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Aortic valve replacement in severe aortic stenosis with left ventricular dysfunction: determinants of cardiac mortality and ventricular function recovery

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Abstract

Objective: The influence of left ventricular (LV) dysfunction on survival of patients with severe aortic stenosis is poorly characterized. Few data are available about preoperative predictors of cardiac mortality and LV function recovery after aortic valve replacement of such patients. The aim of our study was to examine the outcome and the preoperative predictors of postoperative cardiac death and of LV function recovery in these patients. **Methods**: We evaluated 85 consecutive patients with severe aortic stenosis (aortic valve area < 1 cm^2) and severe depression of LV ejection fraction (EF) $< 35\%$ at cardiac catheterization. Among them, 52 underwent aortic valve replacement and they were compared to patients who were not operated on. All patients had a mean clinical follow-up of 53 months and 94% of them had a mean echocardiographic follow-up of 14 months after aortic valve replacement. **Results**: The mean baseline characteristics included: LVEF $28 \pm 6\%$, peak-to-peak transvalvular gradient 51 ± 29 mmHg, aortic valve area 0.63 ± 0.25 cm^2. Thirty-three patients did not undergo aortic valve replacement: 32 of them died within 3 years. Fifty-two patients underwent aortic valve replacement and 16 had a concomitant coronary bypass surgery. In-hospital mortality was 8%. Postoperative NYHA functional class changed from 2.84 ± 0.67 to 1.43 ± 0.44 ($P < 0.001$) and LVEF from $29 \pm 6\%$ to $43 \pm 10\%$ ($P < 0.001$). At follow-up 10 patients died of heart disease. By multivariate analysis, preoperative LV end-systolic volume index (ESVI) was the only covariate of cardiac death (LVESVI/10 ml/m^2, OR 1.3, CI 1.1–1.8, $P < 0.028$). By using a receiver operating characteristic curve, LVESVI ≤ 90 ml/m^2 was the best cut-off value (sensitivity and specificity 78%) to fit with a better survival (93% vs. 63%, $P < 0.01$) and with LVEF recovery after aortic valve replacement (EF improved by $15 \pm 10\%$ vs. $8 \pm 5\%$, $P < 0.001$). **Conclusions**: Despite LV dysfunction, aortic valve replacement appears to change drastically the natural history of severe aortic stenosis. Preoperative LV levels predict different postoperative survival rate and LVEF recovery.

Keywords: Aortic stenosis; Surgery; Survival; Ventricular function

1. Introduction

Severe aortic stenosis carries a poor prognosis when associated with congestive heart failure, the average life expectancy being of less than 2 years without surgical correction [1–3]. Accordingly, despite aortic valve replacement represents the only effective treatment, few data are available on long term survival and changes in systolic function of patients operated after the occurrence of a severe left ventricular (LV) systolic dysfunction [4–6]. Excessive LV afterload may be promptly reversed by aortic valve replacement while, in the presence of a severe contractile impairment ('valvular cardiomyopathy'), LV dysfunction may persist after aortic valve replacement [7]. Thus, differences in the mechanisms determining ventricular dysfunction can markedly influence postoperative recovery of cardiac function and patient's survival.

Predictors of postoperative survival and LV function after aortic valve replacement for severe aortic stenosis with LV dysfunction have not been clearly identified. Therefore, we hypothesized that the rate of postoperative cardiac death and LV function recovery could be related to preoperative indexes of LV dysfunction representative of prevalent

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myocardial damage rather than afterload mismatch. The aim of this study was to examine the outcome of these patients and to further assess potential preoperative predictors of postoperative cardiac death and LV function recovery.

2. Material and methods

2.1. Study population

From the hemodynamic database of our tertiary referral center, we identified all patients from February 1986 to November 2001 who had an aortic valve area <1 cm^2, LV ejection fraction (EF) $<35\%$. Patients were excluded if they were <18 years old, had more than moderate ($>2+/4$) aortic or mitral regurgitation by echocardiography or angiography, had undergone valve replacement or repair previously, or required any valve replacement in addition to aortic valve replacement during the operation. Coronary artery disease was not an exclusion factor. The medical records of 85 consecutive patients who fulfilled the entry criteria for the study were reviewed, including preoperative clinical data, 2D and Doppler transthoracic echocardiographic results, cardiac catheterization hemodynamics and coronary anatomy, and when present operative data.

