GS01-3 **Elucidation of the mechanisms of pulmonary vein myocardium automaticity for pharmacotherapy against atrial fibrillation**

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The pulmonary vein contains a myocardial layer connected to the left atrial myocardium which is capable of generating spontaneous or triggered action potentials, which initiates atrial fibrillation. However, the mechanisms underlying the automaticity of the pulmonary vein myocardium are unclear. Therefore, it is not clarified the effect of anti-arrhythmic agents on the automaticity of the pulmonary vein myocardium, and atrial fibrillation pharmacotherapy by suppression of pulmonary vein myocardium has not been realized. In this study, we studied the mechanisms of the automaticity of pulmonary vein myocardium and evaluated the effect of the conventional and novel anti-arrhythmic agents by using isolated guinea-pig pulmonary vein myocardium in vitro. Our study revealed that noradrenaline induced the automatic activity of pulmonary vein myocardium by stimulating of both α_1 and β_1 receptors. We also clarified that late I_{Na}, a persistent component of sodium current, was contributed to the diastolic depolarization of the action potential on pulmonary vein myocardium. In this symposium, we will discuss on the usefulness of conventional anti-arrhythmic agents for inhibition of the automaticity of pulmonary vein myocardium, and the possibility of the selective inhibition by late I_{Na} inhibitor.