EFFECT OF ANTIARRHYTHMIC PEPTIDE ON VENTRICULAR ARRHYTHMIA INDUCING BY LYSOPHOSPHATIDIC ACID

ACC Poster Contributions
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Background: Lysophosphatidic acid (LPA) is a bioactive phospholipid and has close relationship with cardiovascular diseases. However there are few studies about the proarrhythmia effect of LPA. We therefore investigated the effect and mechanism of LPA and antiarrhythmic peptide 10 (AAP10) on rabbit ventricular arrhythmia.

Method: 24 rabbits were randomly divided into three groups: control group, LPA group and AAP10+LPA group. By using arterially perfused rabbit ventricular wedge preparations, transmural electrocardiogram and action potential from both endocardium and epicardium were simultaneously recorded in the whole process of all experiments by two separate floating microelectrodes. The incidence of ventricular arrhythmia by S1S2 stimulation was recorded. The differences of the contents of nonphosphorylated connexin 43 (Cx43) and total Cx43 between each group were evaluated by western blot. The distribution of nonphosphorylated Cx43 was observed by confocal immunofluorescence microscopy.

Result: Compared with the control group, the QT interval, endocardial action potential duration, transmural dispersion of repolarization (TDR) and incidence of ventricular arrhythmia increased sharply with augmented nonphosphorylated Cx43 in the LPA group (P<0.01). Compared with the LPA group, AAP10 can reduce the QT interval, endocardial action potential duration, TDR and incidence of ventricular arrhythmia with decreased nonphosphorylated Cx43 in the presence of LPA.

Conclusion: LPA could promote the arrhythmia by increasing the content of nonphosphorylated Cx43 which may inhibit the gap junction transmission. Gap junction enhancer AAP10 could prevent the arrhythmic effect of LPA probably by reducing the content of nonphosphorylated Cx43.