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TERMINATION OF SPINDLE OSCILLATIONS BY SHORT-TERM SYNAPTIC PLASTICITY OF THALAMIC RETICULAR CONNECTIONS.

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Sleep spindles consist of waxing and waning 7-14 Hz oscillations that appear in the EEG during the early stages of sleep. Spindle oscillations are generated in the thalamus as a consequence of the intrinsic properties of thalamocortical (TC) and thalamic reticular (RE) cells and their synaptic interactions. Once initiated the waxing of spindles is thought to involve a progressive recruitment of TC cells into the oscillation. In vitro studies suggest that the waning and termination of spindles are caused by a progressive depolarization of TC cells through a Ca^{2+}-dependent upregulation of the H-current. In vivo intracellular recordings of TC cells, however, rarely display a progressive depolarization during spindles. Recent studies have provided evidence for short-term synaptic plasticity of RE-TC connections. In particular, repetitive burst discharges lead to decreased inhibitory responses in TC cells *in vitro* (Ulrich & Huguenard, 1996; Kim & McCormick, 1998), and repetitive thalamic stimulation in vivo results in a reduction of IPSPs in TC cells (Grenier et al., this meeting). Here we show that short-term synaptic plasticity can cause the termination of spindle oscillations in computational models of the thalamus without any change of the H-current in TC cells. The progressive reduction in RE-TC synaptic efficacy during the spindle reduces the evoked hyperpolarization in TC cells and consequently their low-threshold rebound responses. Thus, fewer RE cells are recruited into the next cycle and the oscillation fades. Predictions of the model are being tested in vivo. Research sponsored by NSERC and HHMI.