2.2. Echocardiographic methods

All patients underwent comprehensive 2D and Doppler transthoracic echocardiographic assessment, 64 (75%) patients in our institution and 21 (25%) in other hospitals, <30 days before admission for cardiac catheterization. LVEF were determined by volumetric methods [8]. Aortic valve hemodynamic data were assessed using standard methods, and the aortic valve area was calculated with the continuity equation [9]. Mitral and aortic regurgitation was semiquantitated from 0 (none) to 4 + (severe) by analysis of color flow Doppler [10,11]. All postoperative studies were performed with echocardiography. Median time from surgery to postoperative echocardiography was 14.1 months.

2.3. Angiographic methods

In our center, preoperative hemodynamic assessment of adult aortic stenosis included right and left cardiac catheterization and coronary angiography in all patients. With fluid filled catheter, LV and systemic arterial pressures were recorded simultaneously. Cardiac output and index were measured at the time of cardiac catheterization, and aortic valve area was calculated from the Gorlin equation [12]. Peak-to-peak transvalvular gradient was used. Cardiac output was determined by the Fick method. All patients underwent aortography at 60° left anterior oblique view to evaluate aortic insufficiency. From left ventriculography, end-diastolic volume index (EDVI), end-systolic volume index (ESVI) and EF were calculated by the area-length method with the correction for 30° right anterior oblique projection [13]. Mitral and aortic regurgitation were semiquantitated from 0 (none) to 4 + (severe) [14]. Selective coronary angiography was performed in different angled projections. Coronary artery disease was defined as $\geq 70\%$ lumen diameter narrowing of the left main or major epicardial vessels. Multivessel coronary artery disease was defined as either left main or two or three major epicardial vessel disease. When both preoperative echocardiographic and ventriculographic data of LVESVI, LVEDVI and LVEF were available, the angiographic value were used for statistical analysis. The mean difference in LVEF between modalities was 1% ($P < 0.35$).

2.4. Surgical procedures

Fifty-two patients (61%) underwent aortic valve replacement. All surgical records were reviewed in order to determine the type and size of aortic valve prosthesis and to show whether coronary artery bypass graft surgery was performed concomitantly with aortic valve replacement. The surgical procedures performed, along with cross-clamp and cardiopulmonary bypass times, were included in database for the analysis. In-hospital death was defined as death before hospital discharge. After discharge, death were classified as either cardiac or non-cardiac. Deaths for which a non-cardiac cause was no clearly documented were considered to be cardiac related.

2.5. Statistical analysis

Data are expressed as mean value ± standard deviation for continuous variables, as numbers with percentage for categorical variables. The paired and unpaired Student test was performed to determine intragroup and intergroup differences between mean values for continuous variable, as appropriate. Categorical variables were analyzed by chi-square test or Fisher's exact test as appropriate. Multivariate logistic models were fit to identify preoperative variables related to postoperative cardiac death. Overall survival and death not due to heart disease were estimated by the use of the Kaplan–Meier method. Receiver operating characteristic (ROC) curve analysis was performed to establish the diagnostic accuracy and the value of preoperative end systolic volume index as predictor of cardiac death. Comparison of intergroup changes between preoperative and postoperative echocardiographic values used the non-parametric Mann–Whitney *U*-test. A P value <0.05 was considered significant. Data were analyzed with SPSS for Windows, release 10 (SPSS Inc., Chicago, IL).

