The motor cortical plasticity and dopamine: healthy and patients with PD

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In this communication, I will briefly describe a new plasticity induction method (quadripulse stimulation (**QPS**)), and dopamine influence on the motor cortical plasticity induced by QPS in normal subjects and patients with Parkinson's disease (PD).

What's QPS?

In QPS, one train of four monophasic TMS pluses separated by different inter-stimulus intervals (ISIs) was given every 5 seconds for 30 minutes. The short interval QPS induced a long term potentiation (LTP) like effect and the long interval QPS induced a long term depression (LTD) like effect. The physiological changes induced by QPS were all compatible with those seen in the synaptic plasticity induction in animal experiments.

Dopamine/Dopamine agonist/Zonisamide and plasticity

We compared LTP/LTD effect between placebo condition and the condition after L-Dopa intake in normal volunteers. Dopamine enhanced both LTP and LTD. In contrast, dopamine agonists had no significant influence on LTP/LTD like effects. Zonisamide mildly enhanced LTP like effect. These results may be explained by the idea that dopamine influence on the plasticity is D1 dependent because most dopamine agonists have nearly pure D2 dependent effect. We consider that the motor cortical plasticity is affected by D1 pathways directly from the ventral tegmental area to the primary motor cortex because it is D1 dependent.

Neuroplasticity in PD

In PD patients, QPS induced neither LTP nor LTD like effects in the motor cortex. This lack of plasticity was normalized by L-DOPA intake. The improvement of plasticity sometimes was not in parallel with UPDRS motor score dependent on the nigra-striatal pathway function. This may support my above idea of the dopamine effect on the motor cortical plasticity.