

Changes in Synaptic Function and Excitability in Single Neurons Following Transcranial Magnetic Stimulation

Motor Rehabilitation

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Repetitive transcranial magnetic stimulation, (rTMS) can non-invasively alter the activity of neural circuits for a time outlasting the stimulation period. This has been demonstrated in some studies examining changes in peripheral motor-evoked potentials (MEPs) following application of various rTMS protocols. These prolonged changes have been attributed to the induction of synaptic plasticity. At a single neuron level however, the effects of rTMS are not well understood and the induction of synaptic plasticity or prolonged changes in cellular excitability have not been demonstrated.

Using a rat model we investigated the effects of TMS on activity characteristics of single pyramidal neurons. *In vivo* intracellular sharp-electrode electrophysiological recordings were made from single cortical neurons in urethane-anaesthetized Wistar rats, during the application of TMS. Spontaneous neuron activity was recorded in response to single pulse and rTMS. In addition, post-synaptic potentials (PSPs) elicited using electrical or magnetic stimulation of the ipsilateral hemisphere were investigated both pre and post rTMS.

Action potentials and PSPs were reliably obtained following single pulse TMS, delivered at intensities much lower than those used in many clinical settings. During rTMS trains, spontaneous rhythmical neuronal activity was disrupted and in some cases, neuronal firing was induced. Following rTMS, neuronal excitability was altered as indicated by lasting changes in rheobase current and spontaneous activity. Furthermore, both long term potentiation (LTP) and long term depression (LTD) were observed following particular combinations of rTMS protocols.

These results provide the first indication of the effects that both single pulse and repetitive TMS have on cortical neuron excitability and synaptic plasticity. With a better understanding of these effects it is hoped rTMS protocols may be more effectively targeted to specific neural circuits, in order to optimize clinical treatment of neurological disorders.