3. Results

Baseline clinical and electrocardiographic data are outlined in Table 1. Preoperative hemodynamic and

Table 1
Baseline clinical and electrocardiographic data of the study population

	Total ($n = 85$)	AVR ($n = 52$)	No AVR ($n = 33$)	P
Age (years), mean ± S.D.	68 ± 12	69 ± 12	68 ± 13	0.79
M/F (%)	64:21 (75:25)	38:14 (73:27)	26:7 (79:21)	0.61
Ethiology, *n* (%)				0.16
Degenerative	61 (72)	34 (66)	27 (82)	
Rheumatic	22 (26)	17 (33)	5 (15)	
Congenital	2 (2)	1 (1)	1 (3)	
Comorbidities, *n* (%)				
Renal failure[a]	21 (25)	12 (23)	9 (27)	0.80
Systemic hypertension	45 (53)	23 (44)	22 (67)	0.02
Diabetes mellitus	19 (22)	9 (17)	10 (30)	0.18
Peripheral vascular disease	15 (18)	6 (11)	9 (27)	0.83
Chronic obstructive lung disease	13 (15)	7 (13)	6 (18)	0.55
Preoperative symptoms				
Dyspnea, *n* (%)	83 (97)	51 (98)	32 (97)	1
Angina, *n* (%)	33 (39)	19 (37)	14 (42)	0.65
Syncope, *n* (%)	5 (6)	1 (2)	4 (12)	0.4
NYHA class, mean ± S.D.	2.8 ± 0.7	2.8 ± 0.7	2.8 ± 0.6	0.81
Prior revascularization	8 (10)	3 (6)	5 (15)	0.3
Prior myocardial infarction	14 (17)	5 (10)	9 (27)	0.15
ECG, *n* (%)				
LV hypertrophy	79 (93)	48 (93)	31 (94)	0.57
Rhythm				0.63
Sinus	62 (73)	38 (73)	24 (73)	
Atrial fibrillation	18 (21)	12 (23)	6 (18)	
Paced	5 (6)	2 (4)	3 (9)	

AVR, aortic valve replacement; S.D., standard deviation.
[a] Creatinin level >1.3 mg/dl.

angiographic data are shown in Table 2. Fifty-two patients of 85 (61%) underwent aortic valve replacement and 31% of them (16/52) received a concomitant coronary artery by-pass graft. Thirty-three patients of 52 (39%) refused operation: all of them died within 40 months (Fig. 1). In-hospital mortality in operated patients was 8% (4/52), three patients died of cardiac death, due to low cardiac output. We failed to identify independent predictors of in-hospital mortality.

3.1. Postoperative survival

In-hospital surviving patients underwent a mean clinical follow-up of 53 months (range 18–220 months): 10 patients of 48 (21%) died of heart disease and two (4%) of other causes. Their preoperative clinical, hemodynamic and surgical data are outlined in Tables 3 and 4. By multivariate analysis, LVESVI remained as the only independent covariate of cardiac death (LVESVI/10 ml, OR 1.3, CI 1.1–1.8, $P < 0.028$). By using a ROC curve, the best cut-off value of angiographic LVESVI, for which sensitivity and specificity were equal (78%), was 92 ml/m^2 (Fig. 2). The difference in the area under the ROC curve, obtained for the cut-off value of 92 and 90 ml/m^2, did not show any statistically significant difference in the accuracy of these two threshold values. Postoperative survival curves of patients according to preoperative LVESVI cut-off value of 90 ml/m^2 are shown in Fig. 3.

3.2. Postoperative change in functional class and in LVEF

In 41 (85%) of 48 long term survivors, we observed a significant improvement of NYHA functional class (2.84 ± 0.67 vs. 1.43 ± 0.44, $P < 0.001$). LVEF was assessed with echocardiography in 45 (94%) of 48 in-hospital survivors at a mean follow-up of 14 ± 1 months without knowledge of this study. In 37 (82%) of 45 patients, LVEF increased from 29 ± 6% to 44 ± 10% ($P < 0.001$) and this variation was associated with a statistical significant reduction in LVESVI from 73 ± 21 to 60 ± 20 ($P < 0.001$) without any statistically significant change in LVEDVI (107 ± 28 vs. 104 ± 23, $P < 0.57$). The same cut-off value of preoperative LVESVI, fitted with a better survival, was also predictive

