Acute severe cardiac failure complicating myocardial infarction

Experience with 100 patients referred for consideration of mechanical left ventricular assistance

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One hundred patients were referred with suspected acute cardiac failure following acute myocardial infarction. The diagnosis was confirmed in 72: 31 of these patients underwent elective medical treatment, with 2 survivors (6%); 41 were accepted for counterpulsation, but 9 died before this could be initiated and another 2 died shortly after vain attempts to pass the balloon catheter were abandoned; 30 patients underwent counterpulsation with 14 hospital survivors (47%). Survivor status was usually good. Results of counterpulsation were better in patients who were not shocked (with 5/5 survivors) than in those who were in shock (with 9 of 25 survivors). Results support the view that counterpulsation (alone or combined with corrective surgery) may play an important role in the complications of myocardial infarction provided intervention is early.

Failure of the heart as a pump is the most common cause of hospital death after myocardial infarction. Inefficacy of drug therapy in this condition has prompted more aggressive measures, including use of devices to assist the circulation and urgent revascularization surgery. The most successful device is the intra-aortic balloon counterpulsation system, introduced by Moulopoulos, Topaz, and Kolff in 1962, and popularized over the past 4 years by Austen and colleagues at the Massachusetts General Hospital (Buckley *et al.*, 1970; Sanders *et al.*, 1972a; Leinbach *et al.*, 1973b).

Published results of arterial counterpulsation show a continuing high mortality but it is apparent that different workers have differed in their indications, in the speed with which they have implemented circulatory assistance, in the duration for which they have applied assistance, and in the way they have complemented circulatory assistance with revascularization and other corrective surgery (Kantrowitz, 1970; Buckley *et al.*, 1970; Sanders *et al.*, 1972b; Soroff *et al.*, 1972; Gold *et al.*, 1972; Mueller *et al.*, 1972; Leinbach *et al.*, 1973a; Dilley Ross, and Bernstein, 1973; Scheidt *et al.*, 1973; Windsor *et al.*, 1973).

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We report here the results of a programme of arterial counterpulsation and corrective surgery which was started in 1971. There has been a gradual loosening of criteria for circulatory support, a more expeditious evaluation of patients considered for support, and a waxing enthusiasm for the project.

Patients

One hundred patients were referred for consideration of mechanical assistance over a 22-month period. All these patients were thought to have acute myocardial infarction and severe cardiac failure resistant to medical measures. Of the patients, 57 were referred from other hospitals while 43 had been admitted directly to this hospital with myocardial infarction.

Patients were assessed and placed in different categories (Table 1). Uniformity of criteria over the period was achieved by having two investigators (M. O'Rourke and V. Chang) responsible for assessment and patient selection. Twenty-eight patients did not fulfil the criteria for acceptance either because another diagnosis was established or because continued medical therapy led to sustained improvement. Other diagnoses included dissecting aortic aneurysm (3 patients) and respiratory infection complicating infarction (2 patients).

There were 72 patients who fulfilled the criteria of acute severe cardiac failure complicating myocardial

TABLE I Classification. Patients referred for consideration of mechanical assistance

	No.	Hospital survivors	Hospital survival rate (%)
1) Criteria not fulfilled	28	16	57
2) Criteria fulfilled			
(a) Assistance contra-indicated	19	1	5
(b) Assistance unavailable	12	1	8
(c) Accepted for counterpulsation			
(i) Death before initiated	9	0	0
(ii) Unable to initiate	2	0	0
(iii) Counterpulsation initiated	30	14	47

infarction. Initially all patients were expected to fulfil a strict definition of cardiogenic shock as suggested by the U.S. Myocardial Infarction Research Unit programme (Buckley *et al.*, 1970; Scheidt *et al.*, 1973). Use of this definition necessitated intravascular catheterization and full trial of medical therapy, and entailed long delays in initiating counterpulsation. Such delays in the first 8

months were invariably associated with progressive and relentless deterioration so that death occurred during assessment or that cardiac assistance was initiated with patients in an agonal state. After the first 8 months the definition of shock was altered to enable more rapid evaluation and progression to mechanical assistance. The modified definition of shock required that systolic arterial pressure be less than 100 mmHg, the urine output be less than 25 ml/hour, the peripheral cutaneous circulation be definitely impaired, the left and right atrial pressures be increased, as gauged by clinical and radiographic evidence after correction of haemodynamically significant arrhythmias and after a trial of conventional medical therapy for at least one hour. Where any question of hypovolaemia existed, pulmonary artery pressure was measured directly and shown to be high (diastolic pressure greater than 16 mmHg in relation to mid-chest level in the axilla) before proceeding to counterpulsation. After the first 16 months it was decided to relax the criteria still further so that patients were accepted even in the absence of clinical shock, provided they had unequivocal evidence of severe heart failure refractory to medical therapy, plus at least one other major complication of infarction.

