Scandinavian Cardiovascular Journal

ISSN: 1401-7431 (Print) 1651-2006 (Online) Journal homepage: informahealthcare.com/journals/icdv20

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To cite this article: Farkas B. Vánky, Erik Håkanson, Tamás Maros & Rolf Svedjeholm (2004) Different characteristics of postoperative heart failure after surgery for aortic stenosis and coronary disease, Scandinavian Cardiovascular Journal, 38:3, 152-158, DOI: 10.1080/14017430410029734

To link to this article: https://doi.org/10.1080/14017430410029734

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Different characteristics of postoperative heart failure after surgery for aortic stenosis and coronary disease

Farkas B. Vánky[1], Erik Håkanson[2], Tamás Maros[1] and Rolf Svedjeholm[1]

Objective—Postoperative heart failure (PHF) remains a major determinant of outcome after cardiac surgery. However, possible differences in characteristics of PHF after valve surgery and coronary surgery (CABG) have received little attention. Therefore, this issue was studied in patients undergoing aortic valve replacement (AVR) and CABG, respectively.

Design—Three hundred and ninety-eight patients undergoing isolated AVR for aortic stenosis were compared with 398 patients, matched for age and sex, undergoing on-pump isolated CABG. Forty-five AVR and 47 CABG patients required treatment for PHF and these were studied in detail.

Results—The AVR group had longer aortic cross-clamp time and higher rate of isolated right ventricular heart failure postoperatively. Myocardial ischemia during induction and perioperative myocardial infarction were more common in the CABG group. One-year mortality was 8.9% in the AVR group vs 25.5% in the CABG group ($p = 0.05$).

Conclusions—The incidence of PHF was similar in both groups but different characteristics were found. Isolated right ventricular failure and PHF precipitated by septicemia were more common in AVR patients. PHF was more clearly associated with myocardial ischemia and infarction in CABG patients, which could explain their less favorable survival.

Key Words: aortic valve surgery, complications of surgery, coronary artery bypass surgery, heart failure, prognosis

Adverse outcome in cardiac surgery is closely related to postoperative heart failure (PHF). A recent survey of outcomes in 8641 patients who underwent coronary artery bypass grafting (CABG) operations in northern New England revealed a hospital mortality of 4.5% (1). Importantly, almost two-thirds of all deaths could be directly attributed to PHF. Typically the literature on PHF has focused on CABG or cardiac surgery in general, or on specific aspects or certain treatments (1–9). We found only one paper comparing outcome in patients with PHF after various procedures and it was published in the early 1970s (10). Possible differences regarding characteristics of heart failure after valve and coronary surgery seem to have received even less attention. Therefore, the clinical course of patients undergoing CABG and aortic valve replacement (AVR) for isolated aortic stenosis (AS), representing the two largest groups of adult cardiac surgical patients in our practice, was studied retrospectively in detail with special regard to PHF. The aim was to analyze the characteristics of PHF regarding demographics, presentation, events that may have elicited or contributed to heart failure, treatment and outcome.

MATERIAL AND METHODS

The University Hospital in Linköping is the only referral center in the southeast region of Sweden, serving a population of approximately 1 million. Demographic and peri-procedural data including complications were registered prospectively in a computerized institutional database (Summit Vista for Windows; Summit Medical Systems Inc., Version 1.98.1). All fields were defined in a data dictionary. Data on 1-year mortality and cause of death was retrieved from the Swedish Civil Registry and the National Death Registry.

From 1 January 1995 to 31 December 2000, 4806 patients underwent cardiac surgery. There were 398 patients operated with isolated AVR because of AS without clinically significant regurgitation. To account for evident differences regarding age and sex distribution these patients were compared with a cohort of 398 patients matched for age and sex, undergoing on-pump isolated first time CABG. This matching procedure resulted in two cohorts, both with an average age of 70 ± 10 years and 48% females. From these cohorts, 45 patients undergoing AVR (11.3%) and 47 patients undergoing CABG (11.8%) were found to have been treated for PHF. The records of these patients were scrutinized for further details regarding eliciting events, presentation of heart failure, treatment and outcome. These patients with PHF will be reported in detail and constitute our study groups (AVR group and CABG group; Fig. 1). The protocol was reviewed and approved by the ethics committee for medical research at Linköping University.

