Changing patterns of patients undergoing emergency surgical revascularization for acute coronary occlusion

Importance of myocardial protection techniques

Between 1977 and 1992 a total of 163 consecutive patients underwent emergency coronary artery bypass grafting after acute coronary occlusion (94% after failed angioplasty). Patients were divided into four groups according to the method used for myocardial protection. The crystalloid cardioplegia group included 30 patients operated on from 1977 to 1980; the hypothermic fibrillation group included 60 patients (1980 to 1986); the blood cardioplegia group included 36 patients (1986 to 1989); and the blood cardioplegia with controlled reperfusion group included 37 patients (1989 to 1992). Preoperative data, ischemic time interval, collateral blood flow, intraoperative data, regional wall motion, global ejection fraction, myocardial infarct–specific electrocardiographic changes, enzyme release, rhythm disturbances, mortality, prevalence of intraaortic balloon pumping, and inotropic support were assessed in this retrospective study. Our data indicate that the current spectrum of patients undergoing emergency coronary artery bypass grafting after acute coronary occlusion are at a significantly higher risk compared with those 15 years ago, that is, increase in age (53 ± 1 versus 59 ± 2 years; $p < 0.05$), three-vessel disease (38% versus 3%; $p = 0.004$), acute occlusion of the left main coronary artery (11% versus 0%; $p = 0.02$), preoperative cardiogenic shock (35% versus 3%; $p = 0.007$), prevalence of acute two-vessel occlusion (22% versus 3%; $p = 0.05$), prevalence of previous infarction (59% versus 23%; $p = 0.04$), and duration of ischemia (3.0 ± 0.2 versus 4.1 ± 0.3 hours; $p < 0.05$). Despite the increase in patients with severely compromised ventricular function during recent years, the overall hospital mortality decreased to 5% (2/37) when maximal protection of the ischemic and remote myocardium was performed (preoperative intraaortic balloon pump, combined antegrade/retrograde substrate-enriched blood cardioplegia, warm induction, controlled reperfusion, prolonged vented bypass). Single-vessel disease was always associated with a low mortality, whereas mortality could be reduced with controlled blood cardioplegia in patients with multivessel disease (6%) and cardiogenic shock (15%). The immediate return of regional contractility in the previously ischemic area after controlled reperfusion might serve as an explanation for these favorable results. After unmodified blood reperfusion, normokinesis or slight hypokinesis occurs in only 34% to 46% in the early postoperative period (1 to 4 weeks) in comparison with 86% after controlled blood cardioplegia reperfusion ($p < 0.05$). We conclude that there is a significant increase in risk factors in patients undergoing emergency coronary artery bypass grafting and that improved methods of intraoperative myocardial protection are needed for these compromised patients. (J THORAC CARDIOVASC SURG 1993;106:137-48)

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Since the beginning of percutaneous transluminal coronary angioplasty (PTCA) in 1977, several other techniques have enlarged the armamentarium of the interventional cardiologists (laser, stents, atherectomies, rotablation). These devices, as well as improved catheter techniques, have led to an extension of the indication for the various PTCA techniques to patients with multivessel disease, acute myocardial infarction, and cardiogenic shock.[1] Furthermore, patients with acute coronary occlusion due to dissections after PTCA are not always referred for emergency coronary artery bypass grafting (CABG), but often an attempt is made to reopen the occlusions by reangioplasties or stents. Therefore there has been a tremendous change in the clinical status of the patients referred for surgical treatment after acute coronary occlusion (very proximal occlusions, left main stenosis, cardiogenic shock), and it is not surprising that emergency CABG for acute coronary artery occlusion is associated with an operative mortality of 2.5% to 15.6% and up to 50% frequency of periprocedural myocardial infarction.[2-10]

Myocardial protection plays a major role in patients with acute coronary occlusions because ischemic and remote myocardium are energy depleted at the beginning of the operation.[11] During aortic crossclamping, additional damage might be imposed on a myocardium with limited ischemic tolerance. Various techniques were used for myocardial protection during surgical reperfusion after acute coronary occlusion, that is, crystalloid cardioplegia,[12] continuous hypothermic fibrillation,[13] blood cardioplegia,[14] or blood cardioplegia combined with controlled reperfusion (controlled blood cardioplegia).[14, 15] There is however, no clinical study evaluating these protection techniques and their effect on global and regional myocardial function after surgical reperfusion for acute coronary occlusion in regard to the preoperative status of the patient.

The first therapeutic PTCA was carried out by Grüntzig in September 1977 in Zürich,[16] and 1 month later a PTCA program was started at the Johann Wolfgang Goethe-University in Frankfurt.[17] The present report reviews our 15-year experience (1977 to 1992) with surgical interventions for acute coronary occlusion (94% due to angioplasty failures) with use of crystalloid cardioplegia, hypothermic fibrillation, blood cardioplegia, or controlled blood cardioplegia for intraoperative myocardial protection. The aim of this retrospective analysis was (1) to identify the preoperative condition and risk factors of the patients and their change during the 15-year period and (2) to evaluate the results of emergency CABG in regard to the preoperative risk factors and method used for myocardial protection.

Patients and methods

Patient population. Between October 1977 and February 1992, 163 consecutive patients were operated on for acute coronary occlusion, and they form the basis for this report. They were transferred to our department from five different cardiologic centers in Frankfurt.

The cause for acute coronary occlusion was failed angioplasty in 153 of 163 patients (94%) (Table 1). In the other 10 patients, acute coronary occlusion was caused by laser angioplasty, acute stent thrombosis, routine diagnostic cardiac angiography, acute naturally occurring coronary artery thrombosis, or early graft occlusion.

A cardiac surgical revascularization is considered "an emergency" if it is carried out as soon as possible after acute coronary occlusion. Operations that are delayed for more than 6 to 8 hours are not considered emergencies. Patients referred for emergency surgical revascularization were transported immediately into the operating room. All angioplasties were elective, and patients were administered antiplatelet drugs before the procedure and received heparin during dilatation. Angioplasty was contraindicated in patients with stenosis of the left main coronary artery. PTCA failure that required emergency operation was a determination made by the intervening cardiologist and was defined as one in which the patient had intractable pain associated with persistent electrocardiographic (ECG) changes, cardiac tamponade, cardiogenic shock, or radiographically visible coronary occlusion. Excluded from this classification were PTCAs in which the failures were due to inability to traverse the lesion with the balloon, or to unsuccessful dilatation but without untoward symptoms or ECG changes requiring delayed operation. The intraaortic balloon pump (IABP) was inserted preoperatively when indicated.

The onset of acute coronary occlusion was defined as the time of acute dissection as evidenced by coronary angiography. In patients with naturally occurring occlusions, after angiography or early graft occlusion, the onset of coronary occlusion was defined as the time of origin of chest pain and hemodynamic deterioration and was documented by ECG evidence of hyperacute ST elevation with or without Q waves or loss of R wave progression.