Of the 72 patients meeting the criteria of acute severe cardiac power failure, 19 were classified as being unsuitable for counterpulsation, because it was considered that extreme age or pre-existing disease would

FIG. 1 Age distribution of patients treated with counterpulsation (dark area) and patients dying before this could be started (light area).

FIG. 2 Total experience over the 22-month period.

limit full recovery even if mechanical assistance were completely successful. Another 12 patients could not be offered counterpulsation because the device was already in use.

Forty-one patients were accepted for arterial counterpulsation; 9 of these died before heart assistance could be initiated. In 2 patients the balloon catheter could not be passed from either femoral artery. Both died within hours of abandoning the cannulation attempt. Of the original 100 patients, 30 proceeded to counterpulsation. Ages of patients accepted for counterpulsation ranged from 33 to 64 years (average 54 years) (Fig. 1). Of these, 25 patients fulfilled the amended criteria of cardiogenic shock while five were not shocked.

Fig. 2 outlines referral experience over the 22-month period. After an initial wave of enthusiasm, the number of referrals fell off to 2 a month then gradually increased again to nearly 2 a week in October 1973. The ratio of

patients referred to patients proceeding to counterpulsation remained close to 3:1 over the whole period. Use of the device increased over the period of study (Fig. 3). During the past 4 months the single device was in operation 10 days a month on average, and an increasing number of patients referred could not be treated with counterpulsation because the device was in use.

Procedure

Arterial counterpulsation was performed with an Avco intra-aortic balloon system. The balloon catheter (with 30 or 40 ml displacement, depending on patient size) was inserted through the femoral artery and advanced to the thoracic aorta using the operative technique of Kantrowitz *et al.* (1968). Arterial pressure was monitored either through a catheter advanced from the brachial artery or through a cannula inserted in the

FIG. 3 Console use in hours per month over the 22-month period. Arrows indicate occasions when a patient with acute severe heart failure could not be offered counterpulsation because the device was already in use.

radial artery. Pulmonary artery pressure was monitored through a Swan-Ganz or Devices flow-directed catheter. Monitoring catheters were always used, and, when not inserted before, were passed during the femoral cannulation procedure.

Cardiac assistance was continued until the patient was no longer dependent on the device or until death supervened. However in 2 instances, assistance was discontinued earlier than desired either because of temporary malfunction of the unit or because circulation in the leg appeared to be seriously compromised. When terminated electively the duration of counterpulsation averaged 5.1 days.

Cardioangiography was performed in 21 patients. Indications for this assessment varied over the 22-month period. Initially we attempted to study every patient immediately after pumping had been started. At the end of the period we had elected to withhold angiography in patients who were progressing smoothly on counterpulsation alone, and in others to defer this in expectation of maximal improvement within 24 to 48 hours of pumping. Angiography was performed earlier of course if there was any suggestion of tamponade. In all cases angiography was performed during counterpulsation, and with a view to corrective cardiac surgery.

Fourteen patients proceeded to cardiac surgery, and

another had needle aspiration of the pericardial cavity. In all but 3 of these patients counterpulsation was continued in the postoperative phase for at least 2 days.

Results

Hospital mortality was high (Table 1), with 68 deaths in the 100 patients referred. As expected, there were fewer deaths in the group who did not fulfil the criteria of acute, severe cardiac failure. Most of these patients had suffered massive infarction and many had experienced other complications as well. Mortality rate at 43 per cent was three times our average for all admissions with myocardial infarction.

Mortality rate was extremely high, as expected, in patients with severe failure, but whose age or pre-existing disease precluded active mechanical intervention. In this group there was only one hospital survivor. This group obviously cannot be regarded as a control for those undergoing counterpulsation. A better group for comparison consisted of the 12 patients who met the criteria for mechanical assistance but to whom this could not be offered because the device was already in use. These patients were usually monitored and managed in the same way as those undergoing counterpulsation. Mortality rate in this group was 11/12 or 92 per cent. The nine deaths before counterpulsation in patients provisionally accepted for mechanical assistance are an indication of how quickly many in this group were deteriorating at the time counterpulsation was started.