Received January 23, 2004; accepted February 20, 2004.
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Scand Cardiovasc J 38; 152–158, 2004

 ISSN 1401–7431
DOI 10.1080/14017430410029734

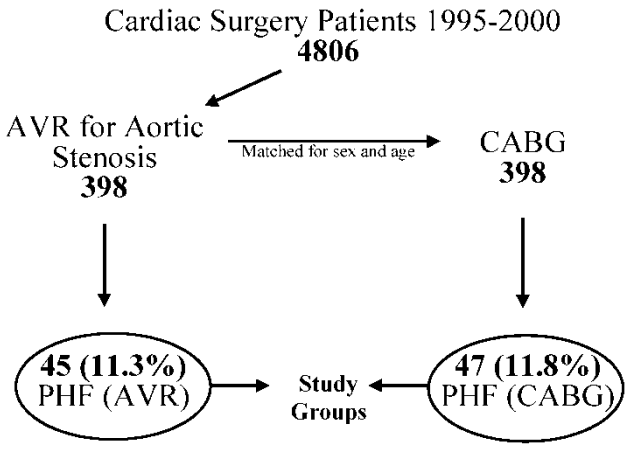

Fig. 1. Study group selection. AVR = aortic valve replacement; CABG = coronary artery bypass grafting; PHF = postoperative heart failure.

Clinical management

The patients underwent surgery using standard techniques with cardiopulmonary bypass (CPB) and aortic cross-clamping. Ringer's acetate and mannitol were used for priming the extracorporeal circuit. Moderate hemodilution (hematocrit 20–25%) and mild hypothermia (33–36°C) were usually employed. Antegrade or combined ante- and retrograde delivery of a cold crystalloid cardioplegic solution (Plegisol™, Abbot, Chicago, IL, US) supplemented with procaine hydrochloride was used for myocardial protection. Weaning from CPB was started at a rectal temperature of 35–36°C. Heparin was neutralized with protamine chloride. Ringer's acetate was used for volume substitution postoperatively. Shed mediastinal blood was routinely retransfused in the intensive care unit (ICU). Postoperative rewarming was facilitated by radiant heat provided by a thermal ceiling.

Monitoring

Arterial, central venous and pulmonary artery pressures were monitored in all patients as well as ECG with ST-segment analysis. A surgically introduced catheter into the pulmonary artery through the right ventricular outflow tract was routinely used for pressure monitoring and blood sampling for measurement of mixed venous oxygen saturation (SvO_2) (11). A Swan–Ganz catheter for cardiac output measurements was used in one-third of the patients whereas transesophageal echocardiography was employed in two-thirds of the patients.

DEFINITIONS

PHF was defined as an unsatisfactory hemodynamic state, usually supported by echocardiographic appearance of ventricular dysfunction, manifested as inability to wean from CPB without supportive measures or hemodynamic deterioration after weaning from CPB that required treatment. Patients treated with correction of volume and vascular resistance or single minor doses of inotropic agents were not classified to have PHF. The supportive measures or treatment consisted of inotropes, metabolic intervention combined with prolonged CPB to enhance recovery of myocardial function and mechanical circulatory assist. Inotropic treatment was defined as continuous infusion for more than 30 min of catecholamines (adrenaline, dobutamine) or a phosphodiesterase inhibitor (milrinone). Metabolic interventions constituted of intravenous glutamate or high-dose glucose–insulin–potassium (GIK). Details about these treatments have been given previously (7). Mechanical circulatory support was achieved with either intra-aortic balloon pump or Hemopump™ (DLP/Medtronic Inc., Grand Rapids, MI, USA).

Emergency operation was defined as a procedure that could not be postponed to the following day and was therefore usually operated on immediately but not later than 24 h from acceptance. Urgent operations were defined as scheduled procedures within 1 week on patients unable to leave the hospital because of unstable angina or hemodynamic state. These procedures were usually performed as soon as possible.

The detection of perioperative myocardial ischemia was mainly based on changes of the ST-segment (rather than fixed values) on ECG lead II and V, but usually implied ST depression exceeding 1 mm or ST elevation exceeding 2 mm. However, the suspicion of myocardial ischemia was frequently supported by regional dyskinesia on the transesophageal echocardiography.

