

# New aspects in action planning and execution in musicians with dystonia

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Recent neurophysiological studies have associated focal-task specific dystonia (FTSD) with impaired inhibitory function. However, it remains unknown whether FTSD also affects the inhibition (INH) of long-term overlearned motor programs. Consequently, the question whether or not musician's dystonia (MD) affects the inhibition (INH) of long-term overlearned motor programs was addressed. By means of electroencephalography (EEG), the neural correlates associated with INH of long-term overlearned motor memory traces were investigated in MD and healthy pianists in a Go/NoGo paradigm. The findings support the hypothesis of a deficient phase coupling between the neuronal assemblies required to inhibit motor memory traces in patients with MD. Furthermore, in NoGo trials, the movement related cortical potentials showed a positive shift after the NoGo signal related to inhibition and was significantly smaller over sensorimotor areas in musicians with MD. Lastly, EMG recorded from the right flexor pollicis longus muscle confirmed that patients with MD had a disrupted INH in NoGo trials.

*Keywords:* music; performance; dystonia; inhibition; EEG

Musician's dystonia (MD), a form of focal task-specific dystonia (FTSD), is characterized by a degradation of these motor memory traces. MD is a movement disorder, which occurs while a musician is playing the instrument, and is marked by the painless loss of voluntary motor control of extensively trained movements (Altenmüller 2003). Recent neurophysiological studies have provided evidence for the hypothesis that musician's dystonia is associated with impaired inhibitory function and abnormal movement preparation

(for a review, see Lim *et al.* 2001). The latter has been reported, for instance, in data of the Bereitschaftspotential (BP) (Deuschl *et al.* 1995) and the contingent negative variation (CNV) (Lim *et al.* 2001). Furthermore, in a recent study (Hummel *et al.* 2002), deficient inhibition of simple motor patterns was demonstrated in six patients with PTSD using transcranial magnetic stimulation (TMS) and EEG-alpha oscillatory activity. However, it remains unknown whether PTSD also affects the inhibition (INH) of long-term over-learned motor programs. Consequently, the aim of the present study was to investigate, using a Go/NoGo paradigm, the neural correlates associated with the activation (ACT) and inhibition (INH) of motor memory traces in pianists with MD during a pianistic motor task under constraint timing conditions with multichannel EEG. Our main hypothesis was that the functional coupling during INH is impaired for pianists with MD compared with healthy pianists.

## METHOD

### Participants

Nine healthy pianists (8 males, age range=26-47 years, mean=36.5 years) and nine pianists with MD (8 males, age range=27-50 years, mean=35.3 years) participated in this study. In all patients, the right hand was affected. All participants were professional pianists (with accumulated practice time over 10,000 hours). Eight of the nine participants in each group were right-handed, according to the Edinburgh Inventory (Oldfield 1971).

### Procedure

Participants were seated at a digital piano (Wersi Digital Piano CT2). The keyboard and the right hand of the participant were covered with a board to prevent participants from visually tracking hand and finger movements. In a modified Go/NoGo study, the task was to play upward C-major scales over two octaves. Scales were played as sixteenth notes, and the tempo was standardized at 80 beats/min for a quarter note (one key stroke every 187.5 ms) and paced by metronome-like auditory cues. Scales were played using the conventional C-major fingering: 1,2,3,1,2,3,4,1,2,3,1,2,3,4,5 (the fingers 1-5 refer to thumb, index, middle, ring, and little finger, respectively). The specifications of the Go/NoGo study were as follows: a first visual cue (S1) indicated that participants should be prepared to start playing. The metronome was started 2,750 ms after S1. Participants were instructed to play the first note of any scale coinciding with the third metronome beat. A second visual

cue (S2) was presented 250 ms before the third metronome beat, indicating that the participant should either execute (Go, green ellipse) or not execute (NoGo, red ellipse) the motor sequence. Continuous EEG was recorded from 22 electrodes placed over the scalp according to the extended 10-20 system referenced to linked mastoids. Data were sampled at 500 Hz; the upper cutoff was 100 Hz, and the time constant was set to DC (DC amplifiers and software by NeuroScan, Herndon, Virginia, USA). One bipolar EMG channel was recorded from surface electrodes positioned over the right flexor pollicis longus muscle, located 6 cm apart. The bandpass filters for EMG were set to 5 Hz (highpass) and 100 Hz (lowpass).

We performed the following analyses of the EEG signals: (1) standard time averaging technique to analyze the slow shift of movement-related cortical potentials (MRCPs), (2) wavelet based time-frequency representations (TFR) to analyze (3) the spatiotemporal dynamics of the inter-electrode phase coupling. All statistical tests were performed by means of nonparametric univariate permutation tests and multivariate synchronized permutations (Good 2005).

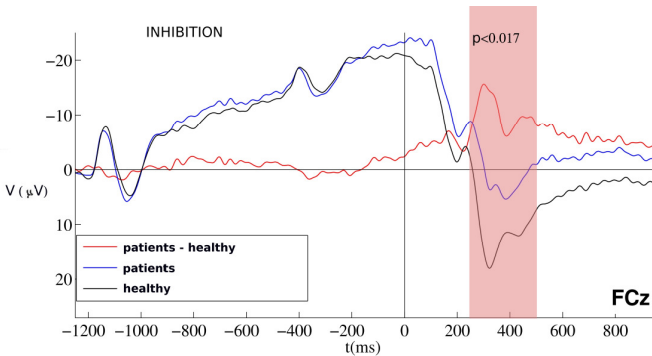
## RESULTS

### EMG analysis

The amplitude mean value in the analysis interval and the EMG peak were selected as EMG activity parameters. In NoGo trials, the selected time window was 0-350 ms to detect whether pianists initiated a movement around 250 ms in spite of the NoGo signal. Burst of EMG activity showed that participants initiated movements of the thumb in some NoGo. Interestingly, however, the first key of the MIDI piano was not actually pressed. This result confirmed that inhibition in our paradigm demanded active suppression of the motor program. The EMG peak was found significantly higher in pianists with MD (mean=14  $\mu$ V, range=5.2-50  $\mu$ V) than in healthy ones (mean=5.5  $\mu$ V, range=3.1-16  $\mu$ V).

