Effects of Thyroid Hormone on the Arrhythmogenic Activity of Pulmonary Vein Cardiomyocytes

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摘要

Abstract

OBJECTIVES: This study was conducted to investigate the effects of thyroid hormone on the electrophysiological characteristics of pulmonary vein (PV) cardiomyocytes. BACKGROUND: Hyperthyroidism is an important etiology of paroxysmal atrial fibrillation (AF). Pulmonary veins are known to initiate paroxysmal AF. METHODS: The action potential and ionic currents were investigated in single rabbit PV and atrial cardiomyocytes with (hyperthyroid) and without (control) incubation of L-triiodothyronineusing the whole-cell clamp technique. RESULTS: Compared with the control cardiomyocytes, hyperthyroid PV and atrial cardiomyocytes had shorter action potential duration. Hyperthyroid PV cardiomyocytes had faster beating rates (1.82 +/- 0.13 Hz vs. 1.03 +/- 0.15 Hz, $p < 0.005$) and a higher incidence of delayed after depolarization (beating: 92% vs. 6%, $p < 0.0001$; non-beating: 45% vs. 3%, $p < 0.005$). However, only hyperthyroid PV beating cardiomyocytes had a higher incidence of early after depolarization (46% vs. 0%, $p < 0.0001$). The ionic current experiments showed that hyperthyroid PV beating cardiomyocytes had larger densities of overall slow inward (2.72 +/- 0.21 pA/pF vs. 2.07 +/- 0.19 pA/pF, $p < 0.05$), overall transient outward (1.39 +/- 0.21 pA/pF vs. 0.48 +/- 0.08 pA/pF, $p < 0.001$) and steady state outward currents (0.78 +/- 0.06 pA/pF vs. 0.58 +/- 0.04 pA/pF, $p < 0.05$) on depolarization and larger transient inward (0.021 +/- 0.004 pA/pF vs. 0.005 +/- 0.001 pA/pF, $p < 0.001$) onrepolarization. By contrast, the hyperthyroid PV non-beating cardiomyocytes had larger densities of overall transient outward (1.01 +/- 0.14 pA/pF vs. 0.37 +/- 0.07 pA/pF, $p < 0.001$), steady state outward (0.61 +/- 0.06 pA/pF vs. 0.44 +/- 0.04 pA/pF,$p < 0.05$) and transient inward currents (0.011 +/- 0.002 pA/pF vs. 0.003 +/- 0.001 pA/pF, $p < 0.05$). CONCLUSIONS: Thyroid hormone changes the electrophysiological activity of the PV cardiomyocytes. Increased automaticity and enhanced triggered activity may increase the arrhythmogenic activity of PVs in hyperthyroidism.