β Oscillations as the Cause of Both Hyper- and Hypokinetic Symptoms of Movement Disorders

To the Editor: Clinical manifestations of extrapyramidal movement disorders are usually divided into two main categories: hyperkinetic (i.e., tremor, athetosis, chorea, and ballism) and hypokinetic (i.e., akinesia, bradykinesia, and rigidity) symptoms.

Despite the different pathological bases of these symptoms, simultaneous emersion of both kinds of symptoms in a single case (like coincidence of rigidity and tremor in a patient with Parkinson's disease) and changing the general appearance of disease from hypo- or hyperkinesia to another group of symptoms suggests a similar origin for both groups of symptoms. With regard to the antikinetic effect of β band oscillatory activity,[1] all pathological procedures involving the basal ganglia can represent hypokinetic features if they persuade the basal ganglia network to oscillate at β band. Aggravation of hypokinesia caused by stimulation of subthalamic nucleus at 15 Hz through deep brain stimulation electrodes also supports the theory.[2] Simultaneous reduction of β band power and intensity of tremor during voluntary movements,[2] finding a roughly linear relationship between the amount of high frequency (at β band) synchronized activity and the patient's tremor, and the fact that tremor cells are usually localized in the regions that also contain cells with a high frequency oscillatory component provide strong support for the role of β frequency in producing hyperkinetic symptoms.[3] Oscillation at β frequency is persistent and synchronized among large populations of neurons and caused by the alteration in network structure. Conversely, activity at tremor frequency is transient, synchronized just in small neuronal populations, and caused by the intrinsic membrane properties of neurons.[4] Therefore the tremor site will vary according to the site of the small neuronal assembly, which temporarily synchronizes at β frequency.

Analyzing frequencies other than β band generated at some stage in movement disorders will help us improve our knowledge about how basal ganglia oscillatory activity transforms into hyperkinetic movements. We cannot discuss the oscillatory activities causing hyperkinesias in ballism and Huntington's disease because chorea usually disappears with the induction of anesthesia; however, dystonia in L-dopa-induced dyskinesias is a hyperkinetic symptom that appears even under anesthesia and can be considered independent of cortical activity. Replacing the 4 Hz–7 Hz oscillatory activity seen in Parkinson's disease with the 7 Hz–10 Hz seen in L-dopa-induced dyskinesias can change the hyperkinetic manifestation of disease from tremor to dystonia and offers a relationship between output frequencies of basal ganglia and hyperkinetic manifestations of disease. Although the peak at β band disappears in the power spectral density of subthalamic nucleus neurons affected by dopamine in L-dopa-induced dyskinesias, it is still present and can be recorded in the contralateral cortex.[5]

According to these observations we can conclude that neuronal synchronization at β band directly leads to the production of hypokinesia and creates a necessary background for hyperkinesia to emerge. Transient appearance of oscillatory activities in other frequencies can result in hyperkinetic movements only when they coincide with β band activity. Since the topography of motor cortex is preserved in the skeletomotor circuit, different sites of involuntary movements can be explained by different sites of oscillations in the basal ganglia.

Alireza Akbari, M.D.
Shahriar Gharibzadeh, M.D., Ph.D.
Neuromuscular Systems Laboratory, Amirkabir University of Technology, Tehran, Iran

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Decision-Making Impairment in a Patient With New Concomitant Diagnoses of Parkinson's Disease and HIV

To the Editor: A 48-year-old man with 8 years of education came under our care in November 2007

for right upper-limb rigidity. In April 2006, he had undergone single photon emission computed tomography (SPECT) with DatSCAN that revealed a normal uptake at nigrostriatal levels. We repeated a SPECT with DatSCAN and revealed a bilateral loss of uptake particularly in the left putamen and in the left caudate nucleus. Uptake was normal in the right caudate nucleus. [^{18}F]fluorodeoxyglucose (FDG)-positron emission tomography (PET) revealed a normal cerebral metabolism. Clinical symptoms, SPECT findings, and the strong positive response during the L-dopa test led us to a diagnosis of Parkinson's disease. The patient also tested positive for human immunodeficiency virus (HIV+).

Neuropsychological evaluation revealed preserved cognitive functioning (episodic memory, attention, executive functions, abstract reasoning, visuospatial skills, limb praxis, and constructional praxis) with only a low level performance, even if within normal range, for delayed free recall on the Rey Auditory Verbal Learning Task. If there was a normal result in classic tasks for the assessment of executive functions based on the dorsolateral prefrontal cortex (Stroop Interference Test, Frontal Assessment Battery, Wisconsin Card Sorting Test, and Trail Making test), then performance on a task assessing executive functions based on the ventromedial prefrontal cortex showed impairment. During the Iowa Gambling Task,[1] a test for the assessment of decision making under ambiguous/uncertain conditions, the patient preferentially selected cards from risky, "long-term disadvantageous" decks, resulting in a final negative outcome (−955). Final balance between choices from advantageous decks [C+D] and choices from disadvantageous decks [A+B] reported a negative score (−6).

Patients who are HIV+ can present decision-making impairment,[2] correlated with deficits of inhibitory processes (Stroop Interference Test) and episodic memory. Patients with Parkinson's disease may also present decision-making impairment,[3] even if the underlying causal mechanism is not known. Some authors[3] propose that decision-making impairment is related to amygdala dysfunction, while other authors[4] suggest that it is related to impaired reinforcement learning, especially from negative feedbacks, due to the effects of dopamine replacement therapy on the orbitofrontal frontostriatal circuit.

Both movement disorders and cognitive dysfunction may represent the initial manifestation of HIV, probably due to the predilection of HIV infection to affect the basal ganglia and frontal white matter.[5] The cognitive performance of our patient (intact executive functions and impaired decision making) shows that the association of two diseases causing frontostriatal impairment, like Parkinson's disease and HIV, may produce a specific neuropsychological pattern, different from that usually associated with Parkinson's disease or HIV.

Our finding shows that in a patient with new concomitant diagnoses of Parkinson's disease and HIV, decision making can become impaired despite intact cognitive functioning. Our finding also confirms the usefulness of gambling tasks as a tool to detect early cognitive impairment in patients with HIV and patients with Parkinson's disease.

Michele Poletti, Psy.D.
Filippo Baldacci, M.D.
Angelo Nuti, M.D.
Claudio Lucetti, M.D.
Caterina Berti, M.D.
Neurology Unit, Versilia Hospital, Lido di Camaiore (LU), Italy
Ubaldo Bonuccelli, M.D.
Neurology Unit, Versilia Hospital, Lido di Camaiore (LU); Neuroscience Department, University of Pisa, Italy

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Altered Sexual Orientation Following Dominant Hemisphere Infract

To the Editor: Human sexual behavior is a complex subject that has proved challenging to scientists of all disciplines. Psychological theories[1] as well as biological concepts[2,3] have been proposed to address sexual behavior and, in particular, human sexual orientation. The subject is further complicated by moral and ethical views widely prevalent in all societies. We report on a case of altered sexual orientation following an infarct in the left middle cerebral artery region.