THE EFFECTS OF CARDIAC RESYNCHRONISATION THERAPY ON INWARD RECTIFIER K+ CURRENT (IK1) IN DYSSYNCHRONOUS ISCHAEMIC HEART FAILURE

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Objectives To investigate the change in inward rectifier K+ current (IK1) in desynchronous ischaemic heart failure and the electrophysiological consequences of cardiac resynchronization therapy (CRT).

Methods The mode of desynchronous ischaemic heart failure of dogs was established by ablation of left bundle branch and ligation of left anterior descending artery (n=14). After CRT for 6 weeks (n=7), the myocytes of interventricular septal and anterior left ventricular wall were dissected and the whole cell membranous clamp was used to detect the IK1, and the hemodynamic and echocardiographic parameters were measured during the process.

Results The QRS intervals and the corrected QT durations in desynchronous ischaemic heart failure were prolonged compared with control (100±23ms vs 53±8 ms, p<0.05; 433±46 ms vs 378±32ms, p<0.05). CRT reduced the prolonged period of QRS and QTc in desynchronous ischaemic heart failure (73±11ms vs 100±23ms, p<0.05; 392±36ms vs 433±46ms, p<0.05). The peak inward IK1 densities in both interventricular septal and lateral myocyte in desynchronous ischaemic heart failure were reduced compared with control group (0.70±0.31 vs 1.60±0.28, p<0.05; 1.20±0.34 vs 1.75±0.31, p<0.05), and there was a significant difference in IK1 in desynchronous ischaemic heart failure between interventricular septal and lateral myocardium (0.70±0.31 vs 1.20±0.34, p<0.05). CRT restored partially these changes in IK1 induced by dyssynchronisation via increasing IK1 in both interventricular septal and lateral myocardium (1.50±0.30 vs 0.70±0.31, p<0.05; 1.65±0.39 vs 1.20±0.34, p<0.05) and reducing the difference in IK1 between interventricular septal and lateral myocardium in dyssynchronous ischaemic heart failure (1.50±0.30 vs 1.65±0.39, p>0.05).

Conclusions CRT reversed partially the IK1 remodelling in desynchronous ischaemic heart failure, whereby reduced the regional heterogeneity of IK1.