Tables 2 and 3 give details of all patients who proceeded to counterpulsation. Ten of these had suffered at least one previous myocardial infarct and 4 had experienced chronic angina pectoris. Despite this all were active and most were gainfully employed. Site of the infarct was anterior in 20, inferior in 8, and both anterior and inferior in 2. The average time for evaluation was 6.8 hours for the first 6 shocked patients and 2.2 hours for the last 6. Of the 25 shocked patients, 6 had complete heart block before counterpulsation. Pacing was initiated in all, but 3 returned to sinus rhythm within 2 days of initiating counterpulsation. Angiography was performed in 19 of the 25 shocked patients and in 2 of the 5 patients in 'pre-shock'. Thirteen of the former and one of the latter proceeded to surgery. Hospital survival rate was 36 per cent (9 of 25 patients) in the shocked group and 100 per cent (5 of 5 patients) in the 'pre-shock' group.

Surgical experience is outlined in Table 4. The shocked group of patients was divided into a group with power failure exclusively and a group with some mechanical complication of infarction. Such complications included rupture of the interventric-

ular septum (3 patients), rupture of the free wall of the left ventricle (2 patients), rupture of a papillary muscle (1 patient), pericardial bleeding (1 patient), and left ventricular aneurysm (1 patient). Patients with power failure were selected for operation because at angiography patent distal vessels had been demonstrated beyond major proximal obstructions. These patients had aorto-coronary bypass only, whereas patients with other complications had corrective surgery, often in association with aorto-coronary bypass. Two of the patients with power failure and 4 of the patients with other complications survived.

Clinical features of patients with muscular disruption were very similar. These usually presented with acute heart failure and hypotension several days after the first symptoms of infarction, and often interrupting an otherwise uneventful convalescence. Particular difficulty was experienced in diagnosing patients with subacute heart rupture and haemopericardium. In these the most reliable features were the timing of deterioration after the first symptom of infarction, the presence of further pain, and the severity of right-sided heart failure (Windsor *et al.*, 1973; O'Rourke, 1973). One patient had none of the classical features of pericardial tamponade and was diagnosed only at exploratory surgery.

In patients with haemopericardium we found counterpulsation effective as a supportive measure during diagnostic study, during preparation for surgery, and postoperatively. This is illustrated in the patient diagnosed at exploratory surgery who remained in a state of apparently irreversible shock despite two procedures for pericardial drainage (Fig. 4). After complete relief of tamponade this patient was comatose, hemiparetic, and totally anuric, with little or no response to inotropic and vasopressor agents. She was discharged 7 weeks after this episode on no treatment, with normal renal and cerebral function, has remained well, and is fully employed.

Of the 30 patients, 16 died despite counterpulsation. Necropsy was performed in 15. All had massive infarction and extensive coronary artery disease (Table 5), and this was obviously the prime determinant of death. Nevertheless we considered that errors in technique or judgement may have contributed to death in 5. One patient had subacute heart rupture and died suddenly from tamponade several hours after pericardiocentesis; we believe he should have had open drainage and infarctectomy. One patient was not paced effectively. One died with renal failure contributed to by aortic dissection which was accidentally induced during institution of cardiopulmonary bypass. Two patients

TABLE 2 Patient data – cardiac failure and shock

Case No.	Age (yr)	Previous cardiovascular disease	Site of infarct	Onset shock → C/P (hr)	Period of evaluation before C/P (hr)	Period on C/P (hr)	Duration in hospital (dy)
1	56	—	Ant.	18	4	11	1
2	49	—	Ant.	12	6	13	1
3	63	MI × 2	Ant.	12	11	46	2
4	48	MI	Ant.	7	8	4	1
5	60	—	Inf.	11	7	4	1
6	57	Mild hypertension	Ant.	8	5	1	1
7	60	MI	Ant.	4	3	124	31
8	46	—	Ant. and inf.	4	1	180	42
9	64	Hypertension	Ant.	4	42	171	47
10	37	—	Ant.	9	1	42	2
11	64	MI	Ant.	8	5	78	5
12	63	—	Inferolat.	6	2	17	27
13	48	Venous thrombosis	Inferolat.	7	3	27 + 93	60
14	52	Hypertension	Inf.	13	2	108	41
15	63	Angina	Ant.	6	1	44	25
16	57	—	Lat.	6	1	115	20
17	55	MI × 3	Inf.	8	2	1	1
18	45	Hypertension	Ant.	10	1	100	4
19	51	Angina	Inferolat.	6	3	135	24
20	63	MI	Ant.	24	3	113	5
21	64	Angina	Ant.	6	2	186	68
22	58	Mild hypertension	Ant.	4	1	66	3
23	57	Hypertension	Inf.	4	2	12	6
24	52	MI	Inf.	4	3	89	4
25	53	MI	Ant. and inf.	7	4	138	26

MI = myocardial infarction.