Complications presented refer to in-hospital events occurring at our institution. Perioperative myocardial infarction (PMI) was diagnosed by biochemical markers of myocardial injury or by findings at autopsy. Aspartate aminotransferase (ASAT) exceeding 3.0 μkat/l with alanine aminotransferase less than half of the ASAT value, usually supported by MB isozyme of creatinine kinase >70 μg/l on the first postoperative morning or by a sustained elevation of troponin-T >2.0 μg/l on the fourth postoperative day, was considered diagnostic for PMI (12). Stroke was defined as a permanent or transient central neurologic deficit. The majority of patients with suspected neurological injury were examined by CT scan. Cognitive dysfunction was not included in the assessment. Sepsis was defined as a septic clinical condition with positive blood cultures. Patients were classified having unstable coronary artery disease by referring cardiologists basically according to criteria used by the FRISC II investigators (13). Preoperative left ventricular systolic function was evaluated by specially trained cardiologists with echocardiography and classified as normal, mildly depressed, moderately depressed or as severe systolic left ventricular dysfunction. Although precise ejection fractions were not routinely given a severe systolic left ventricular dysfunction generally corresponds to an ejection fraction of 0.30 or less. Hospital stay refers to hospital stay at the University Hospital (the majority of patients were discharged to the referring hospitals). The other variables in the database were defined according to STS Cardiac and Thoracic Databases definitions.

STATISTICAL ANALYSIS

Data are presented as mean ± standard deviation (SD). Statistical significance was defined as $p < 0.05$. Non-parametric tests, the Mann–Whitney U-test for continuous variables and Fisher's exact test for categorical variables were used.

RESULTS

Preoperative data

Approximately one of four in the AVR group and one of five in the CABG group had a severe systolic left ventricular dysfunction preoperatively. In the CABG group 83.0% had unstable coronary artery disease, 25.5% had significant left main stenosis and 91.3% had three-vessel disease. The AVR group compared favorably with the CABG group regarding New York Heart Association (NYHA) class IV and preoperative hemoglobin level. However, preoperative atrial fibrillation was more common in the AVR group. Details are presented in Table I.

Intraoperative data

In the AVR group aortic cross-clamp time and CPB time were significantly longer compared with the CABG group. Bioprostheses were used in 33 patients (73%). Aortic root enlargement was carried out in five patients (11%). The distribution of prosthesis size was the following: 19 mm (6.8%), 21 mm (22.7%), 23 mm (34.1%) and >23 mm (36.4%).

In the CABG group a higher proportion of the patients underwent urgent or emergency procedures and myocardial ischemia on induction (ECG changes and/or echocardiographic findings as given under definitions) occurred more frequently. Further details are presented in Table II.

Presentation and treatment of postoperative heart failure

PHF was evident at weaning from CPB in the majority of the patients in both groups (Table III). Presentation of heart failure differed. Seven of the AVR patients (15.6%) were considered to have isolated right ventricular failure, verified by echocardiography in the majority of cases, compared with none in the CABG group ($p < 0.01$).

Septicemia preceded heart failure in 8.9% of the AVR patients compared with none in the CABG group ($p = 0.05$). PMI was found in 13.3% of the patients in the AVR group vs 40.4% in the CABG group ($p < 0.001$). Ten of the 19 patients in the CABG group who developed PMI had ongoing ischemia at the start of the operation. The event assumed to have elicited PHF and presentation of PHF is given in Table III.

In patients that could sustain circulation to permit hemodynamic measurements available data showed an SvO_2 of 53.7 ± 11.0% ($n = 23$) and 48.0 ± 9.3%

Table I. *Preoperative data for aortic valve replacement (AVR) and coronary artery bypass grafting (CABG) patients with postoperative heart failure*

