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Control of bursting activity by modulation of ionic currents

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Our study is focused on modulation of dynamics of single leech heart interneurons (HNs). We consider two models of HNs representing these neurons under two different pharmacological treatments: (1) blocking of Ca^{2+} currents and inhibitory coupling with the Ca^{2+}-containing saline and partial blocking of K^+ currents; (2) decoupling HNs with bicuculline. In (1), an HN demonstrates slow plateau-like oscillations [1,2]. In (2), an HN demonstrates endogenously bursting activity [3]. We analyze how the interburst interval and burst duration could be controlled by manipulating hyperpolarization-activated current, I_h, and persistent Na^+ current, I_P, namely by variation of their conductances and the half-activation voltages, $V_{1/2}$. For example, burst duration increases greatly from 1.7 s to 8.9 s as $V_{h,1/2}$ increased from -30 mV to 4 mV. The interburst interval grows from 0.6 s to 125 s as the $V_{h,1/2}$ decreases from 4 [mV] to -56 [mV] in accordance with a saddle-node bifurcation. In (2), we similarly show that the variation of $V_{h,1/2}$ could be a target for modulation of the bursting. In both cases, we show co-existence of bursting and silence. Interestingly, the co-existence is sensitive to g_h (and to maximal conductance of fast Ca^{2+} current, g_{CaF} too in (2)) and is not sensitive to the maximal conductances of other currents. In (1), if g_h is increased from 4 nS to 8 nS, the bistability is then observed in an almost five-fold larger range of the leak conductance values, g_{leak}. In (2), if either g_h is changed from 4 nS to 8 nS or g_{CaF} is changed from 5 nS to 0 nS, the bistability is observed in an almost two-fold larger range of g_{leak}. If the bistability is an indication of a dysfunctional dynamics, this observation describes a new, potentially pathological role of over-expression of I_h and I_{CaF}.

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