The diagnosis of cardiogenic shock was made according to accepted criteria (hypotension, oliguria, evidence of inadequate peripheral perfusion, elevated left atrial filling pressures, and need for inotropes or IABP or both.[18]

Demographic and preoperative variables analyzed included age, sex, global ejection fraction (EF), extent of coronary artery disease, clinical and ECG evidence of acute myocardial infarction just before PTCA, and distribution of vessels subjected to PTCA. Catheterization data were used to determine the extent of coronary artery disease. Left main coronary artery disease was defined as greater than 50% compromise of vessel diameter, and other major coronary artery vessel disease was defined as greater than 70% narrowing in any view. Global EF was determined from ventriculography or, in some cases, from nuclear medicine studies. Previous myocardial infarction was defined as ECG evidence of a transmural infarction.

Myocardial protection technique. From October 1977 to February 1992, four different methods of myocardial protection were used for emergency CABG. The patients were grouped according to the myocardial protection technique used. In general, between 1977 and 1980 crystalloid cardioplegia was used, between 1980 and 1986 hypothermic fibrillation, between 1986

Table I. *Cause for acute coronary occlusion*

	Crystalloid cardioplegia (n = 30)	*Hypothermic fibrillation (n = 60)*	*Blood cardioplegia*	
			Uncontrolled (n = 36)	*Controlled (n = 37)*
PTCA	100%	100%	97% (35/36)	76% (28/37)
Acute thrombosis*	0%	0%	0%	11% (4/37)†
Angiography	0%	0%	3% (1/36)	5% (2/37)
Laser angioplasty	0%	0%	0%	3% (1/37)
Stent thrombosis	0%	0%	0%	3% (1/37)
Early graft occlusion	0%	0%	0%	3% (1/37)

*Acute thrombosis of the native coronary artery.
†$p = 0.02$ versus hypothermic fibrillation

1989 blood cardioplegia, and thereafter controlled blood cardioplegia. Proximal graft anastomosis was always constructed during systemic rewarming.

Crystalloid cardioplegia. After systemic heparinization (300 IU/kg) and cannulation of the aorta and both venae cavae via the right atrium, cardiopulmonary bypass was conducted either with a Harvey H-200 (William Harvey Research Co., Anaheim, Calif.) or a Galen-Optiflow oxygenator (model 42-201, Cobe Laboratories, Inc., Denver, Colo.) and a Sarns roller pump (model 5000, Sarns Inc./3M, Ann Arbor, Mich.). Pump prime consisted of 1.5 L 5.2% levulose and lactated Ringer's solution in a ratio of 2:1. Rectal temperature of the patients was lowered to 28° ± 2° C. Systemic blood flow was maintained at 2.4 L/min per square meter. The first graft was always placed into the vessel supplying the ischemic area. Crystalloid cardioplegic solution was given at a flow of 200 ml/min up to a total of 1000 ml. The composition of the crystalloid cardioplegic solution is shown in Table II.

Hypothermic fibrillation. For hypothermic fibrillation a two-stage venous cannula was used. Blood temperature was lowered to 25° C, resulting in a rectal temperature of 28° C. After spontaneous ventricular fibrillation had started, the left ventricle was vented via the right superior pulmonary vein. The ischemic perfusion pressure was kept always above 60 mm Hg. Intermittent aortic occlusion was not used. A flow rester (Bio-Vascular, St. Paul, Minn.) was inserted into the coronary artery during the distal anastomosis. Perfusion of each vein graft was done via the perfusion line after the distal anastomosis was completed. Thereafter rewarming was started, and the heart was defibrillated at a rectal temperature of 28° to 30° C.

Blood cardioplegia. After systemic heparinization (300 IU/kg) and cannulation of the aorta and right atrium with a single venous cannula, cardiopulmonary bypass was conducted with a Harvey H-1500 oxygenator (Bard Cardiopulmonary Division, C.R. Bard, Inc., Santa Ana, Calif.) and a Stöckert roller pump (Stöckert Instrumente GmbH, Munich, Germany). Pump prime consisted of 35 ml/kg lactated Ringer's solution. Rectal temperatures of the patients were lowered to 28° ± 2° C. Systemic blood flow was maintained at 2.4 L/min per square meter.

The left ventricle was vented through the cardioplegic needle via the aortic root only if necessary. The aorta was crossclamped, and the blood cardioplegic solution[14] was infused via a roller pump into the aortic root at a flow rate of 250 to 300 ml/min for 3 minutes. After induction of asystole, the high-K^+ bag was switched to the low-K^+ bag (maintenance), and flow was reduced to 200 ml/min. The cardioplegic delivery system consisted of a disposable tubing to deliver a 4:1 blood cardioplegic ratio. Cooling was achieved by putting the tubing in an ice bag. The temperature of the blood cardioplegic solution delivered into the aortic root was in the range of 8° to 14° C, dependent on the flow rate and duration of infusion. A myocardial temperature of 16° to 18° C could be achieved with this multidose regimen. The tubing was attached to a coronary cardioplegic adapter containing four limbs that allow simultaneous cardioplegic distribution into the aorta (13-gauge needle) and into the vein grafts. A reinfusion into the vein graft and the aortic root was done every 20 minutes at a flow rate of 200 ml/min for 2 minutes. The total amount of blood cardioplegic solution used in this group ranged from 800 ml to 2000 ml. After completion of all distal anastomoses, systemic temperature was raised to 35° C, and the aortic clamp was removed. Global warm blood cardioplegic reperfusate or warm blood cardioplegic induction was not employed in this group. Extracorporeal circulation was discontinued either after the patient's temperature was raised to 35° C or cardiac contractions were sufficient to support the circulation.

Table II. *Composition of the crystalloid cardioplegic solution**

NaCl	1.461 gm = 25.0 mmol
KCl	0.373 gm = 5.0 mmol
$CaCl_2$	0.074 gm = 0.5 mmol
$NaHCO_3$	2.100 gm = 25.0 mmol
D, L-Mg-aspartate	0.721 gm = 2.0 mmol
Procaine hydrochloride	1.091 gm = 4.0 mmol
Glucose	1.980 gm = 10.0 mmol
Mannitol	36.440 gm = 200.0 mmol
Hydroxyethyl starch	60.000 gm
pH	7.4
Osmolality	320 mOsml/kg H_2O

*Cardioplegesche Perfusionslösung, Fresenius, Bad Homburg, Germany.