	AVR ($n = 45$)	CABG ($n = 47$)	p-Value	AVR 1-year mortality 8.9% (4/45)	CABG 1-year mortality 25.5% (12/47)
Age (years)	72 ± 10	73 ± 10	NS		
Female gender	37.8% (17/45)	53.2% (25/47)	NS	5.9% (1/17)	36.0% (9/25)
Body mass index (kg/m^2)	26.5 ± 4.1	27.2 ± 5.0	NS		
Diabetes mellitus	26.7% (12/45)	21.3% (10/47)	NS	16.7% (2/12)	20.0% (2/10)
Insulin treated diabetes mellitus	11.1% (5/45)	12.8% (6/47)	NS	20.0% (1/5)	33.3% (2/6)
COPD	15.6% (7/45)	10.6% (5/47)	NS	0.0% (0/7)	40.0% (2/5)
Cerebrovascular disease	13.3% (6/45)	10.6% (5/47)	NS	33.3% (2/6)	40.0% (2/5)
Peripheral artery disease	4.7% (2/43)	8.7% (4/46)	NS	0.0% (0/2)	25.0% (1/4)
Hypertension	43.2% (19/45)	42.2% (19/47)	NS	10.5% (2/19)	36.8% (7/19)
Atrial fibrillation or flutter	22.2% (10/45)	2.1% (1/47)	<0.01	10.0% (1/10)	0.0% (0/1)
NYHA II	14.0% (6/43)	9.8% (4/41)	NS	16.7% (1/6)	0.0% (0/4)
NYHA III	62.8% (27/43)	19.5% (8/41)	<0.001	7.4% (2/27)	25% (2/8)
NYHA IV	20.9% (5/43)	70.7% (20/41)	<0.001	0.0% (0/5)	40.0% (8/20)
Severe systolic LV dysfunction	24.4% (11/45)	20.5% (9/44)	NS	9.1% (1/11)	11.1% (1/9)
Cardiogenic shock	2.2% (1/45)	4.3% (2/47)	NS	100% (1/1)	50.0% (1/2)
Higgins score	4.8 ± 3.4[a]	5.7 ± 3.5[a]	NS		
Euroscore	6.7 ± 2.5[a]	7.6 ± 3.5[a]	NS		
Blood hemoglobin (g/l)	135 ± 15[a]	123 ± 18[a]	<0.001		
Plasma creatinine (μmol/l)	107 ± 20[a]	110 ± 33[a]	NS		
Plasma creatinine >167 μmol/l	0.0% (0/45)	10.6% (5/47)	0.06	–	40.0% (2/5)
Preoperative dialysis	0.0% (0/45)	0.0% (0/47)	NS	–	–

Figures within parentheses give the number of patients with different characteristics from those with available data. One-year mortality for the patients with different demographics is given in the rightmost columns.

[a] Mean ± standard deviation.

COPD = chronic obstructive pulmonary disease; NYHA = New York Heart Association class; LV = left ventricle.

Table II. *Intraoperative data for aortic valve replacement (AVR) and coronary artery bypass grafting (CABG) patients with postoperative heart failure*

	AVR ($n = 45$)	CABG ($n = 47$)	p-Value	AVR 1-year mortality 8.9% (4/45)	CABG 1-year mortality 25.5% (12/47)
Elective operation	73.3% (33)	12.8% (6)	<0.001	9.1% (3/33)	16.7% (1/6)
Urgent operation	22.2% (10)	61.7% (29)	<0.001	0.0% (0/10)	31.0% (9/29)
Emergency operation	4.4% (2)	25.5% (12)	<0.01	50.0% (1/2)	16.7% (2/12)
Cross-clamp time (min)	95 ± 30[a]	55 ± 21[a]	<0.001		
CPB time (min)	134 ± 42[a]	102 ± 32[a]	<0.001		
Average distal anastomoses		4.0 ± 0.9[a]			
Use of ITA		95.7%			

One-year mortality for the patients with different characteristics is given in the rightmost columns.
[a]Mean ± standard deviation.
CPB = cardiopulmonary bypass; ITA = internal thoracic artery.

Table III. *Incidence of potential causes to and presentation of postoperative heart failure*

	AVR ($n = 45$)	CABG ($n = 47$)	p-Value	AVR 1-year mortality 8.9% (4/45)	CABG 1-year mortality 25.5% (12/47)
Ischemia during induction	0.0% (0/45)	31.9% (15/47)	<0.001	–	20.0% (3/15)
Peroperative myocardial infarct	13.3% (6/45)	40.4% (19/47)	<0.01	33.3% (2/6)	26.3% (5/19)
Air in coronary vessels	6.7% (3/45)	2.1% (1/47)	NS	0.0% (0/3)	0.0% (0/1)
Tamponade	2.2% (1/45)	6.3% (3/47)	NS	0.0% (0/1)	100% (3/3)
Septicemia precedes heart failure	8.9% (4/45)	0.0% (0/47)	0.05	25.0% (1/4)	–
Isolated left ventricular failure	75.0% (33/44)	79.5% (35/44)	NS	6.1% (2/33)	17.1% (6/35)
Isolated right ventricular failure	15.6% (7/44)	0.0% (0/44)	<0.01	14.3% (1/7)	–
Left + right ventricular failure	9.1% (4/44)	20.9% (9/44)	NS	25.0% (1/4)	33.3% (3/9)
PHF at weaning off CPB	73.3% (33/45)	57.4% (27/47)	NS	9.1% (3/33)	18.5% (5/27)
Early PHF in the OR	2.2% (1/45)	23.4% (11/47)	<0.01	0.0% (0/1)	9.1% (1/11)
PHF in ICU	20.0% (9/45)	14.9% (7/47)	NS	11.1% (1/9)	42.9% (3/7)

Figures within parentheses give the number of patients with different characteristics from those with available data. One-year mortality for the patients with different characteristics is given in the rightmost columns.
PHF = postoperative heart failure; CPB = cardiopulmonary bypass; OR = operating room; ICU = intensive care unit.

