Surgical repair of postinfarction ventricular septal rupture: Risk factors of early and late death

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Objective: The aim of the study was to identify risk factors of early and late death after surgical repair of postinfarction ventricular septal rupture.

Methods: During a 25-year period, from May 1981 to August 2006, 102 patients underwent repair of postinfarction ventricular septal rupture. Data were collected on clinical, angiographic, and echocardiographic findings; operative procedures; early morbidity; and survival time. Univariable and multivariable analyses were performed to identify risk factors of 30-day mortality and total mortality.

Results: Thirty-day mortality was 33% altogether and decreased from 45% in the first half to 21% in the second half of the period ($P = .01$). Follow-up was a mean of 5.2 ± 6.2 years and a median of 2.9 years (range, 0–26.3 years). Five- and 10-year cumulative survival was 50% and 32%, respectively. Shock at surgical intervention and incomplete coronary revascularization were strong and independent risk factors of both 30-day mortality and poor long-term survival.

Conclusions: Early outcome after repair of ventricular septal rupture improved significantly during time, with 30-day mortality being 21% in the last decade. Five- and 10-year cumulative survival was 50% and 32%, respectively. Shock at surgical intervention and incomplete coronary revascularization were strong and independent predictors of poor early and late survival.

Ventricular septal rupture (VSR) is a fatal complication after acute myocardial infarction (AMI) and represents a major surgical challenge. Thrombolytic therapy and acute percutaneous coronary intervention (PCI) have led to a 5- to 10-fold reduction in the incidence of VSR,^{1–4} but the operative risk remains high. Because of advanced age, heart transplantation and assist devices as a bridge to transplantation are seldom proposed. Although prognosis is extremely poor in medically treated patients,^{1,5,6} it is important to identify patients with excessive risk in whom surgical intervention can be avoided. Several predictors of poor outcome have been identified, including large AMI, preoperative shock, right ventricular infarction, posterior VSR, and large left-to-right shunt. In this single-center, retrospective study of 102 consecutive patients undergoing VSR repair, we describe predictors of 30-day mortality and long-term survival.

MATERIALS AND METHODS

This retrospective study was approved by the institutional review board.

Patients

During a 25-year period, from May 1981 to August 2006, 106 consecutive patients from the south of Norway were referred to our hospital

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with postinfarction VSR. Four patients died before reaching surgical intervention, and therefore 102 patients underwent VSR repair and were included in the study for follow-up. The preoperative cardiologic assessment was done in our institution and included 2-dimensional Doppler echocardiographic analysis and selective coronary angiographic analysis for all patients. Details of revascularization were collected from the operative records and correlated to the angiograms, and each patient was categorized into “yes” or “no” categories for the 2 variables of (1) complete global coronary revascularization and (2) revascularization of the culprit artery (infarct-related artery). Preoperative and operative risk factors of 30-day mortality are summarized in Table 1. There were 76 men and 26 women with a mean age of 67 ± 8 years. Ninety patients had not experienced AMI previously, and 12 patients had a history of 1 previous AMI. Thrombolysis was performed in 19 cases, and acute PCI was performed in 5 cases. Time from AMI to diagnosis of VSR was a median of 5 days and did not change throughout the study period. Time from VSR to surgical intervention decreased from a median of 3 days in the first half to 1 day in the second half of the study period ($P = .02$). Shock at surgical intervention was present in 14 patients and was strictly defined as hypoperfusion leading to acute oliguria/anuria or acute increase in creatinine value to greater than 200 $\mu\text{mol/L}$. None of the patients had preoperative chronic renal failure that could confound this definition. Posterior VSR was slightly more common than anterior VSR. Shunt size was estimated in 78 cases by means of echocardiographic analysis or right cardiac catheterization with oximetry, and two thirds of them had Qp/Qs ratios of greater than 2.5. All patients had significant coronary lesions, and for the whole cohort, the frequency of 1-, 2-, and 3-vessel disease was approximately equal. There was a tendency toward more 1-vessel disease in anterior VSR and more 3-vessel disease in posterior VSR ($P = .10$). The left anterior descending coronary artery was the infarct-related artery in all patients with anterior VSR. The right coronary artery system was the infarct-related artery in all patients with posterior VSR, except in 1 patient with occlusion of a dominant circumflex.

