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Poster presentation

Modeling of frog second-order vestibular neurons using frequency-domain analysis reveals the cellular contribution for vestibular signal processing

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Computational modeling of the vestibulo-ocular circuitry is essential for an understanding of the sensory-motor transformation that generates spatially and dynamically appropriate compensatory eye movements during self-motion. The generation of the neuronal commands for gaze stabilization in the central nervous system depends on the cellular characteristics of the involved neurons as well as on the functional organization of the neuronal circuits in which these neurons are embedded. Thus, any reasonable model must adequately incorporate both the intrinsic membrane properties of the neuronal elements as well as the properties of the networks. Central vestibular neurons in the brainstem are responsible for the major computational step in the transformation of sensory vestibular signals during body motion into motor commands for extraocular muscles that cause compensatory eye motion. In frog, these second-order vestibular neurons (2°VN) separate into two distinctly different functional subgroups (tonic-phasic neurons) based on differences in intrinsic membrane properties and discharge characteristics. Correlated with these cellular properties, tonic and phasic 2°VN exhibit pronounced differences in the dynamics of the synaptic activation following stimulation of individual labyrinthine nerve branches [1]. A detailed physio-pharmacological analysis indicated that the two types of 2°VN are differentially embedded in inhibitory circuits that reinforce the cellular properties of these neurons, respectively, thus indicating a co-adaptation of intrinsic membrane and emerging network properties in the two neuronal subtypes.

In a recently published model [1], available quantitative physiological data on intrinsic and synaptic properties of identified frog 2°VN were used to create a model that combines network parameters with a preliminary subthreshold conductance-based cellular model. While this model helps elucidating the contributions of different ion channels to the synaptic subthreshold responses, it cannot explain the distinctly different firing behavior of tonic and phasic 2°VN during a synaptic activation by vestibular nerve afferents. Therefore, a spiking multi-compartment Hodgkin-Huxley type model was generated. This model was constructed on the basis of frequency-domain data obtained from sharp electrode recordings of 2°VN in an isolated frog whole brain with a newly developed method that allows compensation of the high-resistant electrodes [2]. Because this new model takes into account the nonlinear interactions between potassium- and sodium-conductances in 2°VN, it allows making precise predictions on the relative contributions of cellular and network properties to the spiking behavior of tonic and phasic 2°VN. The possibility to now distinguish between cellular and network contributions for vestibular signal processing is a necessary requirement to help understanding the neural computations that allow head-motion derived sensory signals to be transformed into adequate extraocular motor commands for gaze stabilization.

References

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