($n = 23$) in the AVR and CABG group, respectively, before treatment. At this stage systolic blood pressure was 84 ± 16 mmHg ($n = 30$) and 82 ± 16 mmHg ($n = 31$) while pulmonary arterial diastolic pressure was 22 ± 5 mmHg ($n = 19$) and 18 ± 4 mmHg ($n = 26$) in respective groups. Left ventricular stroke work index was 23.4 ± 10.26 gram-meters/m^2 ($n = 8$) and 17.1 ± 7.36 gram-meters/m^2 ($n = 3$) in patients with available Swan–Ganz data in the AVR group and CABG group, respectively.

Inotropic treatment was given to 93.3% of the AVR group and 83.0% of the CABG group. Metabolic treatment with GIK or glutamate was given to 42.2% and 61.7%, respectively. Mechanical circulatory assist was used less frequently in the AVR group compared with the CABG group (2.2% vs 17.0%; $p < 0.05$). Further details including duration of treatment and the average dose of drugs per hour during the three most intensive hours of treatment are given in Table IV. Three hours after institution of treatment SvO_2 had increased to $64.2 \pm 7.0\%$ ($n = 42$) and $63.8 \pm 7.4\%$ ($n = 44$) in the AVR and CABG group, respectively. The systolic blood pressure had increased to 105 ± 14 mmHg ($n = 45$) and 100 ± 16 mmHg ($n = 46$) while pulmonary arterial diastolic pressure was 18 ± 4 mmHg ($n = 44$) and 17 ± 5 mmHg ($n = 43$) in respective groups. Left ventricular stroke work index was 28.0 ± 13.6 gram-meters/m^2 ($n = 25$) and 28.8 ± 17.9 gram-meters/m^2 ($n = 9$) in patients with available Swan–Ganz data in the AVR group and CABG group, respectively.

Postoperative outcome

ICU stay averaged 3.2 days in both groups. The difference in hospital stay, shown in Table V, was explained by the difference in preoperative hospital stay. Time from operation to discharge was 9.4 ± 4.8 days for the AVR group and 9.1 ± 6.2 days for the CABG group.

Crude 1-year mortality was 8.9% in the AVR group vs 25.5% in the CABG group ($p = 0.05$). Further details are presented in Table V. Mortality figures depending on different pre-, intra- and postoperative characteristics are given in respective tables (significance testing not performed due to small numbers).

Table IV. *Treatment given for postoperative heart failure*

	AVR ($n = 45$)	CABG ($n = 47$)	Dose[a] AVR (μg/kg/min)[b]	Dose[a] CABG (μg/kg/min)[b]	Duration of treatment AVR (h)[b]	Duration of treatment CABG (h)[b]
Inotropic treatment	93.3% (42)	83.0% (39)				
Adrenaline	80.0% (36)	70.2% (33)	0.026 ± 0.019	0.028 ± 0.015	26.1 ± 30.1	29.9 ± 37.5
Dobutamine	8.9% (4)	2.1% (1)	3.14 ± 1.62	2.56	66.2 ± 123.9	17.0
Milrinone bolus 50 μg/kg	2.2% (1)	10.6% (5)				
Milrinone infusion	13.3%* (6)	31.9% (15)	0.50 ± 0.13	0.13 ± 0.08	21.5 ± 28.2	34.3 ± 34.7
Nitroprusside	28.9%* (13)	10.6% (5)	1.18 ± 0.37	1.11 ± 0.40	20.1 ± 23.1	11.3 ± 13.5
Metabolic treatment	42.2% (19)	61.7% (29)				
GIK	40.0% (18)	55.3% (26)				
Glutamate	11.1% (5)	21.3% (10)				
IABP or Hemopump	2.2%* (1)	17.0% (8)			24.0	42.0 ± 9.1

[a] The average dose per hour during the 3 h of most intensive treatment.
[b] Mean $\pm$ standard deviation.
GIK = glucose–insulin–potassium; IABP = intra-aortic balloon pump.
* $p < 0.05$.

