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(Ca-T)) has a role in normal and abnormal automaticity of cardiomyocytes. The aim of this study was to evaluate whether I(Ca-T) contributes to PV electrical activity. METHODS AND RESULTS: By whole-cell clamp techniques in rabbit myocytes, I(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(Ca-L) and I(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 microM) 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(Ca-T) contributes to PV pacemaker activity and triggered activity, which are of functional importance in PV arrhythmogenesis.