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Authors: E. AGABITI-ROSEI - J. ALDERSHILE - G. AMBROSIO - L. BADIMON - JP. BASSAND - A. BAYÉS DE LUNA - M.E. BERTRAND - E. CHAZOV - S. CHIERCHIA - J. CLELAND - D. CLEMENT - D. COKKINOS - J.M. DETRY - R. DIETZ - P. DOMINIAC - I. EDES - R. FERREIRA - H.R. FIGULLA - W. FLAMENG - I. GRAHAM - W. JANUSZEWICZ - J.C. KASKI - P. KEARNEY - W. KLEIN - F. KOLBEL - M. KOMAJDA - W. KÜBLER - T. LÜSCHER - G. MANCIA - W.J. MCKENNA - T. MEINERTZ - J. MLCZUCH - D. MULCAHY - E. O'BRIEN - A. OTO - J. PAPP - W.J. PAULUS - J. POLONIA - I. PRÉDA - L.A. PROVIDENCIA - J. REID - W.J. REMME - W. RUZYLLIO - Z. SADOWSKI - P. SERRUYS - P. SLEIGHT - J. SOLER-SOLER - J. SOMERVILLE - P.G. STEG - H.A.J. STRUIJKER BOUDIER - B. SWYNGHEDAUW - L. TAVAZZI - M. TENDERA - K. THYGESEN - P. TOUTOUZAS - A. VAHANIAN - J.L. VANOVERSCHELDE - C.A. VISSER - J. WIDIMSKY - M. YACOUB - P. ZARCO

LEFT AND RIGHT VENTRICULAR FUNCTION IN MITRAL REGURGITATION: IMPORTANCE OF THE SUBVALVULAR MITRAL APPARATUS

Chronic mitral regurgitation (MR) results in a progressive deterioration in left ventricular (LV) contractile function,¹ despite long-term preservation of LV ejection fraction (EF), related to the low impedance pathway constituted by the left atrium. Indeed, LVEF overestimates ventricular function in MR, and a value of less than 60% is associated with significant muscle dysfunction in this setting.² Unfavorable preoperative LV geometry (LV dilatation with eccentric hypertrophy) resulting from chronic volume overload aggravates the potential for reduced postoperative ventricular performance. Although LV contractile impairment recovers toward normal in most patients after mitral valve surgery, patients with a LVEF of less than 60% before surgery for MR have a poor postoperative outcome.² However, mitral valve repair leads to a better outcome than mitral valve replacement in this condition.³ Transection of part of the subvalvular apparatus during mitral valve replacement usually results in a change in LV geometry, an increase in LV afterload, and depressed LV systolic performance with regional myocardial contractile abnormalities.^{4,5} The papillary muscles play an important role in both LV and mitral valve function. They contract during systole, contributing to ejection by drawing the mitral ring toward the apex, thereby causing shortening of the long axis and sphericity of the chamber. After chordal resection, the papillary muscles can no longer assist contraction at their insertion sites or in contiguous areas.⁶ Moreover, resection of the chordae tendinae may disrupt the muscle bundle alignment and induce contractile abnormalities remote from the area of papillary muscle insertion.

In a canine model, anterior chordal transection (chordae tendinae inserting on the anterior leaflet) had a considerably more deleterious effect on LV systolic function than section of the chordae to the posterior leaflet.⁷ This fact may be explained by the concept of regional afterload distribution. The anterior leaflet is larger and development of tension in the chordae to this leaflet should be greater at a given LV pressure. Indeed, despite preservation of the posterior chordae tendinae, anterior chordal transection during mitral valve replacement in a recent study was associated with significant impairment of LV regional function in the area of anterior papillary muscle insertion (apicolateral area). Anterior chordal transection may also disturb LV torsional deformation during systole.

In contrast, preservation of the mitral valve apparatus during mitral valve surgery for MR, particularly with mitral valve repair, maintaining ventricular shape and decreasing LV volume and

systolic wall stress, improves postoperative LV function and survival.³ Several studies also demonstrated better results in mitral valve replacement with subvalvular preservation,⁸ but these techniques are not in widespread routine use and are not always feasible.

Right ventricular (RV) function is depressed in severe MR.^{5,9} The impairment of RV performance is related to LV volume overload and pulmonary artery pressure. Whereas LV pressure overload improves RV function, LV volume overload impairs septal function and thus RV function. Moreover, it has been found that RVEF is inversely related to pulmonary artery pressure⁹ in MR. Although Borer et al¹⁰ reported a rapid improvement in RVEF after valve surgery, RV function improved after mitral valve repair but remained unchanged after mitral valve replacement in a recent study.¹¹ The lack of improvement in RVEF was explained in part by higher postoperative levels of systolic pulmonary artery pressure in the replacement group compared with the repair group. However, the main finding was that anterior chordal transection induces significant impairment of regional function in the apicoseptal area of the RV.¹¹ These postoperative regional contractile abnormalities after papillary muscle resection may be involved in the observed impairment of RVEF after mitral valve replacement. After anterior chordal transection, chamber shape is modified with dilatation of the left ventricle.⁷ This geometric alteration might influence directly regional RV function in the apicoseptal area. Another explanation lies in the complex architectural structure of the heart. Section of the mitral subvalvular apparatus is followed by a decrease in LV contractility, an increase in regional afterload,⁷ and a decrease in the preload of fibers along the circumference of the heart. These important modifications may induce a RV myocardial fiber disequilibrium predominantly in the apicoseptal area of the right ventricle.

In MR, chronic volume overload leads to LV dilatation and impairment of LV contractile function despite long-term preservation of LVEF. An LVEF of less than 60% is associated with significant impairment of LV systolic function. Depression of RV function is related to LV volume overload and to pulmonary artery pressure. Transection of the chordae tendinae during mitral valve replacement impairs both global and regional LV and RV function. 0

T. LE TOURNEAU - Lille, France

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