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REMODELLING OF CONNEXIN 43 IN ATRIAL MYOCARDIUM OF PATIENTS WITH ATRIAL FIBRILLATION

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Objectives Remodelling of connexins was found accompanying with atrial fibrillation. The aim of the study is to investigate whether it is the remodelling of connexin 43 (Cx43) plays an important role during the initiation and maintenance of atrial fibrillation.

Methods Samples of right atrial appendage were taken from 30 patients with rheumatic valvular disease during surgery. Fibrosis and remodelling of connexin 43 was examined by microscopy technique and analysed by image analyser. the volume fraction of Cx 43 (Cx 43 VF) were studied between atrial fibrillation (AF) and sinus rhythm (SR) groups.

Results Microscopic examination demonstrated that Cx43VF significantly decreased in patients with AF compared to those with SR.

Conclusions It has been established that gap junctions between cells are of great importance for electrical conduction in human heart. Each junction allows small, water soluble molecules to move directly between the cytoplasm of the two cells in contact, which means that both cells share metabolites and even electrical properties. Gap junction between cardiocytes provides the normal pathway for electrical conduction, and accordingly, assures action potential to propagate uniformly. In cardiac tissue, connexin40, connexin43 and connexin45 which construct gap junctions have been detected; connexin40 and connexin43 are the main components of gap junction in the atrium. It is shown that depletion of Cx40 in mice atrial myocardium was accompanied with changes in atrial electrical coupling and enhancement of susceptibility for arrhythmia. Atrial fibrillation is a kind of electricity turmoil in the atrial myocardium. Many studies have demonstrated redistribution and remodelling of connexins in patients with atrial fibrillation. Gap junction distribution becomes progressively more polarised with increasing age in canine atrial muscles and localised to the cell termini. In this experiment, abnormal distribution of Cx43 in the human right atrial appendage was detected. The Cx43VF in patients with AF was significantly lower in comparison with the SR group; the result was similar with the study done by Kostin S *et al*. The change may improve the anisotropy of the impulse conduction through atrium and result in severe heterogeneity, thus to initiate and maintain atrial fibrillation.