Neuronal excitability at physiological and lower temperatures

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Abstract: Objective Therapeutic hypothermia becomes a promising neuroprotectant which contributes to promoting cell survival and reducing neurological damage due to brain injuries such as cardiac arrest and stroke. Decreasing the brain temperature to 32°C can reduce neuronal metabolism, but the mechanism underlying the effect of low temperature on neuronal excitability is not fully understood. The present study is to explore how temperature changes (range: 0-37°C) affect excitability of neocortical pyramidal cells. Methods The passive membrane properties and action potential initiation and propagation of pyramidal cells were measured with whole-cell recording in acute cortical slices. Two-photon calcium imaging was employed to examine dynamics of calcium transients at different temperatures. Results (1) Reducing temperature results in depolarization of the membrane potential, augment of the input resistance and increase in the spike width. The ability of repetitive fire is increased in response to step currents lower than 200 pA but decreased with larger current injections. (2) During cooling, the conduction velocity of action potentials becomes slower. (3) With lower temperature levels (35-0°C), the rise and decay times of calcium transients induced by action potentials at proximal dendrites are prolonged. Conclusion The reduction in temperature produces changes in passive membrane properties in a direction to promote action potential generation, however the excitability of PFC pyramidal cells is actually reduced in terms of spiking activities, and signal conduction is substantially decreased.

Keywords: lower temperature; action potential; passive membrane property