



# **Brain Stimulation: Chapter 42. Parkinson's disease (Handbook of Clinical Neurology)**

*John C. Rothwell, Mark J. Edwards*

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There have been a large number of basic research studies of noninvasive brain stimulation in Parkinson's disease. Initial work focused on measuring: (1) the excitability of corticospinal output with threshold and input–output measures, and (2) the effectiveness of intracortical  $\gamma$ -aminobutyric acid (GABA)ergic inhibitory systems using short-interval intracortical inhibition (SICI), long-interval intracortical inhibition (LICI), and silent period measures. Early suggestions of increased excitability and reduced inhibition have been progressively modified. There are conflicting reports on changes in excitability, silent period, and LICI, and the more consistent reduction in SICI is now viewed as a superimposed excitation rather than a primary deficit in a GABAergic mechanism. A small number of studies have suggested that premovement increases in corticospinal excitability may be prolonged in Parkinson's disease, consistent with the suggestion of slower buildup of the motor command to move; there are also modifications of interhemispheric connections in patients with mirror movements. Transcranial magnetic stimulation (TMS) has also been used to explore the involvement of motor cortex and cerebellum in resting and postural tremors by examining how readily they can be reset by single TMS pulses over each area. It can also probe the effects of deep brain stimulation of motor cortex excitability. Finally, new TMS techniques that examine synaptic plasticity in motor cortex have shown reduced excitability in patients off therapy which is restored when on therapy. Data are also emerging about the possible role of cortical plasticity in compensating for gradual loss of dopaminergic function prior to onset of clinical symptoms.

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