The Property of Ionic Channels in Sinoatrial Node - The Possible Mechanism of Sick Sinus Syndrome

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Background: Abnormal ionic channels in sinoatrial node (SAN) is associated with sick sinus syndrome (SSS). Voltage clock and Ca2+ clock are the important mechanisms for the SAN automaticity. The purpose of this study was to investigate the impact of different ionic channel blockades on SAN. Method: SAN tissues were isolated from 6 rabbits. The electrophysiological characteristics of SAN were recorded by conventional microelectrodes. Ca2+ clock of SAN was investigated by administration of blockade of sarcoplasmic reticulum Ca2+ release (Ryanodine 2 µM) and CaMKII inhibitor (KN-93 3 µM). Voltage Clock of SAN was studied by administration of If current inhibitor (Ivabradine 0.1, 1, 3, 10 µM). Result: Ryanodine (2 µM) and KN-93 (3 µM) decrease rate of SAN for 36% and 16%, respectively. Maximum diastolic potential and action potential amplitude did not change after administration of these drugs. Rate of SAN increased at low dose of Ryanodine (0.2 µM) and decreased at high dose of Ryanodine (2 µM). However, Ivabradine (10 µM) decreased rate of SAN for 34%, but it did not affect the MDP and APA. Conclusion: SAN dysfunction may be related to the dysregulation of Ca2+ handling and If current. Keywords: sinoatrial node, sick sinus syndrome