[10] Basso C, Thiene G, Corrado D, Angelini A, Nava A, Valente M. Arrhythmogenic right ventricular cardiomyopathy. Dysplasia, dystrophy or myocarditis? Circulation 1996; 94: 983–91.
[11] Naito H, Arisawa J, Harada K, Yamagami H, Kozuka T, Tamura S. Assessment of right ventricular regional contraction and comparison with the left ventricle in normal humans: a cine magnetic resonance study with presaturation myocardial tagging. Heart 1995; 74: 186–91.
[12] Sakuma H, Fujita N, Foo TK *et al.* Evaluation of left ventricular volume and mass with breath-hold cine MR imaging. Radiology 1993; 2: 377–80.

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Impact of systolic and diastolic dysfunction on postoperative outcome in patients with aortic stenosis

See page 1977 for the article to which this Editorial refers

In most patients with aortic stenosis, long-term survival after aortic valve replacement is excellent. In the absence of coronary artery disease, operative mortality is less than 2–3% and 10-years survival is more than 85%. Early and late mortality is dependent on various factors such as age, clinical symptoms, severity of valve disease, left ventricular function, presence of coronary artery disease etc. In this issue Lund *et al.*[1] evaluate several parameters, such as left ventricular systolic and diastolic dysfunction as predictors for postoperative outcome in patients with severe aortic stenosis. Left ventricular systolic dysfunction was defined as an ejection fraction of less than 61%, a peak ejection rate of less than 2·29 end-diastolic volumes per second and a prolonged time to peak ejection. Left ventricular diastolic dysfunction was defined as a peak filling rate of less than 2·86 end-diastolic volumes per second in patients younger than 49 years and of less than 2·00 in those older than 50 years. Furthermore, a diminished fast filling fraction of less than 69% in the younger and of less than 55% in the older patients, as well as a reduced late filling fraction and a prolonged time to peak filling, were evaluated as parameters for diastolic dysfunction. Lund *et al.* observed an adverse early and late outcome in patients with diastolic dysfunction either alone or in combination with systolic dysfunction. It is surprising that in patients with severe aortic stenosis the presence of systolic and diastolic dysfunction predicts an unfavourable postoperative outcome, since it is well known that most patients with this valve disorder already have left ventricular diastolic dysfunction at an early stage of the disease when left ventricular hypertrophy is present[2–6].

Left ventricular systolic dysfunction caused by afterload mismatch usually improves after aortic valve replacement, but recovery is delayed or improvement lacking when it results from myocardial dysfunction[7]. Coronary artery disease is an important confounding factor which contributes to left ventricular dysfunction and which is associated with an enhanced risk for an adverse outcome after aortic valve replacement. Recently, the postoperative survival rate in patients with aortic stenosis with reduced left ventricular ejection performance has been shown to be similar to an age-matched control group[3]. In contrast, the presence of coronary artery disease, especially a history of previous myocardial infarction, has been shown to be an independent risk factor for an adverse outcome and reduced survival after valve replacement. In the study of Lund *et al.*[1] more than 40% of all patients had coronary artery disease and some of them had previous myocardial infarction. Thus, early and late mortality may have been influenced by this risk factor, since coronary artery disease influences left ventricular ejection performance significantly and is one of the major causes of left ventricular systolic dysfunction.

Left ventricular diastolic dysfunction has been found in 50 to 60% of all patients with aortic stenosis and has been considered a major cause for the development of congestive heart failure[8]. The most common cause of diastolic dysfunction in patients with aortic stenosis is, however, left ventricular hypertrophy. In the study of Villari *et al.*[9] diastolic dysfunction was defined either as abnormal relaxation, decreased diastolic filling or increased myocardial stiffness. Diastolic dysfunction was observed in approximately 50% of all patients with a normal systolic ejection performance, but was found in 95% of those with depressed systolic function. Villari and coworkers reported that diastolic stiffness increases

