Coop et al. BMC Neuroscience 2010, 11(Suppl 1):P86
http://www.biomedcentral.com/1471-2202/11/S1/P86

BMC Neuroscience

POSTER PRESENTATION Open Access

Single neuron electrophysiology of transcranial magnetic stimulation. I. Passive responses

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From Nineteenth Annual Computational Neuroscience Meeting: CNS*2010
San Antonio, TX, USA. 24-30 July 2010

Transcranial magnetic stimulation (TMS) is a widely used noninvasive stimulation technique that induces neurons to discharge via a rapidly changing magnetic field directed through the scalp. However, the interaction between neural tissue and TMS stimulation is not well understood. Kamitani et al. [1] and Miyawaki et al. [2] have investigated the afterhyperpolarization that follows TMS evoked discharge in a single cortical neuron model, but they described only the discharge response and silent period evoked by a single TMS stimulus without details as to the orientation of the stimulating E-field. We have previously reported that spiking is induced only when the primary electric (E) field is oriented from the soma toward the apical dendrite (antidromic stimulus). In contrast, an E-field oriented in the opposite direction from the apical dendrite toward the soma (orthodromic stimulus) evokes an excitatory potential (EP) considerably larger (3 mV) than that of an excitatory postsynaptic potential (about 0.75 mV at -70 mV resting potential in this system) [4]. The EP is not sufficient to induce cell firing [4]. To better understand this behavior, we explored the passive membrane response of the model system to different stimulus magnitudes.

Methods

We employed the same TMS modeling system as [1]. It is comprised of a multicompartment Layer 5 pyramidal neuron from cat visual cortex [3] and incorporates a modeled TMS stimulus. To determine the source of the orientation selectivity of neuronal response, we removed active membrane channels from the model cell and recorded the passive membrane response along a piecewise continuous axis spanning the length of the neuron.

Results

An orthodromic TMS stimulus evoked a distance-related bimodal depolarization in the basal dendrites (peak: proximal 26 mV, distal 18 mV) that peaked within 0.5 ms of stimulus onset. In the apical dendrites, the stimulus induced a small depolarization (2-3 mV) proximal to the soma. Depolarization converted to hyperpolarization within 250 um of the soma and increased to become larger than 30 mV at the distal dendritic tip (1000 um). The spatial profile of the membrane response was inverted (i.e. depolarization became hyperpolarization and hyperpolarization became depolarization) following an antidromic E-field stimulus.

Conclusion

In response to an orthodromically directed E-field, current driven into the basal dendrites dissipates rapidly into this highly branched structure and thereby prevents action potential generation at the soma. Alternatively, during an antidromic stimulus, the basal dendrites are rapidly hyperpolarized and current driven into the distal apical dendrites rapidly dissipates. However sufficient depolarization is evoked at the soma to initiate cell discharge. This behavior is likely controlled by the primary apical dendrite which exhibits increased resistance for the first 800 um from the soma.

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Published: 20 July 2010

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doi:10.1186/1471-2202-11-S1-P86
Cite this article as: Coop *et al.*: Single neuron electrophysiology of transcranial magnetic stimulation. I. Passive responses. *BMC Neuroscience* 2010 **11**(Suppl 1):P86.