Table 2
Baseline hemodynamic and angiographic data of the study population

	Total ($n = 85$)	AVR ($n = 52$)	No AVR ($n = 33$)	P
LV end-diastolic volume index (ml/m^2), mean ± S.D.	132 ± 38	129 ± 32	137 ± 47	0.36
LV end-systolic volume index (ml/m^2), mean ± S.D.	95 ± 29	90 ± 26	98 ± 34	0.025
Aortic valve area (cm^2), mean ± S.D.	0.63 ± 0.25	0.57 ± 0.2	0.7 ± 0.3	0.028
LV ejection fraction (%), mean ± S.D.	28 ± 6	28 ± 7	28 ± 5	0.81
Peak-to-peak TVG (mmHg), mean ± S.D.	51 ± 29	59 ± 32	45 ± 21	0.03
Mean pulmonary pressure (mmHg), mean ± S.D.	32 ± 13	31 ± 12	34 ± 14	0.33
LV end-diastolic pressure (mmHg), mean ± S.D.	25 ± 9	25 ± 8	25 ± 11	0.99
Systolic arterial pressure (mmHg), mean ± S.D.	120 ± 28	117 ± 29	125 ± 26	0.23
Cardiac index (l/min per m^2), mean ± S.D.	2.3 ± 0.6	2.4 ± 0.7	2.3 ± 0.5	0.38
Coronary artery disease, *n* (%)	27 (32)	17 (33)	10 (30)	0.7

AVR, aortic valve replacement; LV, left ventricular; TVG, transvalvular gradient.

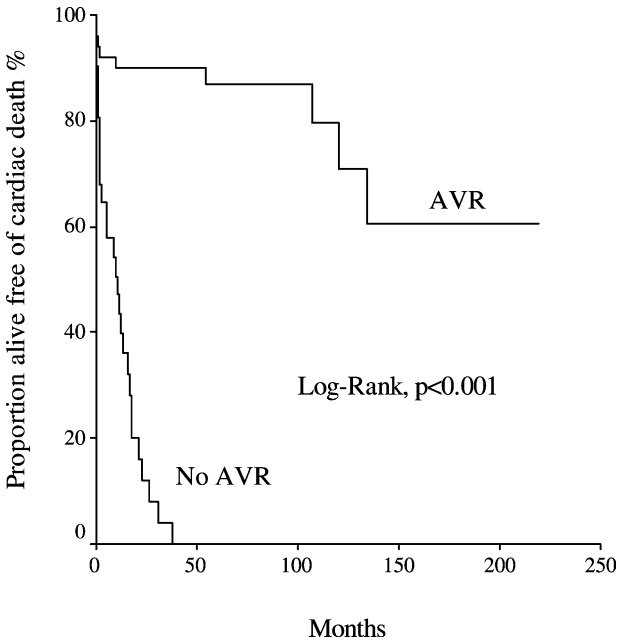


Fig. 1. Kaplan–Meier cardiac survival curves, a comparison of patients operated versus non-operated. AVR, aortic valve replacement.

Table 3
Relation of baseline clinical and electrocardiographic data to cardiac death after discharge in operated patients

	Cardiac death		*P*
	Yes ($n = 10$)	No ($n = 38$)	
Age (years), mean ± S.D.	67 ± 7	69 ± 12	0.25
M/F (%)	7:3 (70:30)	28:10 (74:26)	1
Ethiology, *n* (%)			0.64
Degenerative	5 (50)	25 (66)	
Rheumatic	4 (40)	12 (31)	
Congenital	1 (10)	1 (3)	
Comorbidities, *n* (%)			
Systemic hypertension	7 (70)	15 (39)	0.08
Diabetes mellitus	2 (20)	7 (18)	1
Peripheral vascular disease	0	6 (16)	0.33
Chronic obstructive lung disease	0	7 (18)	0.32
Preoperative symptoms, *n* (%)			
Dyspnea	10 (100)	37 (97)	1
Angina	4 (40)	14 (36)	1
Syncope	0	1 (3)	
NYHA class, mean ± S.D.	2.9 ± 0.9	2.7 ± 0.8	0.95
Prior revascularization, *n* (%)	1 (10)	2 (5)	1
Prior myocardial infarction, *n* (%)	2 (20)	3 (8)	0.34
ECG, *n* (%)			
LV hypertrophy	9 (90)	36 (95)	1
Rhythm			0.8
Sinus	7 (70)	28 (74)	
Atrial fibrillation	3 (30)	8 (21)	
Paced	0	2 (5)	

CABG, coronary artery by-pass graft; PTCA, percutaneous transluminal coronary angioplasty; S.D., standard deviation.

