

T-type Calcium Current in Electrical Activity of Cardiomyocytes Isolated from Rabbit Pulmonary Vein

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摘要

Abstract

INTRODUCTION: Pulmonary veins (PVs) are known to initiate paroxysmal atrial fibrillation. T-type calcium current ($I(\text{Ca-T})$) has a role in normal and abnormal automaticity of cardiomyocytes. The aim of this study was to evaluate whether $I(\text{Ca-T})$ contributes to PV electrical activity. **METHODS AND RESULTS:** By whole-cell clamp techniques in rabbit myocytes, $I(\text{Ca-T})$ was identified in 12 (39%) of 31 PV cardiomyocytes with pacemaker activity, 2 (9%) of 23 PV cardiomyocytes without pacemaker activity, and 2 (15%) of 13 atrial myocytes ($P < 0.05$). Maximum $I(\text{Ca-L})$ and $I(\text{Ca-T})$ densities from PV cardiomyocytes with pacemaker activity were 6.87 ± 2.17 pA/pF and 1.38 ± 0.69 pA/pF, respectively. Nickel (40 μM) decreased the spontaneous activity in 5 (36%) of 14 PV cardiomyocytes (3.1 ± 0.6 Hz vs 2.2 ± 0.5 Hz, $P < 0.05$), reduced the amplitudes of delayed after depolarization from 13 ± 1 mV to 7 ± 1 mV ($n = 4$, $P < 0.05$) and inhibited transient inward currents from 1.2 ± 0.2 pA/pF to 0.7 ± 0.1 pA/pF ($n = 11$, $P < 0.01$). **CONCLUSIONS:** We conclude that $I(\text{Ca-T})$ contributes to PV pacemaker activity and triggered activity, which are of functional importance in PV arrhythmogenesis.