Blood cardioplegia with controlled reperfusion. The left ventricle was vented in all patients by a catheter passed through the left pulmonary veins. In addition to the bags for blood cardioplegic induction and maintenance, a third bag was prepared for the regional cardioplegic reperfusate, containing a calcium antagonist (diltiazem), a higher concentration of glucose, less calcium, and a higher osmolarity.[14]

CARDIOPLEGIC INDUCTION AND MAINTENANCE. The solution and flow for cold induction and multidose maintenance were

Table III. *Preoperative patient characteristics*

	Crystalloid cardioplegia (n = 30)	*Hypothermic fibrillation (n = 60)*	*Blood cardioplegia*	
			Uncontrolled (n = 36)	*Controlled (n = 37)*
Age (yr)	53 ± 1	56 ± 1	57 ± 1	59 ± 1*
Men, women (%)	80/20	82/18	75/25	81/19
Coronary heart disease				
One-vessel	67% (20/30)	55% (33/60)	47% (17/36)	16% (6/37)†
Two-vessel	30% (9/30)	37% (22/60)	33% (12/36)	46% (17/37)
Three-vessel	3% (1/30)	8% (5/60)	19% (7/36)	38% (14/37)‡
Left main	0%	2% (1/60)	3% (1/36)	11% (4/37)§
Mean	1.3 ± 0.1	1.5 ± 0.1	1.7 ± 0.1	2.2 ± 0.1*
Previous infarction	23% (7/30)	42% (25/60)	53% (19/36)	59% (22/37)‖
Cardiogenic shock	3% (1/30)	7% (4/60)	17% (6/36)	35% (13/37)¶
Preoperative arrest	0%	2% (1/60)	3% (1/36)	14% (5/37)#
Preoperative IABP	0%	7% (4/60)	11% (4/36)	16% (6/37)**
Occluded vessel				
LAD	60%	58%	58%	43%
RCA	33%	23%	31%	32%
LCX	7%	17%	17%	16%
Left main	0%	2%	3%	8%
Prevalence of two-vessel occlusion††	3% (1/30)	0%	11% (4/36)	22% (8/37)‡‡
Ischemic time interval (hr)	3.0 ± 0.2	3.6 ± 0.2	3.3 ± 0.3	4.1 ± 0.3*

Data are mean ± standard error of the mean. *IABP*, Intraaortic balloon pump; *LAD*, left anterior descending coronary artery; *RCA*, right coronary artery; *LCX*, left circumflex coronary artery.

*$p < 0.05$ versus CCP.

†$p = 0.005$ versus CCP; $p = 0.007$ versus HF; $p = 0.06$ versus BCP.

‡$p = 0.004$ versus CCP; $p = 0.009$ versus HF; §$p = 0.02$ versus HF.

‖$p = 0.03$ versus CCP.

¶$p = 0.007$ versus CCP; $p = 0.004$ versus HF.

#$p = 0.01$ versus HF; $p = 0.06$ versus CCP.

**$p = 0.04$ versus CCP.

††Includes acute occlusion of the first diagonal branch.

‡‡$p < 0.05$ versus CCP; $p = 0.008$ versus HF.

Table IV. *Prevalence of subtotal occlusion and collateral blood flow*

	Crystalloid cardioplegia (n = 30)	*Hypothermic fibrillation (n = 60)*	*Blood cardioplegia*	
			Uncontrolled (n = 36)	*Controlled (n = 37)*
Collateral flow*	23% (7/30)	28% (17/60)	11% (4/36)	22% (8/37)
Subtotal occlusion due to subtotal artery occlusion	17% (5/30)	20% (12/60)	11% (4/36)	0%
Perfusion catheter	0%	8% (5/60)	17% (6/36)	22% (8/37)
Stent implantation	0%	0%	3% (1/36)	5% (2/37)

*Assessed by coronary angiography.

identical to those used for uncontrolled reperfusion. Combined antegrade and retrograde delivery of blood cardioplegic solution for induction and maintenance was used in the last 23 patients. For retrograde delivery of blood cardioplegic solution, transatrial cannulation of the coronary sinus with a Retroplegia cannula (Research Medical, Inc., Salt Lake City, Utah) was performed. Warm blood cardioplegic induction for 5 minutes with a flow of 250 ml/min was used in patients with cardiogenic shock and was followed by cold induction for 5 more minutes.

CONTROLLED REGIONAL REPERFUSION. At the start of the completion of the final anastomosis, the perfusionist increased the temperature of the blood cardioplegic solution to 37° C. After completion of the last distal anastomosis, warm diltiazem-containing, substrate-enriched controlled regional blood cardioplegic solution was given antegrade into the aorta and all grafts for 2 minutes at 150 ml/min. Thereafter the aortic clamp was removed and the controlled blood cardioplegic solution was given at a flow rate of 50 ml/min only into the graft supplying

Table V. *Intraoperative data*

	Crystalloid cardioplegia (n = 30)	*Hypothermic fibrillation (n = 60)*	*Blood cardioplegia*	
			Uncontrolled (n = 36)	*Controlled (n = 37)*
Aortic occlusion time (min)	31 ± 2	—	34 ± 3	43 ± 3
Bypass time (min)	66 ± 4	71 ± 4	68 ± 5	105 ± 6*
Distal anastomosis	1.5 ± 0.1	1.7 ± 0.1	1.9 ± 0.2	2.2 ± 0.1*
IMA grafts to				
Ischemic area	0%	0%	14% (5/36)	0%
Remote area	0%	0%	6% (2/36)	11% (4/37)†
Additional procedures	0%	2% (1/60)‡	0%	5% (2/37)§
Spontaneous sinus rhythm	50% (15/30)	0%	19% (7/36)	81% (30/37)¶

Data are mean ± standard error of the mean. *IMA*, Internal mammary artery.
*$p < 0.05$ versus crystalloid cardioplegia, hypothermic fibrillation, uncontrolled blood cardioplegia.
†$p = 0.02$ versus hypothermic fibrillation.
‡One patient with aortic valve replacement.
§One patient with aortic valve replacement and another patient with mitral valve replacement.
¶$p = 0.002$ versus uncontrolled blood cardioplegia.

