

1 Introduction

- We predict that I_T should provide the inward current for the generation of low-threshold Ca^{2+} spikes, with rapidly activating and inactivating K^+ current I_A modulating the initial components of these Ca^{2+} spikes. In contrast, the slower kinetics of activation and inactivation of the K^+ current I_{K2} suggest that this current may affect more the later portions of low-threshold Ca^{2+} spikes. The properties of I_h suggest that it is critical to modulation of the voltage-time course of the cell at hyperpolarized membrane potentials and may provide a "pacemaker" potential for rhythmic burst generation [2].
- these currents include I_T , the low-threshold, transient Ca^{2+} current that underlines burst firing; the rapidly inactivating and transient K^+ current, I_A , which may facilitate slow repetitive firing and may interact with I_T during Ca^{2+} -dependent burst firing; a slowly inactivating transient K^+ current, I_{K2} , which may control repetitive firing rate; and a hyperpolarization-activated, mixed cationic conductance with slow kinetics, I_h , which activates on hyperpolarization and which may generate a "pacemaker" potential for the generation of slow oscillations [2].
- The modeled currents were derived wither from *Drosophila* neurons (specify), or (e.g. rat thalamic neurons, etc)...
- We assumed that I_T was composed of a uniform populatio of channels whose inactivation could be completely described by the Boltzmann function.
- Current-Voltage relationship was better reproduced when constant-field equation was used instead of the ohmic one [2].
- T-Type: m^3h format [3]
- Model: slow deinactivation [3]
- T-Type Ca current together with the leakage current suffices to describe the low-threshold spike (LTS)... Outward currents are not required to reproduce the basic shape of the LTS... Each LTS triggerres a burst of fast action potentials that ride on its crest. As such, LTS plays a critical role in linking synaptic input to intrinsic membrane mechanisms of bursting in the relay cell and in supporting the slow membrane oscillations underlying the spondling rhythm [3].
- I_T is de-inactivated by hyperpolarization, thus providing an ionic basis for the so-called post-inhibitory rebound excitation [4].
- This "3 Hz" bursting is primarily due to the interplay between a T-type calcium current I_T , and a non-specific cation "sag" current I_h which has much slower kinetics than I_T and is activated by hyperpolarization [4].
- Physiologists have been interested in the action of calcium on excitable tissues since the days of Ringer (1883). Some of the main facts established (see Brink, 1954) are that increasing the external calcium concentration raises the threshold, increases membrane resistance (Cole, 1949) and accelerates accommodation. Reducing the calcium concentration has the converse effects, and frequently leads to spontaneous oscillations or repetitive activity (e.g. Adrian & Gelfan, 1933; Arvanitaki, 1939). Other observations which may be less well known are that removal of calcium reduces rectification (Loligo nerve, Steinbach,

Spiegelman & Kawata, 1944) and increases the fraction of the sodium carrying system which is in a refractory or inactive condition (Purkinje fibres, Weidmann, 1955). In connexion with the last observation, it is interesting that tissues which do not normally give an anode break response can be made to do so by reducing the concentration of calcium ions in the external medium (Frankenhaeuser, 1957) [1].