The mechanisms of arrhythmia in heart failure (HF) is attributed in part to the reduced repolarization reserve resulted from an upregulation of late Na⁺ current and the downregulation of multiple major K currents (I₆, I₁₂, I₂O and I₃₁). In addition, h₉₆ is defective in HF, further reducing the ability of failing cells to shorten action potential duration (APD) during metabolic stress. While studying rabbit hearts with HF, Dr Masahiro Ogawa made an unexpected observation that there is acute but reversible APD shortening after fibrillation-defibrillation episodes in failing (but not normal) rabbit ventricles. In addition, there are multiple spontaneous ventricular fibrillation (SVF) episodes after defibrillation shocks in rabbit hearts with most severe APD shortening. Recurrent SVF in HF might be due to increased diastolic Ca²⁺ - membrane potential coupling gain due to downregulation of h₁₁, as reported by Dr Mitsunori Maruyama. However, the acute APD shortening may also play a role in SVF by promoting late phase 3 early afterdepolarization (EAD) and triggered activity, leading to SVF. Chua et al subsequently documented that upregulation of the apamin-sensitive potassium current (I₉₃), a current conducted through the small conductance Ca²⁺ activated K⁺ channels, is responsible for postshock APD shortening. These findings suggest that I₉₃ upregulation increases repolarization reserve of HF but may also be proarrhythmic by excessive shortening of APD. I₉₃ may be a novel target for antiarrhythmic therapy for ventricular arrhythmias.

**Keyword:** SK channels