with unilateral movement has been also seen in healthy control
subjects using MEG (Pfurtscheller & Lopes da Silva 1999; Jurkiewicz et al. 2006). We therefore
examined beta oscillations in motor cortices of both hemispheres. We hypothesised that beta
oscillations would be diminished after stroke both at rest and during movement and that this would
be more apparent in those with greater impairment.

Methods:
Subjects:

Twenty-five stroke patients (mean age 49±13 years, range 19-70 years; 7 female, 3 left-handed, 14 dominant-hand affected) participated (see Table 1 for more detailed demographic information). All patients suffered from first-ever stroke with weakness of at least wrist and finger extensors and hand interossei. We excluded patients with other neurological disorders; those unable to perform the grip task; those with metal implants likely to create artefacts in the MEG; those with language/cognitive deficits sufficient to impair cooperation in the experiment. Thirty-two healthy participants (mean age 51±21 years, range 22-82 years; 11 female, 2 left-handed) took part in this study (results from this healthy cohort have been published separately [Rossiter et al. 2014]). Full written consent was obtained from all subjects in accordance with the Declaration of Helsinki. The study was approved by the Joint Ethics Committee of the Institute of Neurology, UCL and National Hospital for Neurology and Neurosurgery, UCL Hospitals NHS Foundation Trust, London.

Behavioural testing:

All patients were scored on the Nine Hole Peg Test (NHPT) (Kellor et al. 1971; Oxford Grice et al. 2003), the Action Research Arm Test (ARAT) (Yozbatiran et al. 2008) and grip strength using a dynamometer. A principal component analysis (PCA) was performed on NHPT and ARAT in order to create a single motor impairment score unaffected by floor and ceiling effects in individual scores as has previously been done in our group (Ward et al. 2003a) (a lower PCA score corresponding to greater impairment).

Motor task:

Participants performed visually cued isometric hand grips with a manipulandum (Ward et al. 2006) during MEG recording. Prior to scanning, maximum voluntary contraction (MVC) was obtained for each subject. Patients used their affected hand for the task whereas control participants used their dominant hand. 60 trials were performed. The cue to perform a hand grip was the appearance of a ‘force thermometer’ on the screen which provided continuous visual feedback about the force exerted. The target force was set at 30% of their MVC and displayed visually. Each grip was sustained
for 3s with an interstimulus interval between 3 and 7 seconds. An identical manipulandum was
placed in the inactive hand to check for mirror movements.

MEG recording:
MEG signals were measured continuously at 600Hz during the task using a whole-head CTF Omega
275 MEG system (CTF, Vancouver, Canada). Head localization was monitored continuously during
the recordings in order to check for excessive movement. The MEG data were pre-processed offline
using SPM8 (Wellcome Trust Centre for Neuroimaging, www.fil.ion.ucl.ac.uk/spm) (Litvak et al.
2011). Data were down-sampled to 300Hz and were filtered from 5-100Hz. Data were epoched from
-1 to +5s where time 0 indicated onset of the visual cue. Trials with large eye blinks or other
artefacts were excluded.

Structural MRI recording:
A 3T Siemens Trio scanner (Siemens, Erlangen, Germany) was used to acquire high resolution T1-
weighted anatomical images (1.3 x 1.3 X 1.3 mm voxels); 176 partitions, FoV = 256 × 240, TE =
2.48 ms, TR = 7.92 ms, FA = 16°). Structural MRIs could not be obtained in 7 of the patients and 4 of
the healthy controls due to MRI contraindications.

Data processing and analysis:
Lead fields were computed using a single-shell head model (Nolte 2003) based on an inner skull
mesh derived by inverse-normalizing a canonical mesh to the subject’s individual MRI image
(Mattout et al. 2007). For subjects without individual MRIs, the canonical mesh was affine-
transformed to fit their MEG fiducials. Coregistration between the MRI and MEG coordinate systems
used three fiducial points: nasion, left and right pre-auricular. Whilst acquiring the structural MRI,
fiducial points were marked with vitamin-E capsules in order to coregister with the MEG fiducials.
The beamforming method is based on the linear projection of sensor data using a spatial filter
computed from the lead field of the source of interest and the data covariance (Van Veen et al.
1997).
Oscillatory changes in the beta band (15-30Hz) between rest and grip were localised using the Linearly Constrained Maximal Variance (LCMV) beamformer (Hillebrand & Barnes 2005; Vrba & Robinson 2001) as part of the SPM8 software. The passive time window was taken from -2.5s to 0s with 0 as the onset of the visual cue to move. The active time window was from 0.5s-3s following the visual onset. The source signal was then extracted from the peak change in beta power which was found in the primary motor cortices, both contralateral and ipsilateral to the moving hand. The source orientation was in the direction yielding maximal signal variance. Morlet-wavelet time-frequency analysis was used to explore the changes in beta across a trial from these locations. These were rescaled in order to show percentage change from baseline (-1 to 0s) and averaged across trials. The mean percentage decrease in beta power (15-30Hz) was then extracted from the 3s movement period for each participant, the percentage beta decrease in contralateral M1 was divided by the percentage beta decrease in ipsilateral M1 in order to create a ratio (MRBD ratio). The absolute baseline beta power (-1s to 0s) was also obtained. In total, 9 different beta parameters were used for subsequent analysis: baseline beta (peak frequency and amplitude) from both motor cortices, MRBD (peak frequency and amplitude change) from both motor cortices, and MRBD ratio. In order to determine differences in beta parameters between the two groups, we performed 2-sample t-tests. In order to examine the relationship between beta parameters and impairment, we performed multiple linear regression, using beta parameters, age, time since stroke and absolute grip strength as explanatory independent variables. Grip strength and time after stroke were not found to explain any of the beta parameters in an exploratory stepwise regression and so were removed from subsequent analysis.

