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Atrial longitudinal strain paramaters predict left atrial reverse remodeling after mitral valve surgery: a speckle tracking echocardiography study

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Purpose: Volume overload in chronic severe mitral regurgitation causes left atrial remodeling. MV surgery usually results in left atrial (LA) volume reduction. In patients undergoing mitral valve surgery, LA reverse remodeling was related to better postoperative clinical outcome and survival previously. However, only few clinical and echocardiographic parameters were suggested to be associated with LA reverse remodeling. In this study we investigated the relationship between LA peak longitudinal strain (reservoir strain) assessed with 2-dimensional speckle tracking imaging (2D STI) and LA reverse remodeling.
Methods: 53 patients (24 females and 29 males, mean age: 45.7±13.5 years) with severe mitral regurgitation and preserved left ventricular systolic function were included in the study. All patients had normal sinus rhythm. The etiology of mitral regurgitation was mitral valve prolapse (MVP) in 37 patients and rheumatic valvular disease in 16 patients. Mitral valve repair was performed in 30 patients while 23 underwent mitral valve replacement. Echocardiography was performed before the surgery and six months later. Left atrial peak longitudinal strain (PLS) was assessed with speckle tracking imaging. LA reverse remodeling was defined as a percent of decrease in LA volume index (LAVI).
Results: Left atrial volume index significantly decreased after surgery (58.2±16.6 ml/m^2 vs. 43.9±17.2 ml/m^2 p: < 0.001). Mean LAVI reduction was 22.5% ± 27.2. There was no significant difference in LAVI reduction between mitral repair and replacement groups (22.1±22.6% vs. 23.1±32.8% p: 0.9). Besides decrease in LAVI was also similar in patients with MVP and rheumatic valve disease (24.4±26.8% vs. 18.2±28.9% p: 0.4). Correlates of LAVI reduction were preoperative LAVI (r: 0.28 p: 0.039), LA PLS (r: 0.36 p: 0.001) and age (r: -0.36 p: 0.007). Furthermore, in multivariate linear regression analysis, preoperative LAVI, LA PLS and age were all significant predictors of LA reverse remodeling.
Conclusion: Left atrial peak longitudinal strain measured by 2D STI, in conjunction with preoperative LAVI and age is a predictor of LA reverse remodeling in patients undergoing surgery for severe mitral regurgitation. We suggest that in this patient population LA GLS may also be used as a preoperative prognostic marker.

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Left atrial remodeling in patients undergoing transcatheter aortic valve implantation: a speckle tracking prospective study

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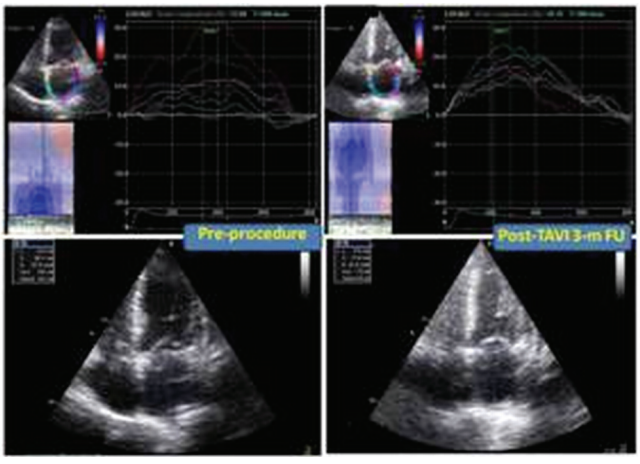
Background: Aortic Stenosis (AS) results in several Left Ventricular (LV) disturbances as well as progressive Left Atrial (LA) enlargement and dysfunction. Transcatheter Aortic Valve Implantation (TAVI) reverses LV remodelling and improves overall systolic function but its effect on LA function remains undetermined. The aim of this prospective study was to investigate the effects of TAVI on LA structure and function.
Material and methods: We studied thirty-two patients with severe symptomatic AS and high surgical risk who underwent TAVI, using standard and 2-dimensional speckle-tracking echocardiography before, at 40-day and at 3-month follow-up.
Results: Following TAVI, mean transvalvular gradient reduced (p<0.001). Both LA mean area index and LA mean volume index decreased at 40-day (16.2±6.4 vs. 12.5±2.9 cm^2/m^2, and 47.3±12.0 vs. 42.8±12.5 mL/m^2, respectively, p<0.05) and values remained unchanged at 3 months. The reduction of LA size was accompanied by a significant increase in global PALS (14.4±3.9% vs. 19.1±4.7%, p<0.001) and global PACS (8.4±2.5% vs. 11.0±4.1%, p<0.05) at 3-month. After the procedure, LA stiffness measurements decreased and became significant at 3-month follow up (p<0.001). Pre-procedural trans-aortic mean gradient and pre-procedural LA volume were identified as predictors of global PALS increase (p<0.0001) while the delta drop in trans-aortic mean gradient as predictors of LA volume index reduction 3 months after TAVI (p<0.0001).
Conclusion: TAVI is associated with significant recovery of LA structure and function suggesting a reverse cavity remodelling. Such functional recovery is determined by the severity of pre-procedural valve stenosis.



