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DISLOCATED AMPLATZER OCCLUDER IN THE LEFT ATRIUM SURGICAL RETRACTION AND PFO CLOSURE

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A 42 year old man with recurrent episodes of TIA's underwent heart catheterization and was diagnosed a PFO defect. Insertion of an 18 mm Amplatzer occluder under TEE guidance was performed and additional angiographic evaluation showed complete closure of the defect (Fig. 1). The next day, further assessment with MRI technique revealed a dislocation with a persistent left-right shunt of 9 %, and the patient was referred to the cardiac surgery department (Fig. 2).

Opening of the right atrium displayed the Amplatzer occluder in the left atrium with an atrial septal aneurysma (Fig. 3). After simple removal of the occluder and excision of the aneurysm, a direct closure was performed and the patient was discharged 5 days latter.

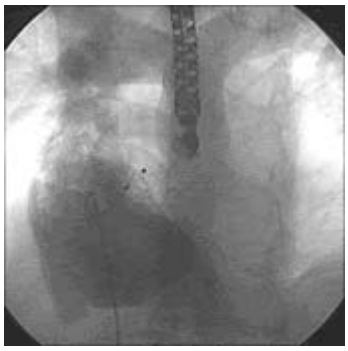

Fig. 1

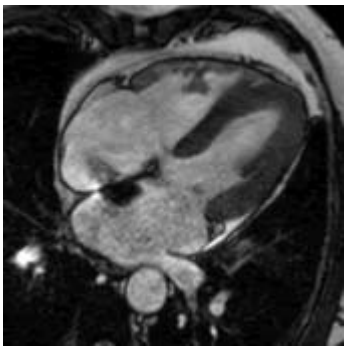

Fig. 2

Fig. 3

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DECREASED ACTIN EXPRESSION IN PATIENTS WITH CHRONIC ATRIAL FIBRILLATION

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Background - Chronic atrial fibrillation is characterized by a severe contractile dysfunction and myolysis. Remodelling of the cellular ultrastructure develops progressively. Myolysis is associated with the replacement of sarcomeres by glycogen. The aim of our study was, to determine if myolysis is represented by a reduction in actin concentration.

Methods - Right atrial samples from 18 patients undergoing elective cardiac surgery were excised and immediately frozen in liquid nitrogen. 8 patients had chronic AF (≥3 month) and 10 patients were in sinus rhythm (SR). Actin concentration was determined by SDS-PAGE, Western blot and quantified by optical densitometry.

Results - Immunoblot analysis demonstrated actin expression in all hearts. In myocardial samples from patients with chronic AF we found a 2.1 fold reduction in actin expression. ($p<0.001$)

Conclusions - The decrease in actin concentration via myolysis, might decrease energy consumption and be an additional mechanism for contractile dysfunction in chronic AF.

Key Words: atrial fibrillation, heat shock proteins, actin, western blot

1. Introduction

In human atrial fibrillation (AF) [1,2] and pacing induced AF [3,4] substantial changes in atrial myocyte architecture[5,6] and function[7,8] have been reported. Severe myocyte alterations, characterized by enlarged myocytes and myolysis, is observed in fibrillating atria.[9]

Remodelling of the cellular ultrastructure develops progressively. The majority of the cardiomyocytes exhibited marked changes in their cellular substructures, with the replacement of sarcomeres by glycogen as the main characteristic.[10]

The first sign of cellular structural remodeling is a more homogeneous chromatin distribution. Sub-structural changes in mitochondria and sarcoplasmic reticulum occurred gradually. Cellular degeneration was absent. The degree of myolysis and glycogen accumulation increased. Almost half of the myocytes in the right atrial free wall were affected by myolysis. [11]

The aim of this study was to examine if myolysis, with the replacement of sarcomeres by glycogen granulas is rep-