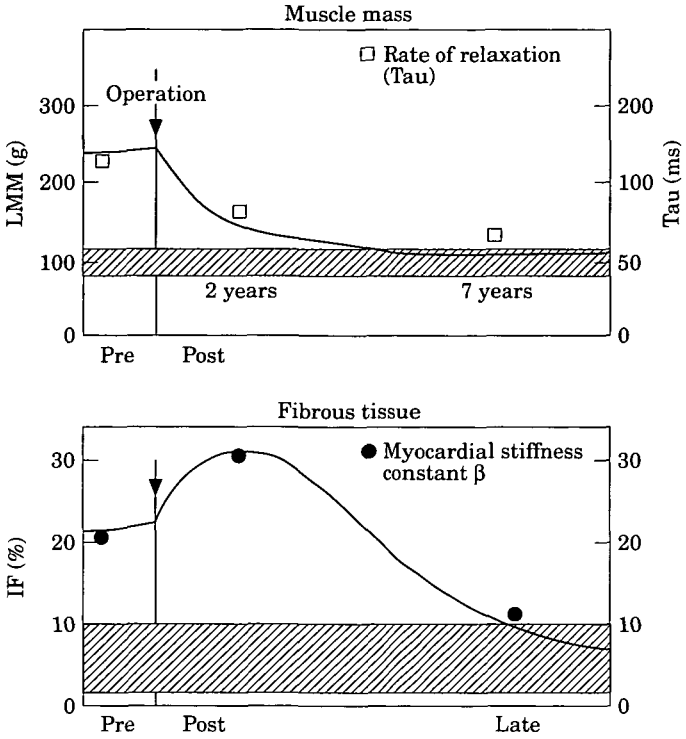

Figure 1 **Regression of muscle (top panel) and fibrous mass (bottom panel) in patients with aortic stenosis after successful valve replacement. The time of operation is indicated by the arrow. Early after operation, there is rapid regression of left ventricular muscle mass, which is normalized late after valve replacement. In contrast, fibrous mass shows a relative increase early after operation due to the rapid decrease in muscular tissue. Late after valve replacement, there is also regression of fibrous mass, which takes more time than regression of muscular tissue. LMM=left ventricular muscle mass; IF=interstitial fibrosis. (Data from Villari *et al.*, Circulation 1995; 91: 2353–8).**

early after valve replacement parallel to the 'relative' increase in interstitial fibrosis when muscle mass is decreasing, whereas relaxation improves with the decrease in left ventricular muscle mass[9].

Late after aortic valve replacement (up to 10 years) both diastolic stiffness and relaxation improve due to the regression of both muscular and collagen tissue. Thus, reversal of diastolic dysfunction in aortic stenosis takes years and is accompanied by a slow regression of interstitial fibrosis, whereas reversal of systolic dysfunction occurs more rapidly due to mechanical unloading, with a rapid decrease in muscle mass. Diastolic filling, however, remains unchanged after valve replacement, indicating that this parameter is relatively insensitive to changes in diastolic function but is highly influenced by age, loading conditions and hypertrophy. Excessive hypertrophy is, however, associated with a significant increase in postoperative mortality[5].

In the study of Lund *et al.*[1], load-sensitive or load-dependent systolic function parameters were used and diastolic function was assessed from pure filling parameters which are sensitive to changes in heart rate, loading conditions, relaxation rate, atrial and ventricular chamber properties, etc[6]. Despite these limitations, Lund and coworkers found that the risk for an adverse outcome is enhanced after aortic valve replacement, when diastolic and systolic dysfunction, either alone or in combination, are present. Furthermore, the presence of isolated diastolic dysfunction is considered to indicate aortic valve replacement.