Table 4
Relation of baseline hemodynamic and surgical data to cardiac death after discharge in operated patients

Characteristic	Cardiac death		*P*
	Yes ($n = 10$)	No ($n = 38$)	
LV end-diastolic volume (ml/m^2), mean ± S.D.	146 ± 39	127 ± 29	0.07
LV end-systolic volume (ml/m^2), mean ± S.D.	108 ± 30	88 ± 23	0.016
Aortic valve area (cm^2), mean ± S.D.	0.65 ± 0.2	0.55 ± 0.2	0.12
LV ejection fraction (%), mean ± S.D.	25 ± 7	28 ± 7	0.10
Peak-to-peak TVG (mmHg), mean ± S.D.	49 ± 27	61 ± 32	0.29
Mean pulmonary pressure (mmHg), mean ± S.D.	32 ± 13	31 ± 12	0.76
LV end-systolic pressure (mmHg), mean ± S.D.	173 ± 31	175 ± 46	0.68
Systolic arterial pressure (mmHg), mean ± S.D.	123 ± 24	115 ± 30	0.33
Cardiac index (l/min per m^2), mean ± S.D.	2.5 ± 0.7	2.3 ± 0.7	0.63
Coronary artery disease, *n* (%)	3 (30)	14 (33)	
Simultaneous CABG, *n* (%)	3 (30)	13 (31)	
Aortic prothesis type, *n* (%)			1
Biological	6 (60)	24 (63)	
Mechanical	4 (40)	14 (37)	
Aortic prothesis size (mm), mean ± S.D.	23 ± 2	23 ± 2	0.92
Cross-clamp time (min), mean ± S.D.	77 ± 36	74 ± 32	0.89
Cardiopulmonary bypass time (min), mean ± S.D.	121 ± 51	113 ± 48	0.98

LV, left ventricular; TVG, transvalvular gradient; CABG, coronary artery by-pass graft. S.D., standard deviation.

of different LVEF recovery after aortic valve replacement. Postoperative LVEF improved by 15 ± 10% in the LVESVI ≤ 90 ml/m^2 group compared with 8 ± 5% in the LVESVI > 90 ml/m^2 group ($P < 0.001$) (Table 5).

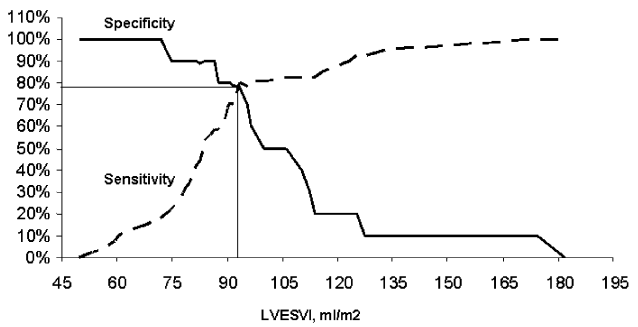
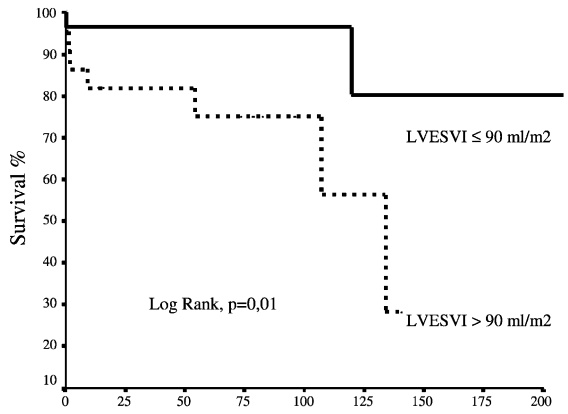


Fig. 2. The value of angiographic LVESVI for which sensitivity and specificity were equal was 92 ml/m^2. LVESVI, left ventricular end-systolic volume index.



