Abstract View

MEMBRANE BISTABILITY IN CAT THALAMIC RETICULAR NEURONS DURING SPINDLE OSCILLATIONS

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The thalamic reticular (RE) nucleus plays a crucial role in regulating the excitability of thalamocortical networks and in generating some sleep rhythms. Current clamp intracellular recordings of RE neurons in cats under barbiturate anaesthesia revealed the presence of membrane bistability in ~20% of neurons. Bistability consisted of two alternate membrane potentials, separated by ~17-20 mV. While non-bistable (common) RE neurons fired rhythmic spike-bursts during spindles, bistable RE neurons fired tonically, with burst modulation, throughout spindle sequences. Bistability was strongly voltage-dependent and only expressed under resting conditions (i.e. no current injection). The transition from the silent to the active state was a regenerative event that could be activated by brief depolarization, while brief hyperpolarizations could switch the membrane potential from the active to the silent state. These effects outlasted the current pulses. Corticothalamic stimulation could also switch the membrane potential from silent to active states. Addition of QX-314 in the recording micropipette either abolished or disrupted membrane bistability, suggesting INa(p) to be responsible for its generation. Thalamocortical cells presented various patterns of spindling that reflected the membrane bistability in RE neurons. Finally, computer simulations predicted a role for RE neurons' membrane bistability in inducing various patterns of spindling in target thalamocortical cells. We conclude that membrane bistability of RE neurons is an intrinsic property, likely generated by INa(p) and modulated by cortical influences, as well as a factor that determines different patterns of spindle rhythms in thalamocortical neurons

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