The patients in the study of Lund *et al.* represent a highly selected group of candidates for valve replacement; they had diastolic dysfunction, but changes in relaxation rate and myocardial stiffness were not assessed. Since most patients with aortic stenosis have diastolic dysfunction, its presence can-

not be an indicator alone for aortic valve replacement. As a general rule, the indication for valve replacement is given when there is: severe valve obstruction (pressure gradient ≥ 50 mmHg and aortic valve area $\leq 0{\cdot}8\ cm^2$); clinical symptoms (angina pectoris, pre-syncope or syncope, congestive heart failure); inadequate pressure rise or pressure fall during exercise.

Conclusions

In the paper of Lund *et al.*[1] several risk factors have been identified which are associated with an adverse outcome after valve replacement, mainly the presence of systolic and diastolic dysfunction. These function parameters seem to be useful prognostic indicators for postoperative outcome, especially in patients with concomitant coronary artery disease. However, previous studies have shown that the occurrence of isolated diastolic dysfunction is no indication for earlier operation when valve stenosis is not severe.

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References

[1] Lund O, Flo C, Jensen FT *et al.* Left ventricular systolic and diastolic function in aortic stenosis. Prognostic value after valve replacement and underlying mechanisms. Eur Heart J 1997; 18: 1977–87.
[2] Lund O. Preoperative risk evaluation and stratification of long-term survival after valve replacement for aortic stenosis. Reasons for earlier operative intervention. Circulation 1990; 82: 124–39.
[3] Connolly HM, Oh LK, Orszulak TA *et al.* Aortic valve replacement for aortic stenosis with severe left ventricular dysfunction. Circulation 1997; 95: 2395–400.
[4] Hwang MH, Hammermeister KE, Oprian C *et al.* Preoperative identification of patients likely to have left ventricular dysfunction after aortic valve replacement. Participants in the Veterans Administration Cooperative Study on Valvular Heart Disease. Circulation 1989, 80: 165–76
[5] Orsinelli DA, Aurigemma GP, Battista S, Krendel S, Gaasch WH. Left ventricular hypertrophy and mortality after aortic valve replacement for aortic stenosis. A high risk subgroup identified by preoperative relative wall thickness. J Am Coll Cardiol 1993; 22: 1679–83.
[6] Hess OM, Villari B, Krayenbuehl HP. Diastolic dysfunction in aortic stenosis. Circulation 1993; 87 (5 Suppl): IV73–6.
[7] Snopek G, Pogorzelska H, Zielinski T *et al.* Valve replacement for aortic stenosis with severe congestive heart failure and pulmonary hypertension. J Heart Valve Dis 1996; 5: 268–72.
[8] Cohn JN, Johnson G, Veterans Administration Cooperative Study Group: Heart failure with normal ejection fraction: The V-HeFT study. Circulation; 81 (Suppl III): III-48–III-53.
[9] Villari B, Vassalli G, Monrad ES, Chiariello M, Turina M, Hess OM. Normalization of diastolic dysfunction in aortic stenosis late after valve replacement. Circulation 1995; 91: 2353–8.

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Pulmonary arterial morphology and intravascular pressure revisited

See page 1988 for the article to which this Editorial refers

The relationship between pulmonary arterial morphology and haemodynamics is important, but still unresolved.

Borges *et al.*[1] have attempted to contribute to this question, using the correlation between intravascular ultrasound imaging and pulmonary haemodynamic data obtained by routine right heart catheterization.

Intravascular ultrasound is a relatively new method and this article is a pioneer work. Intravascular ultrasound is an invasive method and its clinical application is fully justified only when it can obtain substantially more information than other methods.

Borges *et al.*[1] conclude that intravascular ultrasound is capable of assessing vascular muscular hypertrophy and intimal proliferation. Nevertheless, no significant correlation was found between these changes and pulmonary arterial hypertension. This finding, which corroborates previous observations, is not surprising: histological changes within the pulmonary arteries are at the same time the consequences of existing pulmonary hypertension and the cause of its further evolution so that the correlation between these two variables will always be only marginal with a significant overlap. Moreover, the authors demonstrate that intravascular ultrasound cannot predict