THE TIME COURSE OF ELECTRICAL REMODELLING, STRUCTURAL REMODELLING, NEURAL REMODELLING AND ENDOTHELIN-1 LEVEL IN ATRIAL FIBRILLATION

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Objectives The consistency between electrical remodelling, structural remodelling, neural remodelling and endothelin-1 level in atrial fibrillation (AF) has not been reported. The present study aimed to investigate the time course of atrial electrical remodelling, structure remodelling and neural remodelling as well as endothelin-1 (ET-1) in AF rabbit models induced by rapid atrial pacing, and to explore the possible values of ET-1 in predicting the initiation and development of AF so as to guide the treatment for AF.

Methods Forty Adult New Zealand rabbits, rapid atrial pacing (600 bpm) was performed to make paroxysmal AF models. The experimental animals were randomly divided into five groups by pacing time, 0 h group (P0), 4 h group (P4), 8 h group (P8), 12 h group (P12), 24 h group (P24), (n=8). The AF induction rate, AERP, rate adaptation of AERP, ventricular ejection fraction (EF), heart rate variability (HRV), plasma ET-1, FPdur and the field potential amorphous of atrial tissues, anti-growth-associated protein-43 (GAP-43), anti-choline acetyl transferase (ChAT), anti-tyrosine hydroxylase (TH) and anti-endothelin-1 (ET-1) antibodies in atrial tissues were measured after pacing 0 h, 4 h, 8 h, 12 h, 24 h.

Results The success rate of rapid right atrial pacing 24 h in rabbits for building AF model was 95%, 25% rabbit normal heart can be induced to AF. The induction rate at pacing 0 h, 4 h, 8 h, 12 h, 24 h were 25%, 37.5%, 65%, 75%, 85%, respectively. After pacing 8 h, AERP was significantly shortened, the rate adaptive of AERP was significantly decreased, the Fpdur of atrial tissues was significantly prolonged. The shortening of general AERP was inhomogeneous, but the dispersion of Fpdur was increased. The EF was significantly increased after AF 8 h and atrial pathological changes started at AF 24 h. The mean R-R interval was significantly decreased at pacing 4 h, but increased to the maximum at pacing 24 h. The LF and HF was significantly increased at pacing 12 h and 24 h, respectively. The LF/HF was gradually decreased and significantly decreased at pacing 24 h. After pacing 24 h, the density of GAP43-positive, ChAT-positive and TH-positive in the left and right atrium were higher than the density in 0 h group; Furthermore, after pacing 24 h, the left atrium had significantly higher nerve density of GAP43-positive and ChAT-positive than the right atrium. Plasma ET-1 was significantly increased after pacing 12 h and rise to the maximum after 24 h. There was no significant changes of ET-1 in atrial tissues after pacing, it was significantly increased until pacing 24 h.

Conclusions The atrial electrical remodelling, structural remodelling, neural remodelling occurred uniformly at rapid atrial pacing.
The plasma ET-1 level significantly increased after AF persisted for 12 h. The plasma ET-1 level may be used to presume the duration time of AF. The increased atrial tissue ET-1 level after AF persisting for 24 h may be the trigger factors of ‘AF begets AF.’