**Results:**

All patients were able to perform the grip task adequately (the average force was 33±3% for patients and 32±1% for controls) and no mirror movements were observed in either group. Demographic information for the patients is detailed in Table 1.
A change in beta power was seen between rest and grip in all participants in both contralateral and ipsilateral M1. The location of these peaks for the patient group can be seen in Figure 1.

There was no significant between group difference in baseline beta amplitude or frequency in either contralateral or ipsilateral M1. However, MRBD was significantly smaller in patients compared to controls in contralateral (2-sample t-test, p=0.005) (Figure 2) but not ipsilateral M1. The MRBD ratio was also significantly lower in patients compared to controls (2-sample t-test, p=0.02). There was no between-group difference in frequency of peak MRBD in contralateral or ipsilateral M1.

We then examined whether beta parameters correlated with motor impairment in the patient group. Neither power nor frequency of baseline beta oscillations, in either hemisphere, was related to motor impairment. However, MRBD in contralateral (but not ipsilateral) M1 correlated negatively with motor impairment (standardized regression coefficient ($\beta$)=-0.52, p=0.008, $r^2=0.26$). In other words, there was a smaller reduction in beta power during affected hand grip in patients with more impairment. Furthermore, the MRBD ratio correlated positively with motor impairment ($\beta=0.42$, p=0.04, R2=0.17) (Figure 3). Peak frequency of MRBD in contralateral and ipsilateral M1 did not correlate with impairment.

All significant correlations were found whilst controlling for age as a factor (age was included as a covariate in the multiple linear regression). None of the beta parameters were significantly explained by age alone.

**Discussion:**

In this study, we investigated how motor cortex beta oscillations are affected in stroke patients with a range of impairments and at different times after stroke. We report two new key findings: (i) Firstly, contralateral (ipsilesional) M1 MRBD was diminished in patients compared to controls although there was no difference in baseline beta power between groups. (ii) Secondly, in the stroke patients, greater impairment was associated with lower contralateral (ipsilesional) M1 MRBD (and MRBD ratio). No difference was seen in baseline beta power between patients and controls. The only significant differences found were during dynamic changes across movement. This illustrates
the importance of examining state-dependent dynamics during task-related movements, something that is better performed with MEG/EEG than with techniques such as fMRI and MRS which have lower temporal resolution. Our results suggest that these oscillations may have an important role in the mechanisms of impairment of motor control after stroke.

FMRI studies have found more widespread activation in motor networks following stroke and specifically more bilateral recruitment of M1 (Ward et al. 2003b). With good recovery, this tends to revert back to a pattern similar to that seen in healthy controls where the majority of the activation is seen in the contralateral M1 (Dijkhuizen et al. 2003; Ward et al. 2004). Our results indicate that those with more impairment have a more bilateral decrease in beta power across the primary motor cortices during unilateral movement and those with less impairment have more ‘activity’ (larger MRBD) in the contralateral M1 which is similar to the pattern seen in the above fMRI studies. Our results suggest a role for ipsilateral M1 MRBD during movement generation in those stroke patients with more impairment. Given that alterations in network connectivity have also been seen after stroke (Grefkes et al. 2008; Volz et al. 2014) it would be interesting to explore the relationship between oscillatory dynamics in different areas and levels of motor impairment.

Whilst stroke patients and Parkinson’s disease patients have very different pathologies, nonetheless they both show a diminished MRBD in the motor cortex contralateral to the affected hand together with deficits in some aspects of motor control. Clearly the underlying pathophysiology in both conditions is different, but it may be that both groups lack the ability to modulate motor cortex beta power during movement which then reduces their ability to generate volitional descending motor signals.

The amplitude of baseline beta power and MRBD have both been linked to levels of GABA (Hall et al. 2011; Gaetz et al. 2011; Muthukumaraswamy et al. 2012). The spectral characteristics of MEG data therefore have the potential to inform us about cortical inhibitory and/or excitatory processes which themselves are important determinants of the potential for experience dependent plasticity (Benali et al. 2008). Furthermore, approaches to modelling these spectral data will allow inferences about
synaptic physiology in humans in vivo (Moran et al. 2009; Moran et al. 2011; Bastos et al. 2012). Understanding the link between these oscillatory measures, the balance between inhibitory and excitatory processes and the potential for experience dependent plasticity is likely to be important in understanding the mechanisms of ‘priming’ approaches such as non-invasive brain stimulation (NIBS) and pharmacotherapy.

