## Patterns of left ventricular longitudinal myocardial dysfunction in mitral valve prolapse and effects of valve repair

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**Background:** Regional longitudinal left ventricular (LV) dysfunction in patients with mitral regurgitation (MR) due to valve prolapse (MVP) with normal ejection fraction has been recently described, with data pointing at dysfunction of the LV base related to dilatation of the mitral annulus.

**Purpose:** To investigate degree and extent of regional LV dysfunction and its mechanisms in patients with MVP and severe acute (MRa, n=27) or chronic (MRc, n=41) MR and no coronary disease, undergoing surgical valve repair with 3 months follow-up (FU); 20 normal subjects were used as controls (N).

**Methods:** Speckle-tracking echocardiography was performed pre- (Bas), 1 week (1w) and 4 months (4mo) post-operatively to measure longitudinal global (GLPSS, %), regional (RPSS, %) and segmental (SPSS) peak systolic strain. Maximum and minimum mitral annulus (MA) diameters were measured with 3D echo at Bas. We also evaluated: LV end-diastolic volume index (EDVi, ml/m²); ejection fraction (EF, %); left atrial end-systolic volume index (LAVi, ml/m²); non-invasive pulmonary systolic pressure (PSP, mmHg).

**Results:** Risk factors (hypertension, diabetes, atrial fibrillation, smoke and previous stroke) were similar in MRc and MRa. At Bas EDVi was larger by definition in MRc (MRc:  $102\pm21$ , MRa:  $67\pm10$  ml/m², p<0.001) as LAVi ( $101\pm46$  vs  $76\pm31$  ml/m², p=0.035). Both EF ( $65\pm8$  vs  $64\pm8$  ml/m²) and GLPSS ( $-20\pm4$  vs  $-21\pm5\%$ ) were normal, but RPSS was reduced, only

at the base (-13±6 vs -13±6%, p= ns; N: -18±2, p<0.03 vs MRc and MRa) in MRc and MRa, with reduced SPSS localized at anterior, lateral and posterior - but not septal - segments. At 1w, EF decreased in both MRc (47±14%, p<0.001 vs Bas) and MRa (56±10%, p=0.014 vs Bas), together with GLPSS (MRc: -11±4%, p<0.001 vs Bas: MRa: -13±4. p<0.001 vs Bas) driven by a prevalent marked decrease in RPSS (MRc: -7±4%, p<0.001 vs Bas; MRa; -8±5, p<0.001 vs Bas) of the LV base. All patients were alive at 3 months with no MACEs, similar reduction of mean MR grade (MRc:  $4\pm0$  to  $1.9\pm0.7$ , p<0.001; MRa:  $3.9\pm0.3$  to  $0.9\pm0.9$ . p<0.001) and PSP (MRc: 50±23 to 29±5 mmHg, p<0.001; MRa: 42±22 to 32±6 mmHg, p=0.039), normal EDVi (MRc: 70±27, MRa: 49±10 ml/m<sup>2</sup>), dilated LAVi (MRc: 101±46, MRa: 54±13 ml/m<sup>2</sup>), and reduced GLPSS (MRc: -12±5%, p<0.001 vs Bas; MRa; -15±3, p=0.001 vs Bas) and base RPSS (MRc:  $-7\pm6\%$ , p=0.004 vs Bas; MRa;  $-10\pm4$ , p= ns vs Bas). At multivariate analysis, regional dysfunction was not related to the prolapsing scallop, presence of flail, commissure involvement, dimension and geometry of the MA, EF or pulmonary pressures.

**Conclusions:** In patients with MVP and severe MR, there is a specific regional longitudinal dysfunction pattern prevalent at the LV base which may be related to the duration of MR but not to annular dilatation or morphology of the prolapsing leaflets. The dysfunction worsens greatly following acute reduction of preload after surgical repair and is still significant at 4mo FU.