

Angiotensin II and angiotensin II receptor blocker modulate the arrhythmogenic activity of pulmonary veins

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摘要

Abstract

1. Angiotensin II receptor blockers (AII RBs) have been shown to prevent atrial fibrillation. The pulmonary veins (PVs) are the most important focus for the generation of atrial fibrillation. The aim of this study was to evaluate whether angiotensin II or AII RB may change the arrhythmogenic activity of the PVs.
2. Conventional microelectrodes and whole-cell patch clamps were used to investigate the action potentials (APs) and ionic currents in isolated rabbit PV tissue and single cardiomyocytes before and after administering angiotensin II or losartan (AII RB).
3. In the tissue preparations, angiotensin II induced delayed after-depolarizations (1, 10, and 100nM) and accelerated the automatic rhythm (10 and 100nM). Angiotensin II (100nM) prolonged the AP duration and increased the contractile force (10 and 100nM). Losartan (1 and 10 μ M) inhibited the automatic rhythm. Losartan (10 μ M) prolonged the AP duration and reduced the contractile force (1 and 10 μ M).
4. Angiotensin II reduced the transient outward potassium current (I_{to}) but increased the L-type calcium, delayed rectifier potassium (IK), transient inward (I_{ti}), pacemaker, and Na⁺-Ca²⁺ exchanger (NCX) currents in the PV cardiomyocytes. Losartan decreased the I_{to} , IK, I_{ti} , and NCX currents.
5. In conclusion, angiotensin II and AII RB modulate the PV electrical activity, which may play a role in the pathophysiology of atrial fibrillation.