# Schizophrenia-specific deficit in focal motor activation during the movement stages related to the conscious perception of the intention to move 

Stephan Bender, MD ${ }^{1,2,5}$, Johannes Schröder, MD ${ }^{3}$, Franz Resch, MD ${ }^{1}$, Matthias Weisbrod, MD ${ }^{2,4}$<br>1 Department for Child and Adolescent Psychiatry Psychiatric Hospital, University of Heidelberg, Blumenstraße 8, D-69115 Heidelberg, Germany 2 Section for Experimental Psychopathology, Psychiatric Hospital, University of Heidelberg, Voßstraße 4, D-69115 Heidelberg, Germany 3 Section for Gerontopsychiatry, Psychiatric Hospital, University of Heidelberg, Voßstraße 4, D-69115 Heidelberg, Germany<br>4 SRH-Klinikum Karlsbad-Langensteinbach, Psychiatric Hospital, Guttmannstraße 1, D-76307 Karlsbad, Germany 5 Psychosomatic Hospital, University of Heidelberg, Im Neuenheimer Feld, D-69120 Heidelberg, Germany

## Background

The neuronal mechanisms behind schizophrenia-specific first-rank symptoms are still not fully understood. Focal motor system activation during advanced movement programming and execution is supposed to be crucial for the subjective intention to move and the feeling-of-agency, when the motor 'efference' copy is compared to 'reafferent' sensory feedback. Persisting deficits in these movement stages could facilitate the episodic development of schizophrenia-specific first-rank symptoms related to a disturbed "acting self". Lateralised premotor/motor cortex activation preceding the movement could be related with the motor efference copy and the conscious intention to move (Haggard and Eimer 1999). A persisting reduced sensory cortex inhibition by frontal action-initiating areas might result in episodic sensory over-activation (Avikainen et al, 2002). MRP (movement related potential) dipole analysis (Toma et al, 2002) provides the temporal and spatial resolution to separate these different movement stages.

## Methods

Multi-channel lateralized movement-related potentials (LMRPs) were analysed during choice reaction movements in 16 schizophrenic/schizoaffective patients (partial remission with predominant negative symptoms), 18 patients with a non-psychotic major depression (clinical control group) and two healthy control groups age-matched to the respective patient groups (20/23 subjects). We compared cortical motor activation to the activation by reafferent sensory feedback by analyzing -

1) Initial motor potential peak (iMP') lateralization related to conscious intention to move, which results from pre- and primary motor corex activation (at C3/C4; C5'/C6'; CP5 ${ }^{\prime} / \mathrm{CP} 6^{\prime}$, during 120 ms preceding the button press).
2) Lateralized motor postimperative negative variation (mPINV') which reflects postprocessing within the motor system when a movement has already stopped (during 5001000 ms after the button press, same electrodes)

Source analysis: Spatio-temporal equivalent dipole source analysis was conducted using BESA to compare iMP' and lateralised frontal motor potential peak (fpMP') which results from reafferent proprioceptive and somatosensory input from the ongoing movement. Two dipoles fitted on ascending peaks of two separate principal components, were used to explain motor cortex activation (during iMP') and activation by reafferent proprioceptive and somatosensory feedback (during fpMp').

## Results

iMP' was specifically reduced in schizophrenic/schizoaffective patients who showed a significantly less lateralized iMP than their age-matched healthy controls ( $\mathrm{p}=0.005$ ), depressed patients ( $\mathrm{p}=0.0003$ ) or mid-age controls $(\mathrm{p}=0.001$ ). For mPINV' no significant difference between schizophrenic/schizoaffective patients and their age-matched healthy controls ( $\mathrm{p}=0.45$ ) or depressed patients ( $\mathrm{p}=0.36$ ) was found.
Schizophrenia affected the dipole moments of motor cortex activation during the motor preparatory and somatosensory feedback stages in a different way. Activation related to reafferent feedback was nearly unaffected ( $\mathrm{p}=0.71$ ), while motor cortex activation was clearly reduced in schizophrenic subjects ( $\mathrm{p}=0.027$ ). A single dipole was fitted on the peak of the difference wave between healthy subjects and schizophrenic patients, indicated the radial and tangential activation of the precentral gyrus.

## References

Haggard, P., \& Eimer, M. (1999). On the relation between brain potentials and the awareness of voluntary movements. Experimental Brain Research, 126, 128-133.
Avikainen, Forss, N., \& Hari, R. (2002). Modulated activation of the human SI and SII cortices during observation of hand actions. Neurolmage, 15, 640-646.
Toma, K., Matsuoka, T., Immisch, I., Mima, T., Waldvogel, D., \& Koshy, B. (2002). Generators of movement-related cortical potentials: fMRI-constrained EEG dipole source analysis. Neuroimage, 17, 161-173.

## Results

Dipole moment of equivalent dipole 1 (motor system)


Dipole moment of equivalent dipole 2 (proprioceptive/somatosensory reafference)


Dipole source analysis in order to separate activation related to motor output and reafferent sensory input: There were significant differences in primary motor cortex activation between schizophrenic/schizoaffective patients and healthy controls right before the button press (dashed vertical line) while activation due to reafferent sensory input was almost unaffected.


## Discussion

Depressive patients could be distinguished on a neurophysiological level from schizophrenic/schizoaffective patients with negative symptoms. Reduced focused actionrelated motor system activation was associated with reduced parietal pre-movement activation but preserved activation to reafferent sensory feedback, creating an imbalance between 'efference copy' and sensory input specifically in schizophrenic/schizoaffective patients. Our subjects showed no delusions of alien control during the recordings and thus no hyperactive sensory cortex. Instead we found evidence for a persisting imbalance caused by deficient activation of frontal systems. Motor activation deficits could thus represent a vulnerability trait in patients with persisting negative and episodic positive symptoms. Such a trait (a 'hypofrontality' extending to the motor system) could link negative symptoms and executive control deficits with the development of psychotic symptoms (easily disturbed feeling-of-agency).

