**ATRIAL FUNCTIONAL MITRAL REGURGITATION: THE LEFT ATRIUM GETS ITS DUE RESPECT**

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Moderate or greater mitral regurgitation (MR) is the most frequent valve disease in the United States. Competence of the mitral valve requires the temporally and spatially coordinated interaction of the mitral leaflets with the annulus, chordae tendinae, and papillary muscles; dysfunction of any of these components will affect the normal systolic coaptation of the mitral leaflets and cause mitral regurgitation; whether annular dilatation alone is sufficient for the development of MR is controversial. The mitral annulus is a thin fibrofatty ring that is bordered by variable insertions of atrial and ventricular myocardium. The nonplanarity and hyperbolic paraboloid shape of the annulus reduces mechanical stress on the leaflets and the its contraction reduces annular area and facilitates normal leaflet coaptation. Data suggest that anterior mitral leaflet musculature modulates leaflet stiffness, assists valve closure, and influences the three-dimensional geometry and function of the annulus and anterior mitral leaflet. Although functional MR has been associated with atrial fibrillation (AF), more recent data in patients referred for a first AF ablation who had both a baseline echocardiogram and one year clinical follow-up, normal LV systolic function, and at least moderate mitral regurgitation, suggest a more causal relationship; thus, maintenance of sinus rhythm resulted in greater reductions in left atrial volume and mitral annular diameter and less MR than in those whose arrhythmia recurred. While these data are provocative, they conflict with conventional dogma and should be considered hypothesis-generating. The precise mechanism of atrial functional mitral regurgitation is not clear but may be due to the effects that fibrillation-induced atrial remodeling has on atrial functions and synchrony; and annular size, geometry, and function. The prevalence of atrial functional regurgitation is unknown and its significance needs to be rigorously tested. Until then, it provides an intriguing, but untested rationale for aggressive rhythm control of AF.