### Movement-related cortical potentials

Both groups showed similar pre-movement activity over the sensorimotor cortex, characterized by the slow negative MRCP termed as CNV (Figure 1). In our paradigm, the CNV reflected the maintenance of a motor response in readiness (Haider *et al.* 1981). In NoGo trials, the pre-movement negativity returned to baseline levels and later a positive shift post-S2 was observed. The positive peak after S2, which could be related to the inhibition of the



*Figure 1.* Movement-related cortical potentials (MRCPs) analysis. Grand average of the MRCPs at electrode FCz for NoGo trials in pianists with MD (blue line), healthy pianists (black line), and difference (MD minus healthy, red line). The temporal interval of statistical significance ( $p < 0.0017$ , permutation test) in the between-groups difference is indicated by the pink region. (See full color version at [www.performancescience.org](http://www.performancescience.org).)

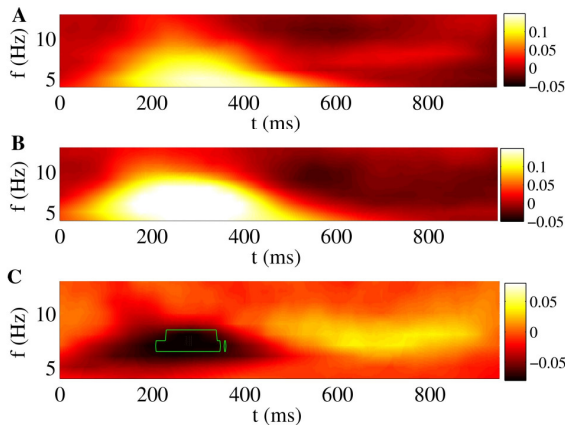
motor pattern, had a significantly larger amplitude across sensorimotor areas in healthy pianists than in pianists with MD ( $p = 0.0016$ ).

### Inter-electrode cortico-cortical phase synchronization

NoGo trials were associated with a robust increase in the degree of global synchronization across the sensorimotor cortex in the theta (4-7 Hz) and lower alpha band (7-8 Hz) with a time span of 200-400 ms, hence coinciding with the latency when the participants were required to begin playing (Figure 2). This increase was significantly more enhanced between 230-330 ms for healthy pianists than for pianists with MD ( $p = 0.004$ , permutation test, univariate test). This effect was due to a weaker phase synchronization in pianists with MD between electrode Cz, representing the supplementary motor areas (SMA), and left premotor and sensorimotor electrodes (FC3, C3, CP3). This result confirmed our main hypothesis, that the functional coupling during INH is impaired for pianists with MD compared with healthy pianists.

## DISCUSSION

Our study focused on the execution and inhibition of long-term overlearned motor programs, due to its relevance in real playing conditions. Our assumption was that in the non-retrieval condition, motor memory traces, strongly activated after the first metronome beat, needed to be suppressed after S2



*Figure 2.* Phase synchronization analysis during INH. The time-frequency plots of the inter-electrode phase synchronization index, averaged across electrodes over sensorimotor and prefrontal areas, are presented for pianists with MD (A), for healthy pianists (B), and for the between-group difference (C, A-B). A pointwise paired permutation test between groups yielded significant differences ( $p=0.004$ ) between 230 and 330 ms and at 7-8 Hz, due to higher global synchronization in healthy pianists than in pianists with MD in this time-frequency window. This region is indicated by the green contour. (See full color version at [www.performancescience.org](http://www.performancescience.org).)

(Hummel *et al.* 2002). Here, we aimed at studying a task that is close to naturalistic piano performance. Accordingly, we imposed high temporal constraints on the task, we used a large sample of patients suffering from MD, and we had healthy musicians as controls.

In this setting, (1) the role of the inter-electrode functional coupling in the sensorimotor integration of inhibitory processes turned out to be the most relevant physiological marker. Our study further showed that in pianists with MD, (2) the non-retrieval of the motor program was associated with a weaker positive shift after-S2 over cortical sensorimotor areas. Finally, (3) the EMG peak in NoGo trials was found to be significantly higher in pianists with MD than in healthy pianists. Our findings, thus, offer evidence that patients with MD, as compared with healthy pianists, have a significantly higher innervation input of the flexor pollicis longus during NoGo trials. This outcome supports the main hypothesis of deficient inhibition in pianists with MD.

The SMA is thought to play an important role in the functional control of movement in that it has direct projections to the primary motor cortex and the spinal chord (Matsuzaka *et al.* 1992). Recent data has proven the suppres-

sive influence of SMA on the primary motor cortex (M1) in motor imagery, thus reflecting the inhibitory function of the forward connection between the SMA and M1 (Kasess *et al.* 2008). Hence, our results could be interpreted as a deficient higher order motor functioning in pianists with MD: the phase coupling between the SMA and the left premotor and sensorimotor cortex, which is required for the non-retrieval of the motor program, is weaker in pianists with MD. Consequently, these data can be regarded as an electrophysiological correlate of the impaired inhibition in pianists with MD.

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