assessed by conventional and tissue Doppler imaging echocardiography. Immediately following PTMC, mitral valve area increased from 0.91±0.29 cm² to 1.86±0.43 cm² (P < 0.0001) and RV outflow tract fractional shortening (RVOTfs) increased from 57±15% to 72±12% (P = 0.002). There was a significant decrease in systolic pulmonary artery pressure from 46.4±32 mmHg to 29.1±13 mmHg (P = 0.04) and RV Tei index from 0.44±0.025 to 0.29±0.17 (P = 0.001), in myocardial acceleration during isovolumic contraction (IVC) at the lateral tricuspid annulus from 0.36±0.11 m/s² to 0.25±0.07 m/s² (P = 0.023), and in isovolumic contraction velocities at the lateral tricuspid annulus from 11.03±3.37 cm/s to 8.50±2.04 cm/s (P = 0.034). Conclusion Immediately after successful PTMC, significant decrease in RV contractility as assessed by IVA was observed whereas other parameters of infundibular and global RV function as assessed by RVOTfs and Tei index showed significant improvement. These discordant results may be related to the relative insensitivity of currently available echocardiography parameters of RV function that are not completely immune to loading conditions.

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Prognosis of infective endocarditis : EIMONA Registry

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Despite the major advances in diagnostic technology, improvements in antimicrobial selection, and advances in surgical techniques, infective endocarditis remains a disease with high mortality rate, nearly 20%.

Aim: To determine predictive factors of mortality of infective endocarditis.

Methods: EIMONA registry includes 220 patients diagnosed with IE according to the Duke University criteria between 1997 and 2007 (125 Men, mean age 36 ± 17 years).

Results: In-hospital mortality was 18% (41 patients). Endocarditis patients who died were significantly older (51 ± 9 versus 28 ± 14, p = 0.023), and had more frequently symptoms of heart failure (57.2% vs 26.5%, p<0.01). Biolog-ical and echocardiographic characteristics were similar, except for serum creat-inine (129.5 ± 24 versus 86.5 ± 30, P=0.07).

Univariate analysis on potential risk factors revealed three significant fac-tors: renal failure, embolic accident and heart failure. These same factors were also independent factors for death in multivariate analysis, with OR=3.2 (95% CI: 1.1-9.6, p=0.03) for renal failure, OR=2.3 (95% CI: 1.1-5, p=0.03) for embolic accidents and OR=4 (95% CI: 1.6-9.8, p = 0.002) for heart failure.

Conclusion: Infective endocarditis was a complex disease with poor prog-nosis; our findings may help to identify higher-risk patients for more aggres-sive treatment or interventions.

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Comprehensive annular and subvalvular repair of chronic ischemic MR provides best long-term results with least ventricular remodeling

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Background: In ischemic mitral regurgitation (IMR), leaflet tethering is caused by post-MI LV and annular remodeling. Severing second-order mitral chordae significantly decreases tethering and MR. We tested whether under-sized ring annuloplasty can improve chordal cutting efficacy by reducing annulus-related tethering.

Methods: Posterolateral MI created chronic remodeling and MR in 28 sheep. At 3 months, sheep were randomized to sham surgery vs isolated annu-loplasty undersized by 2 sizes vs isolated bileaflet chordal cutting vs at the combined therapy (n=7 each). At baseline, chronic MI (3 months) and sacrif-ice (6.6 months) we measured LV volumes and ejection fraction (EF), wall motion score index (WMSI), MR Regurgitation fraction (MRRF) and vena contracta (VC). Mitral annulus area (MAA) and posterior leaflet (PL) restriction angle (PL to MAA) by 2D and 3D echo.

Results: All groups were comparable at baseline and chronic MI, with mild- moderate MR (MRVC 4.6±1.0mm, MRRF 24±2.6%) and MA dilatation (p=0.01). At sacrifice, LV end-systolic volume (ESV) increased by 108 % in controls vs 28% with ring + chordal cutting, less than with each intervention alone (p=0.01). Also, MR progressed to moderate-severe in controls but decreased to trace with ring + chordal cutting vs mild-moderate with ring alone and trace-mild with chordal cutting alone (MRVC 5.9±1.1mm in con-trols, 2.0±0.7 with ring, 1.0±0.9 with chordal cutting, 0.5±0.8 with both, p<0.01). Ring alone did not improve PL mobility, but chordal cutting did alone or with ring (PL restriction angle 54±5° vs. 45±2° with ring, p=NS). In multivariate analysis, LVESV and MAA most strongly predicted MR (r²=0.82, p<0.01). Conclusions: Comprehensive annular and subvalvular repair provides the most effective long-term reduction of both chronic ischemic MR and LV remodeling.

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Investigation of the myxomatous mitral valve prolapse locus on chromosome 16

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Myxomatous mitral valve prolapse (MMVP), also called Barlow disease, is a common cardiac disorder characterized by fibromyoxatous changes in the mitral leaflet tissue causing prolapse. Familial studies suggest a genetic heter-oogeneity and four loci have been identified to date. The MMVP1 locus that we identified in the past in two French families (Disse, Am J Hum Genet 1999) maps on chromosome 16p11-12. The locus interval is relatively large (15 cm). It comprised 185 genes but none has been implicated in the disease, yet.

The objective of this work was to test relevant positional candidate genes. Six genes were tested by direct sequencing of all the coding sequences and of the intron-exon boundaries. For each gene, two affected and two unaffected patients of the two families linked to MMVP1 were tested, as well as 46 probands of smaller MVP families.

We chose 3 genes belonging to the nodal modulators family that interact with proteins of the TGFβ protein superfamily and known to play a role in heart valve formation and in regulation of collagen and extracellular matrix gene expression. The Nomol1, Nomol2 and Nomol3 genes (located on 16p13.11, 16p12.3 and 16p13.11 respectively) extend on approximately 63 Kb and contain each 30 exons. No pathogenic mutation was identified in the coding region of these genes in these families but only known polymorphisms or rare non-coding variants. We also analysed 3 members of the heparan sulfate sul-fotransferase enzymes (HS3ST1, HS3ST2 and HS3ST4) that regulate the heparan sulfate proteoglycan biosynthesis process. The HS3ST2 and HS3ST4 genes are located on 16p12, are extended on approximately123 Kb, but contain each only two exons separated by a large intron. Similarly, the genetic analysis was negative and no pathogenic mutation was identified.

This study illustrates the difficulty to identify the causal gene when the linkage interval and the number of genes are large. Priority should be give to fine mapping of the MMVP1 locus.