Connectivity Analysis during Motor Imagery and Motor Execution using DCM

C. H. Kasess[1,2], R. Cunnington[3], R. Lanzenberger[2], E. Moser[1,4], L. Pezawas[2], and C. Windischberger[1,2]

[1]MR Centre of Excellence, Medical University of Vienna, Vienna, Austria, [2]Department of General Psychiatry, Medical University of Vienna, Vienna, Austria, [3]Howard-Florey-Institute, University of Melbourne, Melbourne, Australia, [4]Center for Biomedical Engineering and Physics, Medical University of Vienna, Vienna, Austria

Introduction

Numerous fMRI studies have examined differences in activation patterns during executed and imagined movements (e.g. Dechent et al. 2004, Lotze et al. 1999). So far, these studies have focused on the extent to which motor-related brain areas were activated while motor execution (ME) or motor imagery (MI) were performed. A single study examined connectivity differences in the functional network using structural equation modeling or SEM (Solodkin et al. 2004), revealing significant differences in the network structure during ME and MI, specifically the connections to the primary motor cortex (M1) which switch signs or disappear during imagery. However, whole-brain data was acquired requiring rather long repetition times (4000ms) which may have limited the effectiveness of connectivity analysis. In this study, we have restricted our investigation to a smaller network in order to increase temporal resolution (TR=300ms) and used dynamic causal modeling or DCM (Friston et al. 2003). In contrast to SEM which estimates different networks for different stimulus conditions, DCM treats changes in the stimuli as perturbations to a single network.

Materials and Methods

Eight healthy subjects participated in this study. Measurements were performed on a 3 Tesla Medspec scanner (Bruker Biospin, Germany) using gradient-recalled EPI. Four axial slices of 5 mm thickness and a 1mm gap were acquired with a matrix of size 64 by 64 voxels, a TE of 40 ms and a TR of 300 ms. Nominal voxel size was 2.96 by 2.96 by 5 mm. Slices were positioned to cover the primary motor cortex (M1) and the supplementary motor area (SMA). The very short TR of 300 ms was chosen to ensure appropriate coverage of the haemodynamic response. In each of the twelve trials the subjects listened via headphones to a voice that counted down from "10" to "0" and back to "5". Subjects were asked to closely attend to the countdown and perform a brief finger movement as soon as they heard "0". The movement consisted of pressing buttons on a small panel, first with the index finger, then the middle finger, and finally the index finger again. Subjects were instructed to perform these movements as rapidly as possible. Following each trial there was a pause of 18 s which allowed the haemodynamic response to return to baseline. Accordingly, each trial lasted 33 s which resulted in a total of 1320 images for each subjects. In a second run subjects were instructed to imagine the finger movement instead of executing it.

Two ROIs, the SMA and the M1, were defined and BOLD signal time courses were extracted for each subject. After high-pass filtering leaving only components with frequencies higher or equal to the trial frequency, each time course was normalized to a variance of one. As a last step, the preprocessed BOLD signals were averaged across subjects and the resulting two time courses renormalized to a variance of one. DCM was used to assess the functional dependences between M1 and the SMA. In DCM the neuronal activity of any given region can be influenced by the activity of other regions. Moreover, external stimuli can influence the activity of a region as well as the strength of connections between regions. Formally this is achieved using a bilinear approximation to the nonlinear dynamics of the neuronal network. Furthermore, a nonlinear biophysical model transforms the activity of each region to simulate the dynamics of the BOLD response. Four inputs to the model were defined. First, a 10 s long rectangular signal representing the countdown (CNT). The execution of the task (imagery and execution) was expressed using a pulse of duration 1.5 s (TASK) starting at the end of the countdown. In parallel there were one input for signaling motor imagery (IMAG), and one for motor execution (EXEC). Both signals were of the same shape as the TASK input. For the DCM approach, the model structure has to be defined a-priori. To still allow some freedom in the modeling process we evaluated 14 different model structures, where half of the models used IMAG to mark the stimulus condition and the other half were structurally equivalent but used EXEC instead of IMAG in order to determine whether the difference in the M1 response would be caused by suppression during imagery or enhancement during execution. After parameter estimation the performance of the different models was compared using Bayes factors based on the Bayes Information Criterion (BIC) which also considers model complexity in model fitness assessment (Penny et al. 2004).

Results

Fig. 1 shows the BOLD responses averaged across subjects and trials. During the actual execution of the finger movement task SMA and M1 were both activated whereas, in the case of motor imagery, only subtle activation was observed in M1.

As expected, the parameter estimates show either a suppressive influence of the IMAG input, i. e. the weighting factor is negative, or an elevating influence of the EXEC input for all models. Overall, all models produced comparable results with respect to the mean squared error. BIC reveals an advantage of using the IMAG input as compared to the EXEC input. Furthermore, in the best model the subtle activation of M1 in motor imagery is caused by a suppressive (i.e. negative) modulation of the connection from SMA to M1 (Fig. 2A). This model also has IMAG as a direct input to SMA to account for the slight decrease in signal during MI. The purely direct model (Fig. 2B) performs significantly worse with a Bayesian factor of 1.6E7 in favor of the indirect model in Fig. 2A.

Discussion

Quantitative results suggest that models with a suppressive influence on the M1 during motor imagery better comply with the measured fMRI data than models with an enhancing influence during motor execution. Furthermore, modulation of the influence of SMA on M1 during MI produces significantly better results than direct suppressive input to M1. In Solodkin et al. (2004) the connection from SMA to M1 becomes negative in the case of kinetic imagery. Our observations support this result since the latent positive connectivity from SMA to M1 (0.1 ± 0.01 s^{-1}) is pulled down through IMAG by -1.06 ± 0.08 s^{-1} (Fig. 2A) so that SMA acts suppressive on M1 during motor imagery. In conclusion, our results clearly demonstrate the potential of DCM analysis to extract additional information on interregional connections, enabling an in vivo verification of connectivity models.

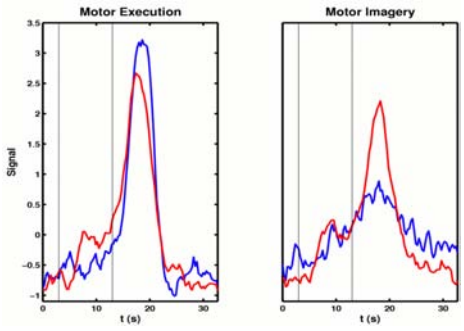

Fig. 1: Activation: The left panel shows the activation of M1 (blue) and SMA (red) during motor execution. The right panel shows the same during motor imagery. Note the substantial reduction in M1 activity during motor imagery. The vertical lines mark the countdown.

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References

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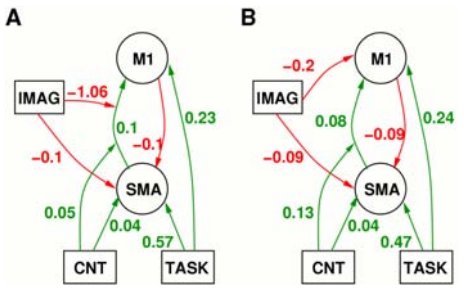

Fig. 2: Model Examples: A shows the best performing model. In B the stimulus condition acts only directly on the network.