Surgical Procedure

The operation was performed through a median sternotomy by using cardiopulmonary bypass (CPB) and moderate systemic hypothermia. Antegrade crystalloid solution (St Thomas II) was infused for cardioplegic arrest in all cases except 2, in which the repair was performed on the beating heart. The VSR was approached through the infarction area of the left

Abbreviations and Acronyms

AMI	= acute myocardial infarction
ASAT	= aspartate aminotransferase
CABG	= coronary artery bypass grafting
CPB	= cardiopulmonary bypass
IABP	= intra-aortic balloon pump
PCI	= percutaneous coronary intervention
VSR	= ventricular septal rupture

ventricle and was repaired with the traditional technique of Daggett and associates⁷ or the infarct-exclusion repair of David and colleagues.⁸ Daggett repair (n = 67) dominated in the first half and David repair (n = 35) dominated in the second half of the series. In Daggett repair the VSR was patched (Dacron or polytetrafluoroethylene patches) in all cases except 1, in which the defect was directly closed with buttressed interrupted sutures. The ventriculotomy was closed with buttressed sutures or, in a few cases, resected myocardium was replaced with prosthetic material. In David repair the infarcted myocardium and VSR were excluded with a large patch of bovine pericardium and in a few cases supplemented with a second patch directly on the VSR. The ventriculotomy was closed without infarctectomy by using buttressed sutures. Glue was not used. No valve procedure was performed. Coronary artery bypass grafting (CABG) was performed when significant coronary artery disease was present and the vessel periphery was deemed suitable for revascularization. The left internal thoracic artery and saphenous vein were used for grafts. Distal anastomoses were performed on the cardioplegic heart, and finally, proximal anastomoses were performed on a beating heart. Sixty-six patients underwent revascularization: CABG in 61 patients, acute PCI of the culprit artery in 3 patients with 1-vessel disease, and acute PCI of the culprit artery followed by CABG in 2 patients with 3-vessel disease. The mean number of distal anastomoses was 1.8. The infarct-related coronary artery was revascularized in 24 patients (left anterior descending coronary artery in 11 patients and right coronary artery in 13

patients). Of 64 patients with disease in remote coronary territories, 39 underwent complete remote revascularization, 17 had incomplete remote revascularization, and 8 had no remote revascularization. Accordingly, 25 patients left the operating room with remote myocardium at ischemic risk as a consequence of incomplete or no revascularization of non-infarct-related arteries. Altogether, 22 patients underwent complete coronary revascularization (culprit artery plus remote arteries). An intra-aortic balloon pump (IABP) was used in 91 patients and was started preoperatively (>24 hours) in 21 patients and perioperatively in 70 patients. IABP was used for a median of 4 days (range, 0–12 days) after the operation.

Study Design, Data Collection, and Statistical Analysis

The study is a cohort analysis of 102 consecutive patients undergoing operations for postinfarction VSR in our hospital during a 25-years period, from May 1981 to August 2006. This dynamic cohort had different entry times (date of operation), and the common closing date was July 1, 2008. Data were collected from patient records, and survival data were entirely based on information from the Norwegian Death Registry. Follow-up was 100% complete and was a mean of 5.2 ± 6.2 years and a median of 2.9 years (range, 0–26.3 years). Continuous data are presented as the mean $\pm$ standard deviation or median (range), and categorical data are frequencies or fractions of patients. End points were 30-day mortality and total mortality (all deaths including 30-day deaths).