One of the limitations of this study is the variability in the patient group. The time since stroke in our cohort ranged from 1 month to 17 years (Table 1). In order to exclude the possibility that time since stroke affected the beta parameters, it was included in an exploratory stepwise multiple regression. It did not explain any of the beta parameters significantly, and we still saw a significant correlation with impairment in the patient group despite this variability. In future studies it would be of value to follow the same patient from the acute through to the chronic stage and see how these parameters altered with time and with recovery. This could tell us something about the time scale of alterations in post-stroke plasticity and the window of opportunity for intensive rehabilitation (Clarkson et al. 2010; Carmichael 2012; Zeiler et al. 2013).

Furthermore, there was a range of ages in the patient group. We have previously reported that beta oscillations are affected by age in that the baseline beta power amplitude increases with advancing age (Rossiter et al. 2014). However in this study, age did not significantly account for any of the variability in the beta parameters when added to an exploratory stepwise multiple regression suggesting that impairment explains the oscillatory dynamics over and above that due to any age effects.

There was also variability in the level of impairment in our patients, although this was something we were explicitly interested in. The amount of grip force an individual had to exert during scanning was scaled to their own maximum voluntary contraction which varied according to level of impairment. We chose to do this rather than use the same absolute force for everyone as it allowed us to study patients who were quite impaired and would otherwise not have been able to perform the task. In order to exclude the possibility that this variability in grip force may have contributed to our results
(Mima et al. 1999), we included grip strength as a covariate in an exploratory stepwise multiple regression. Variation in the beta parameters was not explained by grip strength and it was therefore removed from further analysis. The changes we see in the beta oscillations are unlikely to be accounted for by differences in absolute levels of performance.

One potential issue with this study is the variability in the peak beta coordinates and whether they can be definitively defined as M1 (Figure 1). MRBD has been localised to primary motor cortex in a number of MEG studies previously (Jurkiewicz et al 2006, Gaetz et al 2010, Hall et al 2011, Rossiter et al 2014) and so it is expected that the largest change in beta would be within M1 during this motor task. Our group has also performed this exact same task in fMRI studies and found the largest peak of activation to be within contralateral M1 (Ward and Frackowiak 2003, Ward et al 2003, Ward et al 2008), hence there is an expectation that these changes are in M1. There is also an important issue in MEG analysis relating to co-registration error, either in terms of head movement during scanning or errors in co-registration to MRI. As such, we cannot say with absolute certainty that our data relate to contralateral M1 in every case, but we feel this is the most likely scenario. Nevertheless, we felt it important to localise the peak beta change for each individual and use this coordinate for our further analysis.

In this study we chose to compare the affected hand of the patient group to the dominant hand of the controls. In a separate analysis, MRBD amplitude and frequency during grip from dominant hand were compared with non-dominant hand in our group of healthy controls using t-tests and no significant difference was found. Therefore we think it is appropriate to use the dominant hand of healthy controls for comparison with the affected hand of patients.

In summary, our results suggest that abnormalities in cortical oscillatory parameters may be an important part of the mechanism of motor impairment after stroke. Future work will be directed towards determining whether these oscillatory parameters reflect cortical inhibitory and/or excitatory processes and therefore represent biomarkers of the potential for experience dependent plasticity in the post-stroke brain.
This research was supported by the European Commission under the 7th Framework Program-HEALTH-Collaborative Project Plasticise (Contract no. 223524) www.plasticise.eu (Dr Rossiter), the Canadian Institutes of Health Research (Dr Boudrias), and The Wellcome Trust (Dr Nick Ward).

**Bibliography:**


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Figure legends:

Figure 1: Glass brain showing peak change in beta power between rest and grip with affected hand only, in both ipsilesional and contralesional M1 (grip was performed with the right affected hand – left hand grips were flipped in the sagittal plane so that all data could be included on the same plot) with each dot representing an individual. The affected hemisphere is on the left and the unaffected hemisphere on the right. Results are displayed on a ‘glass brain’ and shown from behind (top left), from the right side (top right) and from above (bottom left).

Figure 2: Boxplot displaying percentage MRBD in contralateral M1 in both the control group and patient group. These were found to be significantly different using a 2-sample t-test (p=0.005).

Figure 3: A) Scatter plot showing percentage MRBD in contralateral M1 during grip compared to baseline against motor impairment score. There was a significant negative correlation between MRBD in contralateral M1 and motor impairment score (β=-0.52, p=0.008). B) Scatter plot showing the relationship between the MRBD ratio of against motor impairment score. This correlation was significant (β=0.42, p=0.04). PCA is a motor impairment score derived from the principle component of the NHPT and ARAT tests. Higher PCA value equates to less impairment.

Patient demographics and raw behavioural scores for affected hand

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Table 1: MCA = middle cerebral artery, ARAT = action research arm test, NHPT = nine hole peg test, PCA = principal component analysis. Bottom row contains the mean±standard deviation for the age and behavioural scores.