Table V. *Postoperative data for aortic valve replacement (AVR) and coronary artery bypass grafting (CABG) patients with postoperative heart failure*

	AVR ($n = 45$)	CABG ($n = 47$)	*p*-Value	AVR 1-year mortality 8.9% (4/45)	CABG 1-year mortality 25.5% (12/47)
ICU stay (days)	3.2 ± 3.9[a]	3.2 ± 3.3[a]	NS		
Hospital stay (days)	11.1 ± 4.8[a]	9.6 ± 5.4[a]	NS		
Stroke	6.7% (3/45)	8.5% (4/47)	NS	33.3% (1/3)	75.0% (3/4)
Plasma creatinine, highest (μmol/l)	135 ± 87[a]	145 ± 76[a]	NS		
Plasma creatinine, at discharge (μmol/l)	106 ± 59[a]	115 ± 44[a]	<0.05		
Plasma creatinine elevation >50%	18.6% (8/43)	18.6% (8/43)	NS	25.0% (2/8)	50.0% (4/8)
Dialysis	4.5% (2/45)	0.0% (0/47)	NS	0.0% (0/2)	–
Intestinal ischemia	0.0% (0/45)	6.4% (3/47)	NS	–	100% (3/3)
30-day mortality	6.7% (3/45)	21.3% (10/47)	0.07		
1-year mortality	8.9% (4/45)	25.5% (12/47)	0.05		

Figures within parentheses give the number of patients with different characteristics from those with available data. One-year mortality for the patients with different complications is given in the rightmost columns.
[a] Mean $\pm$ standard deviation.
SvO_2 = mixed venous oxygen saturation; ICU = intensive care unit.

DISCUSSION

In this study, the incidence of PHF was similar (11.3% vs 11.8%) after AVR for isolated AS and CABG while the eliciting events and presentation of heart failure differed between the groups. PHF in the CABG group was more clearly related to preoperative ischemia and PMI, which could explain why short-term outcome was less favorable.

One of the strengths of this study is that the study cohorts were selected from the complete number of patients undergoing cardiac surgery within an area of Sweden with 1 million inhabitants during 1995–2000. Therefore, no referral selection bias should be present. To account for differences in age and sex distribution between the study groups the CABG group was matched for these factors. Completeness of survival data was ascertained by double-checking the database against the Swedish Civil Registry.

Because of the lack of a generally accepted criteria for defining PHF or low output syndrome and the retrospective character of the study, the diagnosis of PHF was dependent on individual judgement of various surgeons and anesthetists. The main determinant for cardiac output is the metabolic demands of the whole body (14). We have found that in sedated anesthetized patients low cardiac outputs are common even in patients with excellent SvO_2 and evidence of satisfactory recovery of myocardial metabolism (15). Diagnosis of low output syndrome based on Swan–Ganz measurements, hence, carries the risk of including patients that actually do not have heart failure. Routine use of Swan–Ganz catheters may even lead to an overuse of vasoactive drugs and prolonged ICU stay (16, 17). Atrial filling pressures can be difficult to interpret in patients undergoing aortic valve procedures for AS with hypertrophic ventricles, particularly if there is a tendency to outflow obstruction. At our institution

a reliance on markers for adequate circulation (in particular SvO_2) and echocardiographic appearance rather than fixed hemodynamic criteria are employed to diagnose PHF (11, 14). Obviously hemodynamic measures such as atrial filling pressures, systemic pressure, and in selected cases, Swan–Ganz data are used to aid the interpretation. A consequence of this strategy is restrictive use of inotropes both regarding the numbers of patients treated and the dosages employed. To exclude patients with mild transient heart failure (due to premature weaning) that resolved after short prolongation of CPB as the sole measure only patients that required active measures (apart from correction of volume or vasotonus) to facilitate weaning from CPB or to correct deteriorating hemodynamics were chosen for the study groups. Although the numbers might have been higher or lower at other institutions we suggest that the approximate 10% of patients considered to have had PHF give a fair reflection of this clinical problem. The cohort of CABG patients studied here was matched for age and sex with AVR patients with subsequent implications. In spite of this, the incidence of PHF did not differ markedly from what other investigators have reported after CABG (1–3).

