论著

心房颤动患者内向整流性钾电流及Kir2.1 mRNA表达水平的研究

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目的:研究心房颤动(房颤,AF)患者心房肌细胞内向整流性钾电流(Ik1)密度及Kir2.1(编码Ik1)mRNA 表达水平,初步探讨慢性AF患者心房肌电生理重构机制。方法: 胶原酶Ⅱ两步酶解法分离心房肌细胞,膜片钳 全细胞记录法记录离子电流;半定量逆转录聚合酶链反应方法检测心房组织Kir2.1 mRNA表达水平。结果: (1) AF患者心房肌细胞Ik1在超极化状态显著高于窦性心律(SR)组,在膜电位-120 mV时AF组Ik1增加34.04%(P<<mark>▶加入引用管理器</mark> 0.05), 在-30 mV-+10 mV时其外向电流成分显著增加; (2)以GAPDH为内参标基因, AF组和SR组心房肌 Kir2.1 mRNA相对表达量无显著差异(P<0.05)。结论: AF患者右心房肌细胞Ik1密度在超极化状态显著增加 是其电生理重构的重要离子基础之一;AF患者心房肌Kir2.1 mRNA表达水平无显著改变,推测Ik1重构为转录后 调节。

关键词 心房颤动; 钾通道; 膜片钳术; 逆转录聚合酶链反应

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Inward rectifier potassium current and mRNA expression of gene Kir2.1 in human atrial fibrillation

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Abstract

AIM: To investigate alteration of inward rectifier potassium current (IK1) in atrial myocytes and mRNA expression of gene Kir2.1 encoding IK1 in atrial myocardial tissue in patients with chronic atrial fibrillation (AF) compared to that with sinus rhythm (SR).METHODS: Single myocytes were isolated by enzymatic dissociation with the chunk method and the ionic current was recorded using whole cell patch clamp technique. The semi-quantitative reverse transcriptase-polymerase chain reaction (RT-PCR) was used to measure the mRNA expression of Kir2.1 in atrial myocardial tissue, and the gene GAPDH was used as an internal control.RESULTS: (1) The IK1 density was increased in AF group at hyperpolarizing potentials, at -120 mV the current densities was (-5.71±0.65) pA/pF in AF group (n=28 cells from 7 patients) and (-4.26±1.22) pA/pF in SR group (n=35 cells from 9 patients) (P<0.05). AF group displayed increased outward currents at test potentials between -30 mV and +10 mV. (2) The relative quantity of mRNA expression of Kir2.1 in AF and SR group was 0.94±0.10 and 0.90±0.16, respectively (P>0.05).CONCLUSIONS: The increase in IK1 at hyperpolarizing potentials may be related to the atrial electrophysiological remodeling in chronic human AF. The increased IK1 density in atrial myocytes in AF group without alteration of Kir2.1 mRNA expression in atrial tissue suggests that IK1 may be mediated at posttranscriptional levels.

Key words Atrial fibrillation Potassium channels Patch-clamp techniques Reverse transcriptase polymerase chain reaction

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