Univariable analysis of 30-day mortality was performed with 2×2 tables and χ^2 or Fisher's exact tests for categorical data and the 2-tailed *t* test or the Mann–Whitney test for continuous data. Survival curves were plotted according to the Kaplan–Meier method, and differences between curves were pinpointed by using the Breslow test and the log-rank test.⁹

Multivariable analysis was performed for variables that demonstrated statistical significance ($P < .05$) or marginal significance ($P < .2$) in the univariable analysis or that were considered clinically or pathophysiologically important. Accordingly, the following variables were included in the logistic regression analysis and the Cox model: age, sex, diabetes mellitus,

TABLE 1. Patient data and risk factors of 30-day mortality

Variable	All (n = 102)	Survivors (n = 68)	Nonsurvivors (n = 34)	P value
Age (y)	67 $\pm$ 8	66 $\pm$ 8	68 $\pm$ 9	>.2
Male/female sex	76/26	49/19	27/7	>.2
BMI	24.9 $\pm$ 2.7	25.0 $\pm$ 2.7	24.7 $\pm$ 2.8	>.2
Diabetes mellitus	12	8	4	>.2
Previous AMI	12	7	5	>.2
Thrombolysis/acute PCI	24	17	7	>.2
Left main stenosis	9	7	2	>.2
1/2/3-Vessel disease	38/30/34	25/18/25	13/12/9	>.2
Anterior/posterior VSR	47/55	34/34	13/21	>.2
AMI to VSR (d)	5 (1–27)	5 (1–27)	2 (1–14)	.20
VSR to surgical intervention (d)	2 (0–60)	2 (0–60)	1 (0–30)	.09
ASAT _{max} (U/L)*	470 $\pm$ 355	349 $\pm$ 190	665 $\pm$ 468	.01
Shunt size >2.5/<2.5†	50/28	30/24	20/4	.02
Shock at surgical intervention	14	5	9	.01
Aortic crossclamp time (min)	64 $\pm$ 31	60 $\pm$ 29	71 $\pm$ 33	>.2
CPB (min)	116 $\pm$ 45	109 $\pm$ 40	130 $\pm$ 53	.04
Coronary revascularization	66	47	19	.19
Complete revascularization	22	18	4	.09
Culprit revascularization‡	24	20	4	.05

Values are presented as means $\pm$ SD or medians (ranges) or the number of patients or fractions of patients. BMI, Body mass index; AMI, acute myocardial infarction; PCI, percutaneous coronary intervention; VSR, ventricular septal rupture; ASAT, aspartate aminotransferase; CPB, cardiopulmonary bypass. *Maximal aspartate aminotransferase after acute myocardial infarction measured in 42 patients. †Qp/Qs ratio measured in 78 patients. ‡Revascularization of the infarct-related artery.

TABLE 2. Independent risk factors of 30-day mortality by logistic regression analysis

Variable	Level	β	SE of β	OR	95% CI	P value*
<i>Global revascularization</i>						
Shock at surgical intervention	Yes/no	1.57	0.66	4.81	1.31–17.60	.018
Complete revascularization	Yes/no	−1.72	0.70	0.18	0.05–0.70	.014
CPB time	Continuous ($\Delta = 10$ min)	0.16	0.06	1.17	1.04–1.32	.005
Constant		−2.46	0.67	0.09		<.001
<i>Culprit artery</i>						
Shock at surgical intervention	Yes/no	1.55	0.67	4.70	1.28–17.3	.02
Culprit revascularization†	Yes/no	−1.81	0.69	0.16	0.04–0.63	.009
CPB time	Continuous ($\Delta = 10$ min)	0.16	0.05	1.17	1.06–1.29	.004
Constant		2.43	0.67	0.09		<.001

β , Regression coefficient; OR, odds ratio; 95% CI, 95% confidence interval of odds ratio; CPB, cardiopulmonary bypass. *Logistic regression analysis with manual backward elimination. †Revascularization of the infarct-related artery.

