Effects of class I antiarrhythmics on the guinea pig pulmonary vein myocardium

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Pulmonary veins contain a myocardial layer, whose electrical activity is considered to be involved in the genesis of atrial fibrillation. Our previous study revealed that persistent sodium current (late I_{Na}) contributes to the automaticity of the pulmonary vein myocardium. Class I antiarrhythmic drug are used for the treatment of atrial fibrillation, but effects on the automatic activity and the late I_{Na} of the pulmonary vein myocardium has not been examined. In this study, we investigated the effect of class I antiarrhythmics on the automatic activity of the isolated guinea pig pulmonary vein myocardium with microelectrode and voltage clamp experiments. All of the antiarrhythmics examined reduced the maximum rate of rise of tertiapin-induced automatic action potentials. The firing frequency and diastolic depolarization slope were decreased by aprindine, flecainide and propafenone, unaffected by pilsicainide, and increased by cibenzoline and disopyramide. The late I_{Na} induced by ramp depolarization was reduced by aprindine, flecainide and propafenone, and unaffected by pilsicainide, cibenzoline and disopyramide. These results indicate that class I antiarrhythmics have differential effects on automaticity of the pulmonary vein myocardium, and that blockade of the late I_{Na} results in inhibition of automaticity.