TIME (months)		12	60	120	140
LVESVI ≤ 90 ml/m2	Cumulative events	1	1	2	2
	No. remaining	26	26	25	25
LVESVI > 90 ml/m2	Cumulative events	5	6	7	8
	No. remaining	19	18	17	16

Fig. 3. Kaplan–Meier cardiac survival curves following surgery, a comparison of patients with preoperative LVESVI ≤ 90 ml/m^2 versus >90 ml/m^2. LVESVI, left ventricular end-systolic volume index.

4. Discussion

LV dysfunction is a major prognostic indicator of the outcome in patients undergoing aortic valve replacement for aortic stenosis [6]; however, the long term outcome of patients with LV dysfunction who undergo aortic valve replacement has not yet been adequately characterized. In the present long term study spanning 20 years, we undertook also to stratify risk in population, identifying potential preoperative predictors correlated to postoperative cardiac death. Furthermore, we assessed their relation to LV function recovery after aortic valve replacement. Major findings of our study are the following: (a) patients with severe aortic stenosis and LV dysfunction died, if not operated, within few years from diagnosis; (b) despite LV dysfunction, the perioperative risk associated with aortic valve replacement in the study patients is acceptable; (c) postoperative survival at long term follow-up is gratifying; (d) preoperative LVESVI is the only covariate of postoperative survival; (e) different preoperative LVESVI levels identify different degrees and types of postoperative ventricular function recovery.

Table 5
Echocardiographic changes of LV function parameters after aortic valve replacement according to preoperative angiographic LVESVI

	Group 1 LVESVI ≤ 90 ($n = 26$)	Group 2 LVESVI > 90 ($n = 19$)	Group 3 LVESVI >100 ($n = 11$)
LVEDVI changes	−3.2 ± 19	2.1 ± 16	1.6 ± 27
LVESVI changes	−14.2 ± 18*†	−7.0 ± 16	−6.8 ± 17
LVEF changes	15.1 ± 10.3*†	8.1 ± 5.6†	6.1 ± 5.9†

LV, left ventricular; EDVI, end-diastolic volume index (ml/m^2; ESVI, end-systolic volume index (ml/m^2); EF, ejection fraction, %; *$P < 0.05$ between group 1 and 2; †$P < 0.01$ within group.

4.1. Non-operated patients

In our study all non-operated patients died within 40 months. This is in agreement with the common knowledge about the fate of severe aortic stenosis: a rapid death after the onset of heart failure. Although their profile is not completely super imposable to that of operated patients, they still have a severe aortic stenosis (aortic valve area 0.7 ± 0.3 cm^2), a virtually identical LVEF (28%) and a smaller, but still significant, transaortic gradient (45 mmHg). Since the majority of non-operated patients were hypertensive, an elevated arterial pressure at the moment of cardiac catheterization could well have decreased the gradient contributing to underestimate stenosis severity. In summary, this group seems to be very similar in many aspects to that of operated patients. Thus, we may consider the latter as control group.

4.2. Survival after aortic valve replacement and predictors of cardiac death

Operated patients have a narrower valve and less comorbidities respect to non-operated patients. They showed a low perioperative mortality of 8% and an improved survival at long term follow-up. These findings are similar to those in previous series; however, there is only the report of Connolly [6] on surgical or late outcome in a large group of patients with aortic stenosis and substantial LV dysfunction. Many of the reported series have included patients undergoing aortic valve replacement for both aortic regurgitation and aortic stenosis [15,16]. Furthermore, the other reported surgical series are largely older series, and surgical techniques, particularly regarding myocardial preservation, have advanced since that time. In the report of Connolly early mortality was 9% and related, by multivariate analysis, to the presence of coronary artery disease, while late mortality was related to the presence of coronary artery disease in addition to preoperative cardiac output. Since all patients had reduced LV function, further LVEF analysis was not related to survival. As we know from physiology, not all LVEF are created equally, so, in our study, we included LVEF, LVESVI, LVEDVI as potential covariates of outcome hypothesizing that post-operative survival and recovery of LV function might be related to preoperative indexes of LV dysfunction representative of prevalent myocardial damage rather than afterload mismatch. By multivariate analysis, we failed to find independent predictors of in-hospital mortality while LVESVI resulted as independent covariate of total cardiac mortality. In our series, coronary artery disease was less frequent than that reported by Connolly and was treated

successfully in all cases. LVESVI is a well established prognostic indicator in patients with mitral or aortic regurgitation, even if LVEF is normal. Such patients appear to represent a high risk group with an increased incidence of sudden death if not operated on, and with a considerable postoperative mortality once symptoms and/or LV systolic dysfunction developed [17]. Although LVESVI does not intervene as criteria for valve replacement in severe aortic stenosis, it remains a significant prognostic indicator, in the subgroup of patients with severe aortic stenosis and LV dysfunction.

4.3. LV function after aortic valve replacement

In our study, aortic valve replacement was associated with improved LVEF, as shown by others [3,18]. We also studied the relation between preoperative LVESVI and the extent and type of LV function recovery. After aortic valve replacement, LVEF increased markedly, due to a decrease in LVESVI, while LVEDVI remained unchanged. Although the LV volumes have been evaluated and analyzed largely by cineangiography before surgery, and by echocardiogram thereafter, we think that our study findings remain essentially true. LVEF is a non-dimensional parameter, therefore its evaluation is largely independent from the instrumentation used for measuring it. Previous studies have documented acceptable correlations between angiography and echocardiography and have confirmed reproducibility [19,20]. Indeed the mean difference in preoperative LVEF between modalities was 1% ($P < 0.37$). Thus, we are almost sure that EF is actually increased after aortic valve replacement. Problems can arise as far as evaluation of absolute volumes is concerned, because volumes are not dimensionless. However, whatever the means of measurement, changes in LVESVI are much more marked than changes in EDVI, and, since pumping ability is certainly increased, as explained before, we can reasonably hypothesize that increased EF is most probably due to a decrease in LVESVI, without affecting preload significantly. Depending on angiographic LVESVI, we found differences between pre- and postoperative LVESVI and also between pre- and postoperative LVEF. This difference lessens in the group of patients with smaller to those with larger LVESVI, determining a gradient of LV function recovery as expressed by LVEF changes. Although LVEDVI did not change significantly, all patients showed a reduction of LVESVI during follow-up, but changes were still prevalent in the group with smaller preoperative LVESVI. Since at end systole volume is essentially independent of preload and is apparently dependent only upon contractile state and loading condition, we may reasonably consider this behavior to be teleologically explained by a prevalent 'afterload mismatch' as cause of LV dysfunction in the group of patients with mild LV dilatation; in contrast patients with large preoperative LV volume showed less benefit in terms of LVEF and LVESVI changes, which may be reasonably explained by prevalent myocardial damage instead of afterload mismatch. The case of 'valvular cardiomyopathy' may be considered in the latter group [7] with a long term survival trend similar to that reported recently among community subjects in the Framingham Heart Study after the onset of heart failure [21].

In conclusion, we maintain that severe aortic stenosis rarely has clinical contraindications to surgery for cardiac reasons and, even in severely ill patients, the surgical risk is surprisingly low. In-hospital survival does not seem to depend on LV function, although late survival does. Postoperative improvement of LV function is related to preoperative LVESVI, but LVESVI improvement is less in patients with smaller to those with larger preoperative LVESVI determining a gradient of LV function recovery as expressed by LVEF changes. Evaluation of LV function before surgery should include LVESVI measurement, which gives insight both on late survival and LV function recovery after aortic valve replacement in such patients.

4.4. Limitations

This is a retrospective case-series study of consecutive patients admitted to a single center and monitored for nearly 20 years. Our patients represent a 'relatively' small sample size because severe aortic stenosis and left ventricular dysfunction is a rare association, in fact only a few larger series of such patients can be found in literature.

The two groups of patients (i.e. operated and not operated on) are not statistically matched because of the small sample size; however, most of the major prognostic variables are comparable between the two groups (i.e. age, gender, LVEF, CAD, etc.). In spite that the number of patients is small, we were able to demonstrate significant differences in mortality rate and left ventricular function recovery.

At echocardiographic follow-up we lost three out of 52 patients; this should be considered in the interpretation of results, since performing echocardiography may have been an indicator for better outcome.

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