Atrial remodelling after TAVI

MITOCHONDRIA: BEYOND ENERGY METABOLISM IN CARDIAC PATHOPHYSIOLOGY

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Involvement of Monoamine Oxidase a (MAO-A) in mitochondrial fission and autophagy during aging

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Cardiac expression of monoamine oxidase A (MAO-A), an enzyme localized into the mitochondrial external membrane, increases during aging and is usually associated with heart failure. In previous studies, we have observed mitochondrial fission in 2 years old mice and in 30 weeks old MAO-A-transgenic mice. Here, we study the role of fission and autophagy in mitochondrial quality control associated with MAO-A.
Several in vitro and in vivo models were used: (1) cardiomyocytes from neonatal rats or (2) adult rats, infected or not with a MAO-A adenovirus and treated with tyramine, a MAO-A substrate; and (3) an aging model of MAO-A cardiac overexpressing mice (TG-MAO-A) or MAO-A-KO mice. Proteins of interest were studied by western blot and mRNA by Real-Time PCR. In parallel, immunofluorescence and electronic microscopy were achieved.
In vitro, we observe an autophagosome accumulation, via a LC3-GFP staining, in neonatal cardiomyocytes infected with MAO-A and treated with tyramine. These results are accompanied with an increased ratio of LC3-II/LC3-I. These effects were abolished by a ROS scavenger (NAC) or an autophagy inhibitor (3-methyladenin). In vivo, 6 weeks old TG-MAO-A mice present an increased DRP1 and Fis1 mRNA, implicated in mitochondrial fission and a decrease of Mfn1 and Opa1 mRNA, implicated in mitochondrial fusion, compared to littermates. We also observe an increase of Beclin-1 and Parkin proteins in these TG-MAO-A mice at 30 weeks. These variations are correlated with the observation of autophagosomes using electronic microscopy. Conversely, DRP1, Beclin-1 and Parkin proteins remain unchanged in 24 month old MAO-KO mice.
This study is the first to explore the role of monoamine oxidase A in mitochondrial quality control during aging.

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Eicosapentaenoic acid mediates mitochondrial fatty acid composition and fusion protein OPA-1 in associated with preservation of oxidative phosphorylation after myocardial infarction

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Purpose: Eicosapentaenoic acid (EPA) is a first line drug in the management after myocardial infarction. Mitochondria are major contributors to energy metabolism and recent mounting evidence suggests that mitochondrial dynamics, such as fusion and fission, has a pivotal role in regulating mitochondrial function. This study was designed to determine whether oral EPA mediates mitochondrial fatty acid composition, dynamics, and oxidative phosphorylation, leading to the attenuation of cardiac remodeling after myocardial infarction (MI).
Methods and results: Anterior MI was produced in male rats by ligating the left anterior descending coronary artery (MI group). In the EPA-treated group, EPA (1,000 mg/kg/day) was administrated for 12 weeks after coronary ligation (MI+EPA group). Myocardial infarct size and blood pressure were comparable between groups. At 12 weeks after MI, mitochondria were isolated in non-infarcted myocardium and mitochondrial fatty acid composition was determined using gas chromatography mass spectrometry. In EPA+MI group, mitochondrial EPA content was approximately 10 times higher than that in untreated-MI group. Cardiac function was assessed by echocardiography and 2F micro-manometer-tipped catheter at 12 weeks of MI. EPA significantly improved %fractional shortening, +dP/dt, and -dP/dt, and reduced left ventricular (LV) end-diastolic diameter and pressure. In addition, histological examination showed EPA significantly suppressed myocyte hypertrophy and interstitial fibrosis in non-infarcted myocardium by 15% and 30%, respectively. Levels of ATP in cardiac tissue were measured by high-performance liquid chromatography and mitochondrial oxidative phosphorylation was assessed by O2 consumption using isolated mitochondria. After 12 weeks after MI, ATP contents in non-infarcted myocardium were significantly decreased, and mitochondrial complex II, III, and IV activities were also impaired, while EPA