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Motor cortical activities represented by Bereitschaftspotential and paired associative stimulation-induced plasticity in Parkinson's disease and essential tremor

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Objective: To compare the Bereitschaftspotential (BP) and the paired associative stimulation (PAS)-induced plasticity between healthy subjects, Parkinson's disease (PD) and essential tremor (ET).

Background: PD and ET are common movement disorders. Previous evidences showed abnormalities of the BP in PD and ET. The motor cortex excitability modulated by the PAS, a tool for inducing long-term plasticity, was reported abnormal in the patients with PD.

Methods: Ten PD patients (67.5±9.0 years), ten ET patients (63.4±8.7 years) and ten healthy control subjects (68.7±7.1years) were studied. They were requested to move their hand by self-pacing during the recording of BP. PAS consisted of 225 pairs of electrical stimulation of the right median nerve followed by a single transcranial magnetic stimulation (TMS) pulse over the left primary motor cortex (M1). The interval between the electrical stimulation and the TMS keeps 2 ms longer than the individual N20 latency, which is supposed to induce a long-term potentiation at M1. Cortical excitability was measured by twenty single trials of the motor-evoked potential (MEP) recorded from the right APB muscle before and after the PAS intervention. Short-interval and long-interval intracortical inhibition (SICI and LICI) were also evaluated by the paired stimuli with an interstimulus interval of 2 ms and 100 ms, respectively.

Results: BP was measured in early component (1500 to 500 ms before the movement onset), late component (500 ms before to the movement onset) and the frontal peak of motor potential (fpMP). The PD group showed a significant reduction of the early and the late BP compared to the ET group and the control group. The MEP was significantly facilitated by the PAS intervention in the control group but not in the other two groups. In addition, the degree of the MEP change was significantly correlated to the fpMP latency in the control group. SICI and LICI revealed a non-specific reduction after the PAS intervention.

Conclusions: The PD patients and the ET patients demonstrated distinct BP manifestations and impaired PAS effect, suggesting a disease-specific pathophysiology in the motor related cortices.

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Reduced intracortical inhibition in focal hand dystonia: A study of high frequency oscillations

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Objective: To assess if focal hand dystonia (FHD) patients have significant changes in high-frequency oscillations (HFOs) when compared to normal volunteers.

Background: Data from TMS experiments indicate that focal hand dystonia (FHD) patients may have reduced intracortical inhibition. FHD is a sensory-motor disorder. The main cell populations responsible for intracortical inhibition are GABAergic inhibitory interneurons, activity of which is thought to generate HFOs observed in the somatosensory evoked potentials (SSEPs). We hypothesized that median nerve SSEP HFOs would be reduced in FHD patients, indicative of reduced intracortical inhibition. We propose that median nerve distribution is ideal to study in FHD, given that studied body part is affected by dystonia.

Methods: We recorded median nerve somatosensory-evoked potentials (SSEPs) from both sides in eight focal hand dystonia patients and seven normal volunteers. All subjects were right-handed and only the right side was affected. No patients were receiving anticholinergic medications and were at least 3 months from their last chemodenervation. Bar stimulators at the wrists delivered 0.2 ms square wave pulses at 1.2 times motor threshold at different inter-stimulus intervals. EEG data were recorded on both hemispheres with 12 surface electrodes. APB EMG was used to monitor stimulation.

Results: Focal hand dystonia patients (FHD) had similar but longer N20 latencies and smaller average N20 amplitudes, and FHD patients showed similar but slightly smaller average number of