Despite scrutinizing the records of the individual patients, a potentially eliciting event to PHF could only be identified in approximately two-thirds of the CABG patients and one-third of the AVR patients. On the other hand, the surgical procedure per se and in particular cardioplegic arrest contributes to postoperative depression of myocardial function (18). Patients with preoperatively compromised left ventricular function are thus at increased risk of developing clinical evidence of heart failure after surgery (3). Hence, it is conceivable that preoperative left ventricular dysfunction, that was common in both groups, was an important contributor to PHF (3, 19, 20).

Heart failure presented at weaning from CPB or in the early postoperative course in the majority of patients, without inter-group differences. PHF that presented later in the postoperative course was usually preceded by tamponade or septicemia.

PHF in patients undergoing AVR was not as closely associated to preoperative ischemic events and PMI as in the CABG group. However, the few patients in the AVR group with PMI had a mortality rate of a similar magnitude. Although the number of patients are too few to make any definitive conclusions it is conceivable that the lower incidence of myocardial infarction could explain why the short-term outcome seemed more favorable in AVR patients (21). Patients in the AVR group also less frequently required treatment with mechanical circulatory assist, which could suggest less severe heart failure in the AVR patients and/or a lower threshold for using mechanical circulatory assistance in association with ischemia.

The role of right ventricular function has received increasing attention and there are data suggesting that right ventricular dysfunction contributes to low output syndrome in almost half of the cases after CABG procedures (4). However, isolated right ventricular failure was found by Davila-Roman et al. in only 2 out of 31 patients with low output syndrome after isolated CABG whereas it was found in 2 out of 7 patients with low output syndrome after isolated AVR. Our study did not permit a complete analysis of this issue, but in agreement with the findings by Davila-Roman et al. isolated right ventricular failure was a problem mainly associated with heart failure after AVR. The association between isolated right ventricular failure and AVR surgery remains to be clarified. However, it can be speculated that several factors may have contributed such as increased after-load for the right ventricle due to left ventricular hypertrophy and diastolic dysfunction, inappropriate protection of the right ventricle or impaired right ventricular function due to inappropriate de-airing procedures with resulting air embolism into the right coronary artery (4, 22–24).

We acknowledge that it can be difficult to establish the casual relationship between events and heart failure in individual patients and in some patients a combination of factors was present. However, we suggest that the incidence of factors and events potentially responsible for PHF may reflect their relative importance in general for this complication.

Half of the patients in the CABG group with PMI had signs of ischemia at the induction of anesthesia. This is in agreement with previous studies that have identified preoperative ischemia as a major cause of PMI (21, 25). It can be speculated that improved preoperative management of these patients potentially could prevent adverse postoperative outcome.

Mortality figures are presented for respective groups depending on the presence of different pre-, intra- and postoperative characteristics. The numbers were too low to permit meaningful statistical intra- and inter-group comparisons. However, it seems that female sex did not have the same adverse influence on outcome in patients with PHF after AVR as after CABG. As could be anticipated, the few patients with preoperative cardiogenic shock, postoperative renal failure and stroke had high mortality rates.

Our study was not designed to evaluate efficacy of treatment. As previously mentioned, patients in the CABG group were treated more frequently with mechanical circulatory assist. Inotropic agents were used just as much in both groups but there was a shift toward higher use of phosphodiesterase inhibitors in the CABG patients. The use of catecholamines was in general

limited to low or moderate doses (9). Perioperative care and treatment of PHF differed from traditional management by the use of metabolic interventions (7).

In general, judging from the literature, treatment for PHF seems to be quite standardized regardless of type of surgery. The different characteristics of PHF identified in the present study suggest that a greater focus on casual aspects of heart failure, tailoring of treatment and prophylactic measures may be warranted. In cases with a severe ischemic insult and a high suspicion of myocardial infarction the use of treatments that increase myocardial oxygen demand should be avoided or minimized in our opinion (26). Instead early insertion of intra-aortic balloon pump, after-load reduction and other measures that enhance myocardial recovery and the hemodynamic state without unnecessary increase of myocardial workload deserve consideration. In situations with predominant right ventricular failure use of drugs, such as phosphodiesterase inhibitors, that reduce right ventricular after-load and stimulate right ventricular contractility seem advisable.

This study also highlights the need to review criteria and definitions for PHF or low output syndrome. Consensus upon generally and easily applicable criteria would facilitate further research on one of the most important complications after cardiac surgery.

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