left main stenosis, 3-vessel disease, location of VSR, time from AMI to VSR, time from VSR to surgical intervention, shock at surgical intervention, long CPB time, and complete coronary revascularization. Complete coronary revascularization was assessed in 2 ways: (1) complete global revascularization and (2) revascularization of the culprit artery. Because they were internally correlated, they could not be included in the same multivariable model. Aspartate aminotransferase (ASAT) and preoperative shunt values were measured in 42 and 78 patients, respectively. Because of the possibility of selection bias and loss of power, they were not included in the multivariable model. Surgical repair method was not included as a risk factor. This is addressed separately in an upcoming report with a more causative approach by using the strategy of an exposed (David) versus nonexposed (Daggett) cohort on the end-point mortality. Logistic regression was used to identify independent risk factors of 30-day mortality, and Cox proportional hazards regression was used to identify independent risk factors of total mortality.¹⁰ Manual backward elimination of variables was performed based on the following criteria: clinical or pathophysiologic importance, correlation matrix between the variables, and statistical significance of the Wald test. SPSS for Windows (version 13.0; SPSS, Inc, Chicago, Ill) and OpenEpi (version 2.2.1) were used for statistical calculations. The reporting of results is performed in accordance with the Strengthening of Reporting of Observational Studies in Epidemiology guidelines for cohort studies.^{11,12}

RESULTS

Early Morbidity

Reoperation for wound bleeding was performed in 14 patients, and 5 patients underwent reoperation for deep sternal infection. The most common postoperative organ malfunctions were renal and respiratory failure. Four patients with low output required mechanical circulatory assistance beyond IABP to exit the operating room; 3 had univentricular assist with centrifugal pumps (left ventricular assist, 1; right ventricular assist, 2), and 1 had venoarterial cardiac bypass with extracorporeal membrane oxygenation. Two of them died early (on the operating table and at day 13, respectively), and 2 were late survivors (4 and 6 years, respectively). Examination for postoperative shunt was performed by means of echocardiography or oxymetry in 76 patients within the first 30 days after surgical intervention. One third of them had a residual shunt or reshunt, and 12 patients underwent reoperation, the majority within 30 days after the primary procedure.

Thirty-day Mortality

Thirty-day mortality was 33% for the whole cohort and decreased from 45% in the first half to 21% in the second half of the study period ($P = .01$). Significant risk factors of 30-day mortality by means of univariable analysis (Table 1) were large AMI (high ASAT levels), large preoperative shunt ($Q_p/Q_s > 2.5$), shock at surgical intervention, long CPB time, and no revascularization of the culprit artery. Independent risk factors of 30-day mortality were shock at surgical intervention, long CPB time, and incomplete coronary revascularization (Table 2). Both incomplete global revascularization and no revascularization of the culprit artery were independent predictors of 30-day mortality. Anterior or posterior location of the VSR did not influence early outcome.

Long-term Survival

Five- and 10-year cumulative survival for all patients was 50% and 32%, respectively (Figure 1). Significant risk factors of total mortality by means of univariable analysis were advanced age, large preoperative shunt ($Q_p/Q_s > 2.5$), shock at surgical intervention, and long CPB time. Independent risk factors of total mortality were advanced age, shock at surgical intervention, long CPB time, and incomplete coronary revascularization (Table 3). Both incomplete global

FIGURE 1. Cumulative survival after repair of postinfarction ventricular septal regurgitation (VSR) in 102 patients.

TABLE 3. Independent risk factors of total mortality by means of Cox regression analysis

Variable	Level	β	SE of β	RR	95% CI	P value*
<i>Global revascularization</i>						
Age	Continuous ($\Delta = 10$ y)	0.53	0.18	1.70	1.19–2.42	.002
Shock at surgical intervention	Yes/no	0.95	0.36	2.60	1.30–5.20	.007
Complete revascularization	Yes/no	–0.84	0.37	0.43	0.21–0.89	.023
CPB time	Continuous ($\Delta = 10$ min)	0.08	0.03	1.08	1.02–1.15	.002
<i>Culprit artery</i>						
Age	Continuous ($\Delta = 10$ y)	0.55	0.18	1.73	1.22–2.54	.003
Shock at surgical intervention	Yes/no	0.93	0.35	2.54	1.27–5.08	.009
Culprit revascularization†	Yes/no	–0.75	0.34	0.47	0.24–0.93	.029
CPB time	Continuous ($\Delta = 10$ min)	0.08	0.03	1.08	1.02–1.15	.002

β , Regression coefficient; RR, Relative risk; 95% CI, 95% confidence interval of relative risk; CPB, cardiopulmonary bypass. *Cox proportional hazards regression analysis with manual backward elimination. †Revascularization of the infarct-related artery.

revascularization and no revascularization of the culprit artery were independent predictors of poor long-term survival. Anterior or posterior location of the VSR did not influence late outcome.

DISCUSSION

In the prethrombolytic era, 1% to 2% of patients with AMI had VSR. After the introduction of thrombolytic therapy, there has been a 5- to 10-fold reduction in the incidence of postinfarction VSR, and in the GUSTO-I trial of 41021 patients with ST-segment elevation infarction, 0.2% of those treated with thrombolysis had VSR.¹ The same reduction has been demonstrated for patients undergoing primary PCI during AMI.^{2–4} In contrast, we found no reduction in the number of patients admitted with VSR throughout the study period, but thrombolysis or acute PCI was performed in only 24 of 102 patients. VSR remains a surgical challenge, with significant morbidity and mortality. Most studies from the last 2 decades report 30% to 60% early mortality (30-day mortality or death during same hospital stay)^{13–15} or 30-day mortality,^{1,6,16–21} although early mortality has been as low as 23%²² and as high as 81%⁵ in recent reports. However, comparison between reports is unreliable because the natural course of AMI might have changed, and the recruitment of patients for surgical intervention differs significantly. The present study showed a significant reduction in 30-day mortality, from 45% in the first half to 21% in the second half of the study period. The 5- and 10-year cumulative survival, 30-day mortality included, was 50% and 32%, respectively. This is in accordance with other recent studies that report 5-year cumulative survival in the range 40% to 45% and 10-year survival in the range of 25% to 40%.^{13,19,20,23,24} Five- and 10-year survival is usually 10% to 20% higher if patients who die within 30 days are excluded from the analysis.^{14,21,25}

After the introduction of thrombolytic therapy, the average time between AMI and VSR has been reported to decrease from 5 to 7 days to close to 1 day.^{1,5,26–28} In accordance with other recent reports,^{2,29} such a shift was

not supported by our study. Time from AMI to VSR was constant (median, 5 days) for the entire study period and for the 24 patients undergoing thrombolysis or acute PCI. In accordance with previous studies,^{17,21} we found that time from AMI to VSR did not predict early or late outcome, although one other study has reported that shorter time from AMI to VSR increased the early mortality.¹⁹

Previously, surgical intervention was often delayed for several weeks while waiting for stable hemodynamics and for myocardial fibrosis to occur. This will certainly improve outcome because the surgical procedure is easier and the sickest patients die before surgical intervention. Currently, the majority of surgeons follow a more aggressive approach, with immediate start of IABP and urgent VSR repair, usually within 24 hours after diagnosis. Accordingly, patients now undergo operations closer in time to the AMI, when the hemodynamic state is poorer and the myocardium is more fragile for the repair. Time from diagnosis of VSR to the operation decreased significantly during the study period, but the shift to earlier surgical intervention did not increase early or late mortality. This does not necessarily exclude such a correlation but probably reflects the time window for surgical intervention in our study. The patients underwent operations a median of 2 days after the VSR diagnosis, 70% underwent operations within 3 days after VSR, and only 7 patients underwent operations in the late phase (>30 days from AMI). Accordingly, because the majority of patients underwent operations within an early and narrow time window after VSR, this parameter could not discriminate between survivors and nonsurvivors. Our results therefore do not exclude previous findings that surgical intervention even closer in time to the occurrence of VSR can increase the operative risk or that outcome is better when surgical intervention is delayed for weeks or months. Previous studies on the association between the timing of surgical intervention and early mortality are conflicting. Although shorter time from VSR to surgical intervention has been reported to increase the operative risk,^{20,21} the majority of studies show no association.^{13,19,23,30} This discrepancy

can partly be explained by the fact that the definition of early versus late surgical intervention varies between these studies. Not surprisingly, early mortality is higher for patients undergoing operations 1 to 2 days versus several weeks or months after VSR, while it is difficult to detect differences in outcome when comparing patients who undergo operations within the first days after VSR. None of these reports could demonstrate any association between the timing of surgical intervention and long-term survival.

Advanced age was not an independent predictor of 30-day mortality in our study. This corresponds well with several previous reports.^{13,17,19-21,30} Higher age was an independent risk factor for poor long-term survival, which probably only reflects that older persons have a shorter life expectancy.

Long CPB time was a moderate risk factor of 30-day mortality and total mortality, with the strongest effect on early outcome. Prolonged CPB was not a predictor of cumulative mortality in 30-day survivors. This suggests that the enhanced total mortality was a result of many postoperative deaths and that prolonged CPB had no persisting biologic effect. Even though CPB can provoke end-organ failure by triggering cascade systems and inducing inflammation, the adverse effect of prolonged CPB might be seriously confounded by underlying factors because CPB time can be a surrogate for a complicated and time-consuming procedure or for difficulties in weaning a severely damaged heart from CPB. The VSR repair and CABG procedures themselves probably did not contribute to the differences in CPB time between survivors and nonsurvivors because there was no significant difference in aortic crossclamp time.

Shock at surgical intervention was the strongest independent risk factor of 30-day mortality (odds ratio, 4.8). This corresponds well with other reports, in which shock has been identified as an important determinant of early death.^{14,15,17,19,30} We found that shock was also a predictor of total mortality (relative risk, 2.6), which is in contrast to several previous studies.^{8,14,19,21,30} However, shock was not a predictor of cumulative mortality in 30-day survivors. This suggests that the enhanced total mortality was a result of many postoperative deaths and that shock had no persisting biologic effect. The definition of shock varies widely in a clinical setting, and different studies use divergent definitions or have no definitions at all. We used a clear and strict definition of preoperative shock at surgical intervention: hypoperfusion leading to acute renal failure manifested by oliguria/anuria or an acute increase in creatinine value to greater than 200 $\mu\text{mol/L}$. Cardiogenic shock depends largely on the amount of damaged myocardium; the degree of left ventricular necrosis predicts shock in anterior VSR, and the degree of right ventricular necrosis predicts shock in posterior VSR.^{8,31} The AMI is usually biventricular in patients with VSR, and especially right ventricular dysfunction has been shown to be a strong predictor of early death.²³ Heart failure also depends on the amount of shunting, which is

again dependent on VSR size, on ventricular function, and on systemic versus pulmonary vascular resistance. In accordance with these findings, univariable analysis showed high maximal ASAT values after AMI and large preoperative shunt to be risk factors for 30-day mortality. Because these variables were measured in only a subset of patients, they could not be included in the regression analysis. The quality of preoperative echocardiographic analysis was suboptimal for a systematic quantification of myocardial function. Therefore although the extent of myocardial damage and preoperative shunt size were associated with poor early outcome, we could not expose the exact underlying hemodynamic mechanisms for development of shock.

The role of coronary revascularization during VSR repair is controversial. It can be argued that revascularization of transmurally infarcted myocardium is not logical, and because the culprit artery is often entrapped in the suture line of the ventriculotomy, it can be impossible to bypass.^{7,32,33} Coronary angiographic analysis is time-consuming and potentially dangerous in an unstable patient and can lead to contrast-induced renal failure. However, we have not lost any patients during coronary angiography, and this is also the experience from several other studies.^{8,23,32,34} We avoid contrast injection of the left ventricle to minimize the risk of cardiotoxic reactions and renal failure. A majority of studies have found no effect of concomitant CABG on early mortality^{14,16,18-22,34,35} or long-term survival,^{14,16,18,19,21} whereas CABG has been reported to reduce midterm survival in 30-day survivors.²⁰ Other studies support our findings that revascularization is associated with improved early³² and late^{22,32,34,35} survival, which indicates that concomitant CABG can control the added risk of coronary artery disease. We showed that complete global coronary revascularization and revascularization of the culprit artery both resulted in improved 30-day survival and long-term survival.

The frequency of 1-vessel versus multivessel disease differs between studies covering the same time period as ours, with a proportion of 1-vessel disease varying from 20% to 40%^{13,15,22} up to 50% to 60%.^{6,19,29,31,34} The effect of revascularization in a particular VSR cohort will depend on the pattern of coronary disease because revascularization will be more important in widespread coronary disease than in patients with only 1-vessel disease of a culprit artery. As many as 63% had 2- or 3-vessel disease, which explains the importance of complete revascularization in our cohort. Revascularization of the infarct-related artery itself was an important predictor of improved outcome. If not beneficial for the infarcted myocardium itself, revascularization of the culprit artery might improve perfusion of the ischemic border zone and control ventricular arrhythmias.

Even though complete coronary revascularization was associated with improved early and late survival, we have no proof of a direct cause-effect relationship. The positive effect might be seriously confounded by the quality of the

target vessels because CABG is difficult or impossible in patients with severely diseased and calcified coronary arteries. Accordingly, patients with better coronary arteries can undergo a more extensive coronary revascularization. In most retrospective studies, CABG is performed when indicated and technically possible. The extent of coronary revascularization is therefore a surrogate for the degree of coronary disease; patients with multivessel disease receive more distal anastomoses than patients with 1-vessel disease. No retrospective studies can, with certainty, validate the effect of CABG during VSR repair. One would need a large prospective trial with a control group in which CABG to operable arteries is deliberately omitted, and such a study will probably never be performed.

Thirty-day mortality decreased significantly from 45% in the first half to 21% in the second half of the study period. A similar reduction in operative risk during the years 1980 to 1992 (n = 109) has been described by Cox and coworkers,¹⁷ whereas 3 other authors found no reduction in early mortality during the recent decades: Killen and associates¹³ (1970-1994, n = 68), Labrousse and colleagues¹⁴ (1971-1991, n = 85), and Mantovani and coworkers²¹ (1983-2002, n = 50). The reasons for our improved early results in recent years are complex. Although time from AMI to VSR was constant throughout the study period, the natural course of AMI might have changed on introduction of thrombolysis and acute PCI. There was a shift toward more immediate surgical intervention in the second half of the study period, which might²⁰ or might not^{13,23,30} increase early mortality. Finally, the surgical repair technique has changed, and surgical experience and patient care might have improved. Probably these factors have stronger influence on operative risk than on long-term survival, for which cardiac pathology and comorbidity are more important. Accordingly, long-term survival was not influenced by year of surgical intervention. Twenty surgeons participated in the study. Thirteen surgeons performed less than 5 procedures, 4 surgeons performed 5 to 10 procedures, and 3 surgeons performed more than 10 procedures. Although surgeon was not a predictor of outcome, 5 of the most-frequent operating surgeons were residents in the first half of the study period and more experienced consultants during the last part. A learning curve effect can therefore partly explain improvement of early outcome during time. Naturally, observation time was shorter for patients undergoing operations in more recent years, making the statistical analysis less sensitive in detecting potential differences in late outcome.

In conclusion, repair of postinfarction VSR is still associated with high operative risk, with our 30-day mortality being 21% for the last decade. The 5- and 10-year cumulative survival was 50% and 32%, respectively. Shock at surgical intervention and incomplete coronary revascularization were strong and independent risk factors of 30-day mortality and poor long-term survival. Anterior or posterior location

of the VSR did not influence outcome. We recommend immediate coronary angiography and IABP support, followed by urgent surgical repair, which should include complete coronary revascularization.

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