Abstract View

ENHANCEMENT OF NEURONAL RESPONSE DUE TO FAST INHIBITION

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Inhibition, while usually associated with reducing spike robability, can in some cases enhance firing. Postinhibitory rebound (PIR) is the classical example of inhibition-induced excitation: firing after quick release from long-lasting hyperpolarization. However, PIR can also occur following a single fast-decaying inhibitory conductance transient (IPSG) if it is strong enough, i.e. suprathreshold-for-PIR. More generally, a subthreshold-for-PIR inhibition can also induce a spike (postinhibitory facilitation, PIF) if it is paired with a lagging but precisely timed subthreshold EPSG, as we have shown previously in auditory brain stem neurons (in vitro) and in computational models for them. Here we explore with a computational model the occurrence and statistical properties of such IPSG-EPSG pairings along with other mechanisms involving inhibitory synapses that contribute to enhancement of firing rates. We present random independent Poisson trains of EPSGs and IPSGs to a Hodgkin-Huxley membrane model. The model's spike rate is reduced in the presence of inhibition if the IPSG's (alpha-function) time constant, τ_{inh}, is much larger than that of the membrane's intrinsic negative feedback processes, *h* and *n*. But if τ_{inh} is approximately smaller than τ_h and τ_n, about 2 ms in our case, an enhancement of the spike rate can occur. This enhancement is the net result of the preclusion of some would-be spikes by nearly coincident IPSGs and summated EPSGs and three positive factors: near coincidence of weak IPSGs leading to a net suprathreshold-for-PIR stimulus that triggers a spike, PIF that is effected by the pairings of well timed subthreshold-for-PIR IPSGs and subthreshold EPSGs, and a near coincidence of subthreshold EPSGs. We conclude that some neurons may be selectively tuned to fire in response to precisely timed IPSG-EPSG pairings.
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