Prevention of Ventricular Arrhythmias With Sarcoplasmic Reticulum Ca2+ ATPase Pump Overexpression in a Porcine Model of Ischemia Reperfusion

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Résumé en anglais	Background— Ventricular arrhythmias are life-threatening complications of heart failure and myocardial ischemia. Increased diastolic Ca2+ overload occurring in ischemia leads to afterdepolarizations and aftercontractions that are responsible for cellular electric instability. We inquired whether sarcoplasmic reticulum Ca2+ ATPase pump (SERCA2a) overexpression could reduce ischemic ventricular arrhythmias by modulating Ca2+ overload.Methods and Results— SERCA2a overexpression in pig hearts was achieved by intracoronary gene delivery of adenovirus in the 3 main coronary arteries. Homogeneous distribution of the gene was obtained through the left ventricle. After gene delivery, the left anterior descending coronary artery was occluded for 30 minutes to induce myocardial ischemia followed by reperfusion. We compared this model with a model of permanent coronary artery occlusion. Twenty-four–hour ECG Holter recordings showed that SERCA2a overexpression significantly reduced the number of episodes of ventricular tachycardia after reperfusion, whereas no significant difference was found in the occurrence of sustained or nonsustained ventricular tachycardia and ventricular fibrillation in pigs undergoing permanent occlusion. Conclusions— We show that Ca2+ cycling modulation using SERCA2a overexpression reduces ventricular arrhythmias after ischemia-reperfusion. Strategies that modulate postischemic Ca2+ overload may have clinical promise for the treatment of ventricular arrhythmias.

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