Table VI. *Prevalence of hospital mortality, postoperative IABP, and postoperative inotropic support and serum release of creatine kinase and myocardial band-isoenzyme of creatine kinase*

	Crystalloid cardioplegia (n = 30)	*Hypothermic fibrillation (n = 60)*	*Blood cardioplegia*	
			Uncontrolled (n = 36)	*Controlled (n = 37)*
Hospital mortality				
Overall	3% (1/30)	13% (8/60)	11% (4/36)	5% (2/37)
Single vessel	0% (0/20)	3% (1/33)	6% (1/17)	0% (0/6)
Multivessel	10% (1/10)	26% (7/27)	16% (3/19)	6% (2/31)
Shock	100% (1/1)	100% (4/4)	50% (3/6)	15% (2/13)
IABP	—	15% (9/60)	39% (14/36)	19% (7/37)
Duration (days)				
1	—	33% (3/9)	14% (2/14)	14% (1/7)
2	—	22% (2/9)	71% (10/14)	57% (4/7)
3	—	11% (1/9)	7% (1/14)	14% (1/7)
4	—	22% (2/9)	7% (1/14)	14% (1/7)
5	—	11% (1/9)	—	—
Inotropic agents	54% (13/24)	67% (35/51)	49% (17/35)	46% (17/37)
Duration (days)				
1	38% (5/13)	31% (11/35)	18% (3/17)	53% (9/17)
2	23% (3/13)	17% (6/35)	29% (5/17)	24% (4/17)
3	15% (2/13)	17% (6/35)	18% (3/17)	24% (4/17)
4	8% (1/13)	14% (5/35)	18% (3/17)	—
5	8% (1/13)	6% (2/35)	6% (1/17)	—
>5	8% (1/13)	14% (5/35)	12% (2/17)	—
CK_{max} (U/L)	848 ± 173	1398 ± 419	1067 ± 336	816 ± 156
CK-MB$_{max}$ (U/L)	46 ± 11	65 ± 28	59 ± 17	57 ± 8

Data are mean ± standard error of the mean.
IABP, Intraaortic balloon pump; *CK*, creatine kinase; *CK-MB*, myocardial band-isoenzyme of creatine kinase.

the region revascularized for acute coronary occlusion for an additional 18 minutes. In patients with acute occlusion of the left main coronary artery, flow was increased to 100 ml/min and given into the vein graft supplying the left anterior descending coronary artery and the left-circumflex coronary artery regions. Normal blood was delivered into the remainder of the heart. The total amount of blood cardioplegic solution used for controlled reperfusion ranged from 2000 to 3200 ml. After this 20-minute phase of controlled regional reperfusion, total bypass was prolonged for an additional 30 minutes to maintain low oxygen requirements in the regional segment and to allow it to recover its oxygen utilization capacity gradually. Thereafter extracorporeal circulation was discontinued. Additional bypass was used if cardiac output was not satisfactory.

Table VII. *Regional contractility in the previously ischemic area after 1 to 2 weeks postoperatively (blood cardioplegia) resp. 4 weeks (crystalloid cardioplegia, hypothermic fibrillation) (using a scoring system 0 to 4*)*

	Crystalloid cardioplegia (n = 18)	*Hypothermic fibrillation (n = 37)*	*Blood cardioplegia*	
			Uncontrolled (n = 21)	*Controlled (n = 35)*
Normokinesis	33% (6/18)	22% (8/37)	29% (6/21)	49% (17/35)†
Slight hypokinesis	11% (2/18)	24% (9/37)	5% (1/21)	37% (13/35)
Severe hypokinesis	22% (4/18)	14% (5/37)	14% (3/21)	6% (2/35)
Akinesis	22% (4/18)	38% (14/37)	52% (11/21)	9% (3/35)‡
Dyskinesis	11% (2/18)	3% (1/37)	0%	0%
Mean	1.7 ± 0.3	1.7 ± 0.2	1.9 ± 0.3	0.7 ± 0.28§

Data are mean ± standard error of the mean.

*Scoring system: 0 = normokinesis; 1 = slight hypokinesis; 2 = severe hypokinesis; 3 = akinesis; 4 = dyskinesis.

†$p = 0.07$ versus hypothermic fibrillation.

‡$p = 0.02$ versus hypothermic fibrillation; $p = 0.007$ versus uncontrolled blood cardioplegia.

§$p < 0.05$ versus crystalloid cardioplegia, hypothermic fibrillation, uncontrolled blood cardioplegia.

Table VIII. *Global EF (%)*

	Crystalloid cardioplegia (n = 29)	*Hypothermic fibrillation (n = 52)*	*Blood cardioplegia*	
			Uncontrolled (n = 14)	*Controlled (n = 17)*
Before acute coronary occlusion	72 ± 3	75 ± 1	65 ± 4	56 ± 7
1-4 wk postop	70 ± 3	67 ± 4	48 ± 7	55 ± 4

Data are mean ± standard error of the mean.

Table IX. *Myocardial specific ECG changes (MI-ECG) and prevalence of postoperatively developed rhythm disturbances*

	Crystalloid cardioplegia	*Hypothermic fibrillation*	*Blood cardioplegia*	
			Uncontrolled	*Controlled*
MI-ECG	23% (5/22)	45% (25/56)	47% (17/36)	46% (17/37)
Rhythm disturbances	50% (10/20)	46% (26/57)	53% (18/34)	22% (8/37)

Parameters

Regional wall motion. Preoperative assessment of regional contractility either by echocardiography or by radionuclide ventriculography was not performed in order to limit the ischemic interval as much as possible. Intraoperatively (after median sternotomy and pericardiotomy) regional wall motion of the ischemic area was evaluated macroscopically by the surgeon. Postoperatively follow-up evaluation of the regional function was determined by transthoracic echocardiography at the first and seventh postoperative days and with radionuclide ventriculography 1 day before discharge from the surgical ward (seventh postoperative day).

Regional wall motion was analyzed from either radionuclide ventriculography or echocardiography with the use of a scoring system[15]: grade 0, normal contractility; grade 1, mild to moderate hypokinesia; grade 2, severe hypokinesia; grade 3, akinesia; grade 4, dyskinesia.

RADIONUCLIDE VENTRICULOGRAPHY. Electrocardiograph-gated acquisitions were done at rest after in vivo labeling of red blood cells with stannum chlorine (first injection) and 550 MBq technetium 99m (second injection 20 minutes later). Every acquisition lasted 5 minutes. The investigation was carried out in a supine position and left anterior oblique view (30° to 45°). Data processing was done using a fully automated program according to the procedure proposed by Standke and coworkers.[19] The regional EF was calculated in nine equiangular sectors. Sectorial EF values were displayed as a profile and compared with normal values. Hypokinesia was determined if sector values of an individual patient decreased under the normal profile. For classification of dyskinesis or akinesis, we performed a regional Fourier phase analysis, and it was displayed in colors.

ECHOCARDIOGRAPHY. Two-dimensional echocardiography was performed using a movable Sonotron Kardio CV 60 (Diasonic-Sonotron, Co., Mainz, Germany) studying the parasternal long- and short-axis, the apical two- and the apical four chamber views in regard to the wall motion. The left ventricle was divided into three segments according to the blood supply: left anterior descending coronary artery (anterolateral wall), circumflex artery (lateral wall), and right coronary artery (inferior and basal wall).

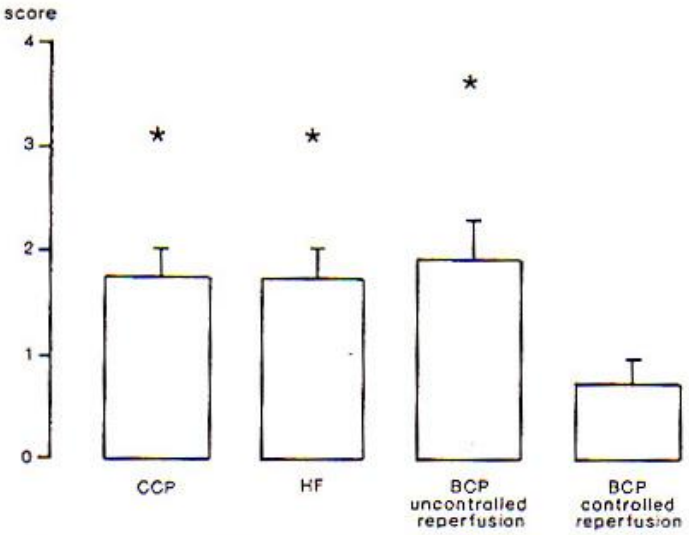

Fig. 1. Regional contractility in the previously ischemic area after 1 to 4 weeks postoperatively. The ordinate represents the regional contractility with use of a scoring system with 0 = normal contractility, 1 = mild to moderate hypokinesis, 2 = severe hypokinesis, 3 = akinesis, 4 = dyskinesis. Note (1) limited immediate return of regional shortening after ischemic periods between 3 and 4 hours, if the initial reperfusate was normal blood given to the previously ischemic segment by using either crystalloid cardioplegia *(CCP)*, hypothermic fibrillation *(HF)*, or blood cardioplegia with uncontrolled reperfusion *(BCP)*; (2) significantly improved immediate recovery of systolic function if blood cardioplegia with controlled reperfusion was used *(right bar)*. $^{*}p < 0.05$.

Global EF. Global EF was measured preoperatively with ventriculography and postoperatively either with ventriculography or radionuclide ventriculography.

Extent of collateral blood flow. Angiographic flow in the distal portion of the occluded artery (either partial antegrade flow or retrograde collateral flow) was recorded. In the group [illegible] controlled blood cardioplegia only patients with total coronary occlusion were included, whereas a few patients with subtotal narrowing of the coronary artery were present in the other groups (crystalloid cardioplegia, hypothermic fibrillation, blood cardioplegia) (see Table IV).

The prevalence of the insertion of perfusion catheters was noted. In all patients acute coronary occlusion was confirmed intraoperatively. After incision of the coronary artery supplying the ischemic area, antegrade flow was virtually absent during the cardioplegic infusions, respectively, during hypothermic fibrillation.

Duration of ischemia. The duration of ischemia was defined as the time period between onset of symptoms until start of reperfusion.

Electrocardiogram. Preoperatively and 2 and 7 days postoperatively, a 12-lead electrocardiogram was recorded and examined according to the Minnesota code for detecting ECG evidence of myocardial infarction.

Enzymes. Preoperatively and 4, 24, 48 hours, and 7 days postoperatively venous blood samples were taken for serial serum determinations of creatine kinase and creatine kinase-myocardial band isoenzyme. Creatine kinase-myocardial band concentrations were measured using a solid-phase, two-site immunoenzymatic assay (Tandem-E CKMB II, Hybritech, Inc., San Diego, Calif.).

Rhythm disturbances. Postoperatively, rhythm disturbances were evaluated by continuous ECG monitoring from the time of discharge from the operating room until 48 to 72 hours postoperatively. New episodes of rhythm disturbances were divided into arrhythmias and conduction disturbances. The following rhythm disturbances were recorded: bradycardias (pulse rate less than 60 beats/min) persisting longer than 10 minutes or necessitating pacemaker stimulation; sinus tachycardia (pulse rate greater than 120 beats/min lasting longer than 10 minutes); premature ventricular beats (greater than 12 beats/min).

Severe ventricular reperfusion arrhythmias were defined as multifocal premature ventricular contractions, runs of ventricular tachycardia, or ventricular fibrillation requiring intensive medical treatment or electric countershock or both.

Spontaneous defibrillation. Spontaneous defibrillation was defined as resumption of sinus rhythm after removing the aortic clamp without prevalence of ventricular fibrillation. Defibrillation was carried out if ventricular fibrillation persisted during a 0.5- to 1-minute interval after the aortic clamp was removed.

Mortality. Mortality was defined as any hospital death.

IABP and the need for inotropic support. Preoperatively and postoperatively the need for implanting an IABP or inotropic support or both was recorded. Inotropic support was defined as the need for dopamine (greater than 4 μg/min per kilogram), dobutamine, or epinephrine administration for more than 30 minutes. Dopamine used in low doses only for enhancement of renal perfusion was not included.

Statistics. Data were analyzed by means of the Epistat Statistical Computer Package and the Stat-View II provided by the Department of Biomathematics of the Johann Wolfgang Goethe-University. Parametric data are expressed as mean ± standard error of the mean. Group data were compared by analysis of variance and nominal data by Fisher's exact test. Significant differences were defined as probabilities for each test of p less than 0.05.

Results

The results are shown in Tables III to IX and in Fig. 1.

Patient profile. The preoperative patient characteristics differed tremendously between 1977 (crystalloid cardioplegia) and 1992 (controlled blood cardioplegia). The age of the patients increased significantly (53 ± 1 versus 59 ± 1; $p < 0.05$), as did the extent of coronary heart disease. The majority of patients protected with either crystalloid cardioplegia or hypothermic fibrillation had one-vessel disease (67% respectively 55%) (see Table III); in contrast, patients protected with controlled blood cardioplegia had an increased prevalence of three-vessel disease (38% versus 3%, $p = 0.004$) and left main artery stenosis (11% versus 0%; $p = 0.02$). This is also reflected by the mean number of diseased vessels (2.2 ± 0.1 versus 1.3 ± 0.1; $p < 0.05$). In addition, the prevalence of previous myocardial infarction increased significantly (59% versus 23%; $p = 0.04$). Patients in cardiogenic shock were only rarely operated on during the period during which crystalloid cardioplegia or hypothermic fibrillation was used (3% resp. 7%), whereas 35% of the patients in the controlled blood cardioplegia reperfusion group were preoperatively in cardiogenic shock (35% versus 7%; $p = 0.004$). This increased prevalence of patients in preoperative cardiogenic shock is also reflected by the significantly increased prevalence of preoperative insertion of IABP: 0% in the group with crystalloid cardioplegia and 16% ($p = 0.04$) in the group with controlled blood cardioplegia.

In all groups the left anterior descending coronary artery was the vessel most often occluded. More patients were operated on with acute occlusion of the left main coronary artery in the group with controlled blood cardioplegia than in the group with either crystalloid cardioplegia or hypothermic fibrillation, however (8% versus 0%). The more aggressive approach by the cardiologists is also reflected by the prevalence of acute two-vessel occlusion. Simultaneous angioplasty of two major coronary arteries in one patient with subsequent occlusion virtually did not occur in the groups protected with crystalloid cardioplegia and hypothermic fibrillation, whereas in those patients protected with blood cardioplegia the prevalence was 11% and 22% (uncontrolled respectively controlled reperfusion).

The ischemic interval was significantly prolonged in patients who were protected with blood cardioplegia followed by controlled reperfusion (4.1 ± 0.3 hours) compared with crystalloid cardioplegia (3.0 ± 0.2 hours; $p < 0.05$), hypothermic fibrillation (3.6 ± 0.2 hours), and blood cardioplegia with uncontrolled reperfusion (3.3 ± 0.3 hours; $p < 0.05$) (see Table III). The percentage of patients having well-developed collateral myocardial blood flow was similar in all groups (see Table IV).

Intraoperative data (see Table V). The higher prevalence of patients with multivessel disease in the groups protected with blood cardioplegia is also reflected by the increased number of distal anastomoses in this group. The number of distal anastomoses was significantly higher in the group with controlled reperfusion (2.2 ± 0.1) compared with crystalloid cardioplegia (1.5 ± 0.1; $p < 0.05$), hypothermic fibrillation (1.7 ± 0.1; $p < 0.05$), or uncontrolled blood cardioplegia (1.9 ± 0.2; $p < 0.05$). This has led to a longer aortic occlusion time in the group with controlled reperfusion. The prolonged bypass time in this group was due to the necessity for having a 30-minute-period of beating empty state after removal of the aortic clamp.

Internal mammary artery grafts were rarely used in this hemodynamically severely restricted cohort of patients. The prevalence of spontaneous sinus rhythm after removal of the aortic clamp was highest in the group with controlled blood cardioplegia (81%) compared with the other groups.

Hospital mortality (see Table VI). The overall hospital mortality was very low (3%) in the group in which the majority of patients (67%) had single-vessel disease. With an increased prevalence of patients with multivessel disease and preoperative cardiogenic shock, the overall mortality increased to 13% resp. 11%. Controlled blood cardioplegia, however, was able to decrease the overall hospital mortality to 5%, despite the very high prevalence of multivessel disease, acute left main coronary artery occlusion, preoperative cardiogenic shock and previous infarctions.

The value of a maximal myocardial protection strategy is best reflected in the patient group with preoperative cardiogenic shock. Whereas the mortality was 100% in the group with crystalloid cardioplegia or hypothermic fibrillation, it decreased to 50% when blood cardioplegia was used and was reduced further to only 15% if controlled blood cardioplegia was used for myocardial protection.

Prevalence of postoperative IABP and inotropic support. There was no difference in the prevalence of postoperative IABP between groups. In the majority of patients IABP was necessary for 2 days. The need for

postoperative inotropic support varied between 46% and 67% (see Table VI).

Enzyme release. There was no difference among groups in the release of either creatine kinase or the myocardial band–Isoenzyme of creatine kinase (see Table VI). The relatively high values for the maximal creatine kinase–myocardial band and creatine kinase release reflect the substantial ischemic injury to which the patients were subjected because of the acute coronary occlusion.

Regional wall motion. Only controlled blood cardioplegia produced consistent immediate return of regional contractility in the previously ischemic area (see Table VII and Fig. 1). The regional contractility score was significantly lower in this group (0.7 ± 0.2; $p < 0.05$) compared with crystalloid cardioplegia (1.7 ± 0.3), hypothermic fibrillation (1.7 ± 0.2), and uncontrolled blood cardioplegia (1.9 ± 0.3). Without attempts to control the conditions of the initial reperfusion and the composition of the reperfusate, 33% to 52% of the patients had either akinesis or dyskinesis after 7 days postoperatively (see Table VII). In contrast, only three patients with controlled blood cardioplegia (9%) had this regional functional abnormality at the seventh postoperative day ($p = 0.007$). Similarly, the number of patients who showed a normal contractility or only a slight hypokinesis in the previously ischemic segment was significantly higher in the controlled blood cardioplegia group (86%) compared with the other three groups (44%, 46%, 34%; $p = 0.04$).

Global EF. The decreased preoperative cardiac performance during the last 15 years is also reflected by the decrease in global EF between groups: patients operated with controlled blood cardioplegia had a lower EF (56% ± 7%) compared with crystalloid cardioplegia (72% ± 3%), hypothermic fibrillation (75% ± 1%), or blood cardioplegia (65% ± 4%). The postoperative EFs did not differ from the preoperative values (see Table VIII).

Myocardial infarct–specific ECG and rhythm disturbances. Infarct-specific ECG signs were noted in 23% to 47% of patients without differences between groups (see Table IX). The prevalence of reperfusion arrhythmias was reduced with controlled blood cardioplegia (22%) compared with crystalloid cardioplegia (50%) or hypothermic fibrillation (46%; $p = 0.07$) (see Table IX).

Discussion

This is a retrospective analysis of the results of emergency revascularization after acute coronary occlusion from a consecutive series of 163 patients between 1977 and 1992. There was no rigid, prospectively designed protocol to manage these patients, and improvements in preoperative and postoperative treatment, operative procedures, and the conduct of cardiopulmonary bypass might also have influenced the results. Furthermore, the retrospective nature of this study led to incomplete data sets in some patients. Therefore, comparison of reperfusion methods and myocardial protection techniques must be cautiously made. Clearly, a prospective, randomized trial would be helpful in this area, but none of the surgical series dealing with revascularization after acute coronary occlusion are prospectively randomized. The only recently published randomized, controlled study of surgical reperfusion for evolving infarction was presented by Koshal and colleagues,[20] but patients more than 65 years of age were excluded, as were those with previous infarctions, cardiogenic shock, and renal failure. The reason for the retrospective nature of almost all studies is the small number of patients with acute coronary occlusions referred for surgical treatment.

Our data show a significant deterioration in the preoperative cardiac performance and myocardial reserve capacity of patients referred for emergency CABG during the last 15 years, which is consistent with the reports of others.[21-23] There is a significant increase in age (53 versus 59 years), multivessel disease (84% versus 33%), previous myocardial infarction (59% versus 23%), cardiogenic shock (35% versus 3%), preoperative cardiac arrest (14% versus 0%), simultaneous occlusion of two coronary arteries (22% versus 3%), acute occlusion of the left main coronary artery (8% versus 0%), ischemic time (4.1 versus 3.0 hours) and a decrease in single-vessel disease (16% versus 67%) and global EF before coronary occlusion occurred (56% ± 7% versus 72% ± 3%) (see Tables III and VIII). The importance of these risk factors is supported by studies that define reduced global EF,[8, 24] multivessel disease,[24] cardiogenic shock,[8] age more than 65 years,[8] and absent collateral blood flow[8] as predictors of operative mortality after surgical interventions for acute infarction. In our study the rise in these risk factors was associated with an increase in hospital mortality from 3% (1977 to 1980) to 13% (1981 to 1986) respectively 11% (1986 to 1989) (see Table VI).

The rise in risk factors is related to the fact that cardiologists become increasingly persistent in attempting to correct PTCA complications. Patients with acute coronary occlusions are often treated with repeat balloon dilatation, thrombolysis, implantable stents, or "stack" balloons, or coronary dissections are sometimes "tacked up" by other balloon techniques. If these maneuvers fail to revascularize the ischemic area and the patient remains hemodynamically stable, he/she will receive further conservative treatment. Only if the hemodynamic condition of the patient is deteriorating is surgical treatment advocated.

Our data show that hospital mortality was low (3%; see Table VI) in the crystalloid cardioplegia group in which the majority of patients had single-vessel disease (67%), multivessel PTCA was seldom attempted (3%), cardiogenic shock (3%) and previous infarction (23%) were infrequent, and preoperative global EF was in the normal range before the onset of acute coronary occlusion (72% ± 3%). Even without reperfusion, acute coronary occlusion in single-vessel disease carries a low risk of mortality.[13] Even though perioperative mortality was low, however, immediate recovery of regional contractility was poor (33% of all patients had dyskinetic or akinetic segments) despite short ischemic intervals (3 hours).

An increased prevalence of previous infarction (42%), cardiogenic shock (7%), and multivessel disease (45%) has led to an increased overall mortality of 13% in the group operated on with hypothermic fibrillation (see Tables III and IV). In this group single-vessel disease again carried a low risk (3%), while mortality in multivessel disease increased to 26%. All four patients in preoperative cardiogenic shock died.

A further increase in patients with previous infarctions (53%), cardiogenic shock (17%), multivessel disease (53%), prevalence of acute two-vessel occlusion (11%), and acute left main occlusion (3%) (see Table III) did not result in an increase in overall operative mortality. In contrast, hospital mortality decreased in patients with multivessel disease from 26% (hypothermic fibrillation) to 16% and for patients in cardiogenic shock from 100% (hypothermic fibrillation) to 50% (see Table VI). The results in this sicker patient group might be explained by the improvement in the function of remote myocardium protected by blood cardioplegia, because the recovery of regional contractility in the previously ischemic area was not different from the other groups in which normal blood was used as the primary reperfusate (see Table VII).

During the last period (1989 to 1992), additional improvements in perioperative myocardial protection were used: preoperative insertion of IABP,[25] warm blood cardioplegia induction,[26] substrate enrichment,[27] combined antegrade and retrograde delivery of cardioplegic solution,[28, 29] controlled reperfusion,[14, 15] and prolonged vented bypass.[30] This resulted in a decrease in the overall mortality (5%) despite a further increase in risk factors (84% multivessel disease, 35% cardiogenic shock, 59% previous infarctions, 8% acute occlusion of the left main coronary artery, 22% acute two-vessel occlusion, 14% preoperative cardiac arrest). Furthermore, mortality in multivessel disease decreased from 26% (hypothermic fibrillation) respectively 16% (blood cardioplegia) to 6%, and in patients with preoperative cardiogenic shock, from 100% (crystalloid cardioplegia, hypothermic fibrillation) respectively 50% (blood cardioplegia) to 15% (see Table VI). This decrease in mortality is accompanied by a substantial return of segmental shortening in the previously ischemic area in 86% of all patients (see Table VII and Fig. 1).

Our data on early regional contractility (see Table VII) are in agreement with experimental studies of controlled respectively uncontrolled reperfusion, that is, no difference was noted on regional contractility between all groups in which the initial reperfusion was normal blood given at systemic pressure and no attempts were made to vent the left ventricle (that is, crystalloid cardioplegia, hypothermic fibrillation, and uncontrolled blood cardioplegia). Only 22% to 33% of the patients recovered normal systolic shortening after 1 to 4 weeks postoperatively after normal blood reperfusion, and the previously ischemic segment was nonfunctional (i.e., dyskinesis or akinesis) in 39% to 52%. In contrast, control of the initial reperfusion (controlled blood cardioplegia) restored normokinesis in 49%, and some contractility (hypokinesis and normokinesis) was observed after 1 to 2 weeks in 91% of all patients. Only three patients (9%) had akinetic segments in the previously occluded region, and long-term follow-up will show if this represents reversible ("stunned") or irreversible injured cardiac muscle. One of the three patients with postoperative akinetic wall motion was reperfused by implanting a stent in the dissected and previously occluded coronary artery after 2 hours of ischemia before he was referred for surgical revascularization. It was shown experimentally that controlled reperfusion does not reverse regional wall motion abnormalities if normal blood reperfusion precedes controlled reperfusion.[31]

The inability of normal blood reperfusion after either crystalloid cardioplegia or hypothermic fibrillation to restore immediate contractility even after short ischemic intervals (30 minutes) is supported by a recent experimental study comparing hypothermic fibrillatory arrest and crystalloid cardioplegia followed by normal blood reperfusion during revascularization for acute coronary occlusion.[32] The infarct sizes after only 30 minutes of ischemia, measured by vital staining (nitroblue tetrazolium chloride) and expressed as percent of the region at risk, were not different between groups (18.1% [crystalloid cardioplegia] respectively 18.8% [HF].[31]

Reperfusion injury after regional ischemia may not critically affect systemic hemodynamics if enough remote myocardium that can develop hypercontractility is left. In the present study total loss of contractile function in the reperfused segment was not accompanied by a reduction in global hemodynamics, such as global EF (see Table IX), if the majority of patients in this cohort have single-vessel disease and therefore hypercontracting remote

myocardium (72% versus 70% EF, preoperatively versus postoperatively). In patients with reduced myocardial reserve (such as previous myocardial infarction or chronically depressed myocardium in severe coronary heart disease), however, global EF will decrease if the previously occluded area is not able to contract and contributes to the generation of cardiac output.

We conclude from this study that there has been a tremendous increase in risk factors in patients with acute coronary occlusions referred for emergency CABG. Maximal protection of the remote and ischemic myocardium in combination with complete revascularization is necessary to improve the surgical results in this challenging patient group.

REFERENCES

1. Gunnar RM, Bourdillon PDV, Dixon DW, et al. ACC/AHA guidelines for the early management of patients with acute myocardial infarction. Circulation 1990;82:664-707.
2. Reul GJ, Cooley DA, Hallman GL, et al. Coronary artery bypass surgery for unsuccessful percutaneous transluminal coronary angioplasty. J THORAC CARDIOVASC SURG 1984;88:685-94.
3. Cowley MJ, Dorros G, Kelsey SF, Van Raden M, Detre KM. Emergency coronary bypass surgery after coronary angioplasty: the National Heart, Lung, and Blood Institute's Percutaneous Transluminal Coronary Angioplasty Registry experience. Am J Cardiol 1984;53:22C-6C.
4. Golding LAR, Loop FD, Hollman JL, et al. Early results of emergency surgery after coronary angioplasty. Circulation 1986;74(Pt 2):III26-9.
5. Ferguson BT, Muhlbaier LH, Salai DL, Wechsler AS. Coronary bypass grafting after failed elective and failed emergent percutaneous angioplasty. Relative risks of emergent surgical intervention. J THORAC CARDIOVASC SURG 1988;95:761-72.
6. Barner HB, Lea JW IV, Naunheim KS, Stoney WS. Emergency coronary bypass not associated with preoperative cardiogenic shock in failed angioplasty, after thrombolysis, and for acute myocardial infarction. Circulation 1989;79(Pt 2):I152-9.
7. Edwards FH, Bellamy RF, Burge JR, et al. True emergency coronary artery bypass surgery. Ann Thorac Surg 1990;49:603-11.
8. Athanasuleas CL, Geer DA, Arciniegas JG, et al. A reappraisal of surgical intervention for acute myocardial infarction. J THORAC CARDIOVASC SURG 1987;93:405-14.
9. Guyton RA, Arcidi JM, Langford DA, et al. Emergency coronary bypass for cardiogenic shock. Circulation 1987;76(Pt 2):V22-7.
10. Flameng W, Sergeant P, Vanhaecke J, Suy R. Emergency coronary bypass grafting for evolving myocardial infarction. J THORAC CARDIOVASC SURG 1987;94:124-31.
11. Beyersdorf F, Acar C, Buckberg GD, et al. Studies on prolonged acute regional ischemia: III. Early natural history of simulated single and multivessel disease with emphasis on remote myocardium. J THORAC CARDIOVASC SURG 1989;98:368-80.
12. Pelletier LC, Pardini A, Renkin J, David PR, Hebert Y, Bourassa MG. Myocardial revascularization after failure of percutaneous transluminal coronary angioplasty. J THORAC CARDIOVASC SURG 1985;90:265-71.
13. Akins CW. Early and late results following emergency isolated myocardial revascularisation during hypothermic fibrillatory arrest. Ann Thorac Surg 1987;43:131-7.
14. Beyersdorf F, Sarai K, Maul FD, Wendt T, Satter P. Immediate functional benefits after controlled reperfusion during surgical revascularization for acute coronary occlusion. J THORAC CARDIOVASC SURG 1991;102:856-66.
15. Allen BS, Buckberg GD, Schwaiger M, et al. Studies of controlled reperfusion after ischemia. XVI. Early recovery of regional wall motion in patients following surgical revascularization after eight hours of acute coronary occlusion. J THORAC CARDIOVASC SURG 1986;92:636-48.
16. Grüntzig A, Myler A, Hanna ES, Turina M. Transluminal angioplasty of coronary artery stenosis [Abstract]. Circulation 1977;56(Pt 2):I84.
17. Kaltenbach M. Transluminal angioplasty of coronary artery stenosis [Abstract]. Circulation 1979;60:264.
18. Scheidt S, Ascheim R, Kelley T. Shock after acute myocardial infarction: a clinical and hemodynamic profile. Am J Cardiol 1970;26:556-64.
19. Standke R, Hör G, Klepzig H Jr, Maul FD, Bussmann WD, Kaltenbach M. Sectoranalysis of left ventricular function by fully automated equilibrium radionuclide ventriculography. Int J Card Imaging 1985;1:87-97.
20. Koshal A, Beanlands DS, Davies RA, Nair RC, Keon WJ. Urgent surgical reperfusion in acute evolving myocardial infarction. A randomized controlled study. Circulation 1988;78(Pt 2):I171-8.
21. Naunheim KS, Fiore AC, Fagan DC, et al. Emergency coronary artery bypass grafting for failed angioplasty: risk factors and outcome. Ann Thorac Surg 1989;47:816-23.
22. Selinger SL, Berg R Jr, Leonard JJ, Coleman WS, DeWood MA. Surgical intervention for acute myocardial infarction. Tex Heart Inst J 1984;11:44-51.
23. de Feyter PJ, van den Brand M, Jaarman GJ, et al. Acute coronary occlusion during and after percutaneous transluminal coronary angioplasty. Frequency, prediction, clinical course, management, and follow-up. Circulation 1991;83:927-36.
24. Roubin GS, Harris PJ, Bernstein L, Kelly DT. Coronary anatomy and prognosis after myocardial infarction in patients 60 years of age and younger. Circulation 1983;67:743-8.
25. DeWood MA, Notske RN, Hensley GR, et al. Intraaortic balloon counterpulsation with and without reperfusion for myocardial infarction shock. Circulation 1980;6:1105-12.
26. Rosenkranz ER, Buckberg GD, Laks H, Mulder DG. Warm induction of cardioplegia with glutamate-enriched blood in coronary patients with cardiogenic shock who are

dependent on inotropic drugs and intra-aortic balloon support. J THORAC CARDIOVASC SURG 1983;86:507-18.

27. Rosenkranz ER, Okamoto F, Buckberg GD, Robertson JM, Vinten-Johansen J, Bugyi H. Safety of prolonged aortic clamping with blood cardioplegia. III. Aspartate enrichment of glutamate blood cardioplegia in energy-depleted hearts after ischemic and reperfusion injury. J THORAC CARDIOVASC SURG 1986;91:428-35.
28. Drinkwater DC, Laks H, Buckberg GD. A new simplified method of optimizing cardioplegic delivery without right heart isolation: antegrade/retrograde blood cardioplegia. J THORAC CARDIOVASC SURG 1990;100:56-64.
29. Loop FD, Higgins TL, Panda R, Pearce G, Estafanous FG. Myocardial protection during cardiac operations: decreased morbidity and lower cost with blood cardioplegia and coronary sinus perfusion. J THORAC CARDIOVASC SURG 1992;104:608-18.
30. Laschinger JC, Grossi EA, Cunningham JN Jr, et al. Adjunctive left ventricular unloading during myocardial reperfusion plays a major role in minimizing myocardial infarct size. J THORAC CARDIOVASC SURG 1985;90:80-5.
31. Quillen J, Kofsky ER, Buckberg GD, Partington MT, Julia PL, Acar C. Studies of controlled reperfusion after ischemia. XXIII. Deleterious effects of simulated thrombolysis preceding simulated coronary artery bypass grafting with controlled blood cardioplegic reperfusion. J THORAC CARDIOVASC SURG 1991;101:455-64.
32. Greene PS, Cameron DE, Griffiths EM, DiNatale JM, Gardner TJ. Does hypothermic fibrillatory arrest improve myocardial protection during emergency revascularization? Ann Thorac Surg 1989;48:38-42.