Subthalamic neuronal activity in patients with obsessive-compulsive disorders or Parkinson’s disease

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Marie-Laure Welter, Pierre Burbaud, Anne-Hélène Clair, Amy Bastian, Eric Bardinet, Sara Fernandez-Vidal, Paul Sauleau, Marion Simonetta-Moreau, Stéphane Besnard, Bernard Pidoux, Jérôme Coste, Jérôme Yelnik, Luc Mallet, STOC Study Group

Paris, France

Objective: Study the subthalamic (STN) neuronal activity in patients with Obsessive Compulsive Disorders (OCD).

Background: Dysfunction in the basal ganglia circuitry has been implicated in obsessive and compulsive disorder (OCD). In a recent clinical research program, high frequency electrical stimulation of the STN has proved to be efficient in alleviating obsessions and compulsions in OCD patients and permitted to study neuronal activity in this disorder (Mallet et al, 2008).

Methods: Unit neuronal activity of STN neurons were recorded in awake OCD patients at rest and compared to data obtained in patients with Parkinson’s disease (PD). The mean firing rate and interspike intervals were calculated for each cell. The firing pattern was classified as regular, irregular or bursting (Kaneoke and Vitek, 1996). Neuronal activity was also sampled for each period and epochs of elevated discharge rate were classified as burst using a Poisson surprise analysis. Spike trains with S>=3 were considered to be bursts. Percentages of action potentials and duration with S>=3 and mean S value were calculated for each cell. The precise localization of neuronal activity recordings was performed using a 3-D deformable basal ganglia atlas with a particular reference to STN subterritories.

Results: 156 STN neurons were isolated in 11 OCD patients and 113 neurons in 10 PD patients. In comparison to PD, the mean discharge frequency of STN neurons was lower in OCD patient (24.1 +/- 14.1 Hz vs 32.1 +/- 17.7 Hz, P<10^-3) with a higher burst type activity (p<0.03). The mean S value was higher in OCD patients (7.0 +/- 3.5 vs 5.9 +/- 1.9, P<10^-2) with a higher mean percentage of action potentials (39.0 +/- 13.5 vs 32.8 +/- 14.4 %, P<10^-3) and duration with S>=3 (17.7 6 4.7 vs 14.9 6 5.6 %, P<10^-4).

Conclusions: In OCD patients, the subthalamic neuronal activity seems abnormal with an increase in the bursting type activity. This is in line with the hypothesis of the role of basal ganglia, and the subthalamic nucleus, in the physiopathology of this disorder.

References: