Impaired Left Ventricular Function in Chronic Aortic Valve Disease: Survival and Function After Replacement by Björk-Shiley Prosthesis

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SUMMARY Postoperative survival and left ventricular function were studied in 128 patients who underwent isolated aortic valve replacement by the Björk-Shiley valve between 1973 and 1977. The average follow-up was 2.1 years. Patients with associated coronary artery disease or mitral valve disease were excluded. Preoperative ejection fraction ranged from 15-84%. Forty-two patients were restudied by cardiac catheterization 9.1 ± 1.1 months (mean ± SEM) after valve replacement. The hospital mortality was 11%. Preoperative type of valve lesion, functional class, cardiothoracic ratio, and ejection fraction (EF) had no significant effect on postoperative survival up to 4 years. After operation, left ventricular mass (LVMI) and peak systolic wall stress (PSWS) fell significantly, while EF and mean normalized systolic ejection rate (MNSER) increased in aortic insufficiency and in aortic insufficiency. Neither in aortic stenosis nor in aortic insufficiency was there a significant relation between preoperative ejection fraction and postoperative LVMI, EF, MNSER and PSWS. We attributed this to a marked improvement of left ventricular function in patients with preoperative impaired ventricular function. Six patients with paravalvular leak at restudy had a significantly lower EF and MNSER, and a higher PSWS than patients without leak. Patients without leak had normal EF, MNSER and PSWS when compared with 10 normal persons, but LVMI remained moderately elevated. Postoperative transprosthetic gradient was 11.9 mm Hg (range 0–64 mm Hg).

We conclude that impaired cardiac function is completely restored after aortic valve replacement by Björk-Shiley valve, if valve function is good. Patients with impaired cardiac function preoperatively did not have a poorer prognosis after operation than patients with normal function.

THE TIMING of operative intervention and the choice of valve prosthesis are based on long-term survival and the degree of hemodynamic improvement after implantation of the prosthesis. The long-term results of aortic valve replacement using the Starr-Edwards prosthesis have been reported, but comparable data on the Björk-Shiley tilting disc valve prosthesis have not been published. The tolerance of the myocardium for its hemodynamic burden is generally considered to be an important factor in the timing of operation. Determining the time of detrimental myocardial changes is a permanent challenge for surgeons and cardiologists. Severe myocardial dysfunction is often irreversible after valve replacement by Starr-Edwards prostheses. However, there are no systematic correlations available between preoperative left ventricular function (as evaluated invasively by angiographic parameters) and the late results after aortic valve replacement by Björk-Shiley prosthesis in larger series of patients. In this study, we correlate postoperative hemodynamics of patients with different degrees of preoperative myocardial dysfunction with the 4-year survival rate after aortic valve replacement by the Björk-Shiley valve.

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(more than 75% narrowing of the vessel) were not included in the study. Categorization of patients according to predominant valvular lesion showed that 48 patients had predominant aortic stenosis, 17 had aortic stenosis and significant regurgitation, and 63 had predominant aortic insufficiency (systolic transvalvular gradient less than 15 mm Hg). The 17 patients with mixed valve lesions were included in the group with predominant stenosis.

Indications for aortic valve replacement included one or more of the following: angina pectoris, syncope, congestive heart failure, and the presence or progression of cardiac enlargements on serial chest roentgenograms. Low ejection fraction was not a contraindication for valve replacement. Aortic valve replacement was performed on an emergency basis in one moribund patient.

Preoperative mitral valve disease was excluded in all patients by analysis of the left atrial pressure curve and by an additional angiographic study of the left ventricle, when needed.

Follow-up

The follow-up status of patients was determined in September 1977, either during a clinical visit or by means of a questionnaire sent to the patient. For patients who were alive at the time of the survey, the survival duration was calculated from the day of cardiac surgery. For patients who were dead at the time of survey, the primary physician or the family was contacted to identify probable cause of death. The minimum interval between surgery and the first day of the survey was 6 months. Survival rate estimates and standard errors were computed using the life table method and formulas described by Cutler and Ederer. The differences of survival rates at each yearly interval were tested by a z score.

Catheterization Studies

All patients in the study underwent right- and left-heart catheterization within 3 months before operation. Angiographic studies of the left ventricle were performed in 81 patients. Forty-two of these latter patients were restudied 9.1 ± 1.1 months after operation (range 4–36 months). Patients operated in 1975 and 1976 were requested to undergo repeat catheterization. Patients who consented underwent restudy. Thirty-six patients were reinvestigated within 12 months after operation. Only six patients who were operated before 1975 and were contacted in 1975 or 1976 consented to repeat catheterization and were restudied more than 12 months after valve replacement. Restudy included selective coronary arteriography by the Judkins technique. Control data were obtained from 10 normal subjects investigated for chest pain who had normal coronary arteries on angiography and no evidence of valvular or myocardial heart disease.

No premedication was given before catheterization. All patients received digoxin before and after the operation. Catheters (#8.5 F Brockenbrough) were positioned transeptally in the left ventricle via femoral vein puncture and retrogradely in the ascending aorta via femoral artery puncture. Pressures were recorded on an Oscillomink direct-writing system with P23Db Statham transducers before injection of contrast material. Pre- and postoperative studies were performed with the same equipment and catheter manometer systems. These systems have uniform amplitude response to 15–20 Hz, with resonant frequencies of more than 50 Hz. Left ventricular end-diastolic pressure was measured after the "a" wave. Single-plane, 35-mm cineangiograms of the left ventricle were filmed at 48 frames/sec in the 30° right anterior oblique projection after 50 ml of Urografin-76 was injected. During left ventricular injection through the Brockenbrough catheter in patients with aortic stenosis, no mitral regurgitation was seen in normally conducted sinus beats. The aortic pressure pulse and the ECG were recorded simultaneously at a paper speed of 100 mm/sec.

We performed aortic root angiography in all patients to estimate the degree of aortic regurgitation. Then, selective coronary arteriography was carried out by the Judkins technique. Left ventricular volumes and ejection fractions were determined with the area-length method. Correction factors for magnification and pin cushion distortion were obtained. The earliest well-opacified cardiac cycle was chosen for analysis, excluding extrasystolic and postextrasystolic beats. Calculated volumes were corrected to true volumes. We found a close agreement of stroke volume determined simultaneously by angiography and by the thermodynlation technique in 15 patients without heart disease: 44.8 ± 2.8 (SEM) ml/m² and 45.4 ± 2.8 ml/m² (p > 0.05), respectively. These values for normal stroke volume agree with those reported by others. The mean normalized systolic ejection rate was calculated as suggested by Peterson et al. End-diastolic wall thickness of the left ventricular free wall was measured in the right anterior oblique projection, and left ventricular muscle mass was determined according to the method of Rackley et al., with the modification introduced by Trenouth et al. Peak systolic circumferential (equal-torial) wall stress (PSWS) was calculated using the formula of Falsetti et al. and the method of Gaasch et al., despite some controversy in the literature regarding the calculation of PSWS. This method contains three assumptions: 1) that peak wall stress occurs at approximately the same time as maximum left ventricular pressure, 2) that about one-third of the stroke volume has been ejected at this point, and 3) that the change in eccentricity (L/M) with respect to the change in volume is linear. We compared the PSWS values calculated by the method of Gaasch et al. to the maximum wall stress (WS max) calculated by the frame-by-frame analysis in 12 patients with aortic valve disease and found a close correlation: WS max = 23.6 ± 0.92 PSWS (r = 0.951; SEM = 7.3).

Statistical evaluation was done using the t test for paired and unpaired observations.
Operative Techniques

The iliac artery was canulated for total cardiopulmonary bypass, for which a roller pump and a disposable oxygenator were used. The left ventricle was decompressed with a left ventricular vent. Preservation of the myocardium was attained by cardioplegic arrest of the heart. The aortic valve was replaced by a Björk-Shiley tilting disc valve prosthesis (average size A26.5 ± 0.6; range A21–A29).

Results

Postoperative Survival

The hospital mortality rate (within 30 days after operation) was 11% for the entire series. Myocardial failure caused low output syndrome in the majority of early deaths (table 1). The six operative cardiac deaths had an average functional class of 3.17 ± 0.31, and a cardiothoracic ratio of 0.57 ± 0.02. Ejection fractions were available in only two of these patients (20% and 54%). Operative survivors had an average functional class of 2.86 ± 0.06, a cardiothoracic ratio of 0.53 ± 0.01, and an ejection fraction of 52.0 ± 1.9%. No significant difference in these parameters was found (p > 0.05). Furthermore, there was no significant difference in hospital mortality rate between patients with aortic stenosis and those with aortic insufficiency (p > 0.05) (fig. 1A). At follow-up evaluation, 7% of patients who had survived the first 30 postoperative days later died. As summarized in table 1, late death was sudden in three patients and cerebrovascular accident occurred in two other patients. No late death was due to congestive heart failure. Survival curves were constructed by dichotomization of the entire series on the basis of three different variables (fig. 1). The 4-year survival rate was 80% for patients with aortic stenosis and 78% for patients with aortic insufficiency (fig. 1A). There was no significant difference between the groups. Comparison of patients in functional classes II and III with patients in class IV also showed no significant difference (fig. 1B). There was no significant difference in survival between patients with a cardiothoracic ratio ≤ 0.54 and patients with a ratio ≥ 0.55 (fig. 1C).

Figure 2 is a scattergram relating preoperative ejection fraction to postoperative survival up to 4 years. The lower normal limit for the ejection fraction (mean value of controls minus 2 sd) was 55% and is shown by the interrupted vertical line. There was no significant difference in mortality between the group with impaired left ventricular function (ejection fraction < 55%) and the group with normal function (chi square = 1.39; p > 0.05).

Changes After Valve Replacement

Clinical data and hemodynamic measurements could be compared in 42 patients before and after valve replacement (table 2). At comparable heart rates (p > 0.05) before and after operation the following changes were noted: 1) Functional class and cardiothoracic ratio improved significantly in aortic stenosis and in aortic insufficiency. 2) Left ventricular systolic pressure, left ventricular end-diastolic pressure and mean left atrial pressure fell significantly in both groups, while diastolic aortic pressure increased (table 2). 3) Left ventricular volume and mass decreased in aortic stenosis and aortic insufficiency after operation (fig. 3). This regression of hypertrophy was associated with a significant augmentation of ejection fraction from 54.9 ± 4.6 to 70.7 ± 1.9% in aortic stenosis and from 53.0 ± 2.8 to 61.0 ± 2.1% in aortic insufficiency. 4) Mean normalized systolic ejection rate increased in both groups and PSWS decreased significantly (fig. 3). 5) After operation, ejection fraction and mean normalized systolic ejection rate were lower in aortic insufficiency than in aortic stenosis (p < 0.01) at comparable levels of PSWS (p > 0.05).

Correlation Between Preoperative Ventricular Function and Postoperative Hemodynamic Result

The relation between preoperative ventricular function, as estimated by the ejection fraction, and postoperative hemodynamics is shown in figure 4. There was no significant relation between preoperative ejection fraction and postoperative left ventricular mass, postoperative ejection fraction, postoperative mean normalized systolic ejection rate, and postoperative PSWS in aortic stenosis (figs. 4A, C, E and G) or in aortic insufficiency (figs. 4B, D, F and H). Patients with paravalvular leak were not included in fig. 4. Patients with an impaired preoperative ejection fraction had a more marked improvement in pump function than patients with a normal ejection fraction (fig. 3). After operation, we found no significant relation of parameters to preoperative ejection fraction.

Effect of Paravalvular Leak on Postoperative Ventricular Function

Significant aortic regurgitation was found postoperatively in six patients. Two patients had a mod-
erate ventricular septal defect just below the aortic prosthesis, and one developed anterior asynergy due to complete occlusion of the anterior descending branch of the left coronary artery. Before operation, the coronary arteries had been normal in this patient. These seven patients constituted the group with aortic valve replacement with paravalvular leakage. We compared postoperative hemodynamic data of this group with data of 10 control subjects and with data of patients without leakage after valve replacement (table 3; fig. 5). Patients with postoperative leak have a significantly lower ejection fraction and mean normalized systolic ejection rate as well as a higher PSWS than patients without leakage (fig. 5). Patients without leak have a normal ejection fraction, mean normalized systolic ejection rate, and PSWS when compared with controls, although some hypertrophy persisted, probably caused by a moderate but significant elevation of peak systolic left ventricular pressure (table 3).

The patients with Björk-Shiley aortic prosthesis had an average peak systolic transprosthetic gradient of
11.9 mm Hg (range 0–64 mm Hg). The highest gradient was measured in a patient with a small A-21 prosthesis.

**Discussion**

The postoperative survival rate of patients with preoperative impaired ventricular function was not inferior to that of patients with normal ventricular function. This correlated well with the hemodynamic result: Aortic valve replacement produced a considerable reduction of hypertrophy and a significant increase of cardiac function after normalization of intraventricular and aortic pressures. Patients with preoperative impaired ventricular function showed a drastic improvement postoperatively and reached a normal level, while patients with normal ventricular function showed little change. Thus, left ventricular dysfunction, which is a well-known determinant of natural survival in chronic aortic valve disease, is corrected and improved after successful aortic valve replacement.

The operative mortality was 11% for this series. When functional class and cardiothoracic ratio of patients who died because of myocardial failure were compared with the respective data of operative survivors, no significant difference was detected. Unfortunately, ejection fraction was not measured preoperatively in all patients who died early postoperatively from myocardial failure. Nevertheless, no clear-cut relation between preoperative ventricular impairment and postoperative myocardial failure could be established by the data presented.

The postoperative survival up to 4 years could not be predicted from clinical parameters: diagnosis, functional class and cardiothoracic ratio. Furthermore, no predictive value of the ejection fraction could be confirmed in our patients. These results are in accordance with those of Hirshfeld et al. and Lee et al., but differ from those of others who showed a predictive value of functional class and cardiothoracic ratio. Several factors may be responsible for the different observations: 1) exclusion of patients with coronary artery disease, 2) duration of follow-up period, and 3) type of prosthesis used. It has been shown that a very important predictive variable for postoperative survival is the presence of associated coronary artery disease in aortic valve disease. Only a single study has evaluated the effect of coronary artery disease on late survival and described a poorer survival when coronary artery disease was present than when it was absent (3-year survival rate 60 vs 85%).

In the present study we excluded all patients with significant coronary artery disease. Only limited conclusions can be drawn from our results since the observation period was relatively short and it is necessary to continue the follow-up. However, in our follow-up to 4 years, no significant difference was detected.
years, we found no patient who died from myocardial failure. This unexpected result is further confirmed and explained by the hemodynamic postoperative findings, which show normalization of cardiac function even in patients with impaired myocardial function preoperatively. Since the Björk-Shiley prosthesis obviously has a low transprosthetic gradient compared with other mechanical valves and porcine heterograft valves, part of our results may be attributable to the quality of the prosthesis used. The predictive value of the preoperative ejection fraction as an invasive parameter was previously tested in two studies. Only very few and incomplete observations are reported in these communications — the first study did not mention the type of prosthesis used and the other gave information about a collection of patients after aortic, mitral and double valve replacement. The latter report, although dealing with a variety of valves and therefore differing from the present study, revealed no significant difference in preoperative ejection fraction between late postoperative survivors and late postoperative deaths.

Left ventricular function was evaluated by ejection phase parameters and peak systolic wall stress was determined as an estimate of afterload. We calculated PSWS using the method of Gaasch et al. Some assumptions that are not entirely correct must be made when using this method. For example, peak
Aortic valve replacement produced a significant increase in ejection fraction and mean normalized systolic ejection rate and a fall in PSWS. Since acute increases of afterload are known to produce an acute depression of ejection phase parameters, these postoperative changes could be attributable to reduced afterload without any change of cardiac inotropic state instead of improved myocardial function. It has been shown that an acute experimental augmentation of afterload (supravalvular aortic constriction) caused a reduction of percent shortening of minor diameter as well as of shortening velocity and an increase of PSWS. During the chronic development of myocardial hypertrophy, however, a stepwise restoration of percent shortening and shortening velocity was found while wall stress returned to normal. This adaptive mechanism was successful when left ventricular mass was augmented to 136% of normal. Our preoperative hemodynamic data, measured during the resting state, showed an increase in PSWS (123% of normal in aortic stenosis and 120% in aortic insufficiency) and a decrease in mean normalized systolic ejection rate (74% of normal in aortic stenosis and 69% in aortic insufficiency). This imbalance was found when left ventricular mass was 250% of the normal level in aortic stenosis and 253% in aortic insufficiency. Thus, a moderate imbalance between ejection phase parameter and wall stress became evident only when hypertrophy was most advanced. This suggests that the adaptive mechanism of hypertrophy that has been shown in experimental animals must have been effective in our patients as well, and began to fail when hypertrophy exceeded a certain level.

We have reported hemodynamic studies from this laboratory which were carried out in patients with aortic stenosis who had normal ejection fraction, mean normalized systolic ejection rate and normal PSWS at rest. Left ventricular mass was augmented to 163% of normal in this group. A pharmacologic stress was applied to these patients by infusion of the positive inotropic drug, isoproterenol, and a stress ventriculogram was obtained. During infusion (associated with an increase of heart rate to 135 beats/min), a significant depression of ejection phase parameters and an increase in PSWS was found. In a subgroup of these patients, the lactate metabolism of the heart was evaluated at rest and during isoproterenol infusion, and we found a highly abnormal lactate production in patients with aortic stenosis during isoproterenol, but not in control subjects. From these results we concluded that the imbalance of shortening velocity and wall stress during isoproterenol stress represents an acute depression of the previously normal inotropic state and is caused by myocardial ischemia. Experimental data in dogs with supravalvular aortic stenosis support this concept. Thus, evidence exists that the imbalance between ejection phase parameters and PSWS is a sensitive indicator for depression of the inotropic state of the myocardium. Restoration of this imbalance after valve replacement represents improvement of the inotropic state.

Malfunction of the prosthesis has impeded complete restoration of myocardial function in some of our patients. We found a significantly higher end-diastolic volume and a significantly lower ejection

**Figure 3.** Preoperative (BEFORE) and postoperative (AFTER) hemodynamic parameters in aortic stenosis and in aortic insufficiency. LV mass = left ventricular mass; EF = ejection fraction; MNSER = mean normalized systolic ejection rate; PSWS = peak systolic wall stress. Interrupted lines denote cases with postoperative leak.

wall stress does not always occur exactly when left ventricular pressure reaches its maximum. However, we found a good correlation between PSWS calculated according to Gaasch et al. and data determined using the frame-by-frame analysis within the same group of patients. In addition, our values for normal patients are comparable to those reported by others.
fraction and mean normalized systolic ejection rate in patients with leakage than in controls. Patients without leakage had normal ejection phase parameters and normal PSWS, indicating normal ventricular function after operation. Therefore, incomplete recovery after aortic valve replacement often may be more related to problems of the prosthesis than to problems of the myocardium. Earlier studies that evaluated left ventricular function invasively after aortic valve replacement found less convincing results in patients with aortic insufficiency.\textsuperscript{54, 55} Our data also showed lower postoperative ejection fraction and mean normalized systolic ejection rate in aortic insufficiency than in aortic stenosis. Valve replacement in pure aortic insufficiency removes volume overload but often imposes some pressure load on an enlarged chamber by changing the type of overload. This situation does not occur in aortic stenosis and may account for the difference observed.

In summary, operative mortality in isolated aortic valve replacement by the Björk-Shiley prosthesis was considerable in the series presented, but was not affected by preoperative type of valve lesion and preoperative function as estimated from functional class and cardiothoracic ratio. The 4-year survival rate was not different between patients with impaired left ventricular function (as estimated by ejection fraction) and patients with normal function preoperatively. No late death was due to myocardial failure; three late deaths were sudden and unexpected. Impaired cardiac function normalized completely after valve replacement by the Björk-Shiley valve, provided there was no postoperative leak. This may explain the absence of cardiac failure in operative survivors.
Figure 5. Postoperative hemodynamic parameters are compared with controls. After aortic valve replacement (AVR), patients are divided according to postoperative result into those with leakage and those without leakage. Patients without leakage have normal level of ejection phase parameters and normal peak systolic wall stress (PSWS) after operation, but patients with leakage do not. LV = left ventricular; EF = ejection fraction; MNSER = mean normalized systolic ejection rate.
Table 3. Hemodynamic Data of Patients with Björk-Shiley Valve With and Without Leakage

<table>
<thead>
<tr>
<th></th>
<th>Controls (n = 10)</th>
<th>Aortic valve replacement without leakage (n = 35)</th>
<th>p value</th>
<th>Aortic valve replacement with leakage (n = 7)</th>
<th>p value</th>
<th>Aortic valve replacement without leakage (n = 35)</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart rate (beats/min)</td>
<td>75.4 ± 3.6</td>
<td>81.7 ± 3.8</td>
<td>NS</td>
<td>80.3 ± 3.6</td>
<td>NS</td>
<td>81.7 ± 3.8</td>
<td>NS</td>
</tr>
<tr>
<td>Left ventricular systolic pressure (mm Hg)</td>
<td>123.2 ± 4.6</td>
<td>143.5 ± 5.8</td>
<td>&lt;0.01</td>
<td>143.7 ± 6.1</td>
<td>&lt;0.05</td>
<td>143.5 ± 5.8</td>
<td>NS</td>
</tr>
<tr>
<td>Aortic systolic pressure (mm Hg)</td>
<td>123.2 ± 4.6</td>
<td>131.9 ± 5.7</td>
<td>NS</td>
<td>130.6 ± 7.9</td>
<td>NS</td>
<td>131.9 ± 5.7</td>
<td>NS</td>
</tr>
<tr>
<td>Aortic diastolic pressure (mm Hg)</td>
<td>71.8 ± 3.4</td>
<td>78.4 ± 2.5</td>
<td>&lt;0.05</td>
<td>77.9 ± 2.6</td>
<td>NS</td>
<td>78.4 ± 2.5</td>
<td>NS</td>
</tr>
<tr>
<td>Left ventricular end-diastolic pressure (mm Hg)</td>
<td>11.2 ± 1.2</td>
<td>11.8 ± 1.1</td>
<td>NS</td>
<td>13.9 ± 1.2</td>
<td>NS</td>
<td>11.8 ± 1.1</td>
<td>NS</td>
</tr>
<tr>
<td>Mean left atrial pressure (mm Hg)</td>
<td>8.8 ± 1</td>
<td>8.3 ± 0.6</td>
<td>NS</td>
<td>10.9 ± 1.2</td>
<td>NS</td>
<td>8.3 ± 0.6</td>
<td>NS</td>
</tr>
<tr>
<td>End-diastolic volume (ml/m²)</td>
<td>76.7 ± 6.6</td>
<td>88.4 ± 4.8</td>
<td>NS</td>
<td>181.2 ± 42.9</td>
<td>&lt;0.002</td>
<td>88.4 ± 4.8</td>
<td>NS</td>
</tr>
<tr>
<td>Left ventricular mass (g/m²)</td>
<td>72.6 ± 4.6</td>
<td>117.1 ± 4.3</td>
<td>&lt;0.001</td>
<td>188.5 ± 47.5</td>
<td>&lt;0.002</td>
<td>117.1 ± 4.3</td>
<td>NS</td>
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<tr>
<td>Ejection fraction (%)</td>
<td>68.7 ± 2.2</td>
<td>67.4 ± 1.4</td>
<td>NS</td>
<td>52.4 ± 4.8</td>
<td>&lt;0.01</td>
<td>67.4 ± 1.4</td>
<td>&lt;0.01</td>
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<tr>
<td>Mean normalized systolic ejection rate (end-diastolic volumes/sec)</td>
<td>2.36 ± 0.09</td>
<td>2.46 ± 0.07</td>
<td>NS</td>
<td>1.74 ± 0.17</td>
<td>&lt;0.01</td>
<td>2.46 ± 0.07</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Peak systolic wall stress (dyn-10⁴/cm²)</td>
<td>292.8 ± 18.7</td>
<td>261.2 ± 8.9</td>
<td>NS</td>
<td>332.2 ± 21.6</td>
<td>NS</td>
<td>261.2 ± 8.9</td>
<td>&lt;0.01</td>
</tr>
</tbody>
</table>

Values are given as mean ± SEM.

References

46. Cohn PF, Gorlin R: Dynamic ventriculography and the role of the ejection fraction. Am J Cardiol 36: 529, 1975
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F Schwarz, W Flameng, F Langebartels, M Sesto, P Walter and M Schlepper

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