C/P = Counterpulsation

TABLE 3 Patient data – cardiac failure without shock

Case No.	Age	Previous cardiovascular disease	Site of infarct	Complications	Onset symptoms → C/P (hr)	Period on C/P	Duration in hospital (dy)
1	37	—	Anterior	Refractory pulm. oedema, sinus tachycardia to 120/min, recurrent cardiac pain	52	144	27
2	45	MI × 2	Anterior	Severe LV failure, recurrent VT and VF	77	132	30
3	64	MI × 1; hypertension	Anterior	Severe LV failure, recurrent cardiac pain, runs of malignant ventricular ectopics	22	138	21
4	54	—	Anterior	Pulm. oedema, progressive sinus tachycardia to 120/min, hypotension	84	60	24
5	46	Angina	Anterior	Refractory pulmonary oedema, progressive sinus tachycardia to 130/min, hypotension	33	186	30

Other therapy	Angio graphy	Surgery	Survival
—	✓	—	—
Pacing	✓	✓	—
Pacing	✓	—	—
—	✓	—	—
Pacing	✓	—	—
—	—	—	✓
—	✓	✓	—
—	✓	—	—
Pacing	—	—	—
—	✓	✓	✓
—	✓	✓	✓
—	✓	✓	✓
—	✓	✓	—
Attempted pacing	—	—	—
—	✓	—	—
—	✓	✓	✓
—	✓	✓	—
—	✓	✓	✓
—	✓	✓	—
—	✓	✓	—
—	✓	✓	—
—	—	—	✓

Other therapy	Angio-graphy	Surgery	Survival
—	—	—	✓
—	✓	✓	✓
—	✓	—	✓
—	—	—	✓
—	—	—	✓

died with resistant abnormalities in cardiac rhythm and unacceptably high (1.6 and 3.1 ng/nl) serum digoxin concentrations (Carroll *et al.*, 1973; Morrison and Killip, 1973).

Of the 30 patients undergoing counterpulsation, 6 developed clinical features of digitalis toxicity in association with high serum levels of digoxin at some time during the hospital course despite adjustment of drug dosage with altered renal function. We were led to suspect that hypotensive patients had an unusual circulatory distribution of digoxin, and have come to use this drug sparingly or not at all.

There were few complications of counterpulsation and none were life-threatening. One patient developed ischaemia of the leg which was severe enough and sufficiently persistent to warrant early removal of the balloon catheter. Fogarty catheters were used to extract thrombus in the femoral artery of 5 patients when the catheter was removed. One patient had a wound infection in the groin. There were no long-term complications whatever either from the femoral artery balloon catheter or from the monitoring catheters. Our most serious complication of counterpulsation was in a patient (not included in this series) who could not be weaned from cardiopulmonary bypass after aortic valve replacement. This patient developed a small aortic dissection originating at a tear in the aortic arch. It is believed that this may have been induced by headward movement of the catheter tip when the patient later sat up in bed (O'Rourke and Shepherd, 1973). The dissection was an incidental finding at necropsy.

Survivor status is outlined in Table 6. Fourteen patients left hospital. There have been 2 late deaths, one with chronic cardiac failure 12 months after initiation of support and one with proven reinfarction 9 days after discharge. All other patients are ambulant and the majority have returned or plan shortly to return to work or previous activities.

The results of counterpulsation in patients with 'preshock' were particularly pleasing. There were no hospital deaths in this group (though one patient died 9 days after discharge with reinfarction during an uneventful convalescence). In these patients prognostic indices (Peel *et al.*, 1962; Norris *et al.*, 1969) predicted mortality rates of 60 per cent and 59 per cent, respectively. Counterpulsation was associated with immediate and lasting relief of ischaemic pain, when present, with decrease in frequency of ventricular arrhythmias in the first few hours, with relief of pulmonary oedema within hours and clearing within days, with decrease in diuretic requirements, and improvement in general well-being. Improvement was main-

TABLE 4 *Surgical experience*

Patient	Age (yr)	Complication	Operative procedure	Outcome
<i>Group A. Cardiogenic shock</i>		(i) <i>Power failure</i>		
2	49		Attempted CAG	Death
15	63		CAG × 3	Death
19	51		CAG × 3	Survival
21	64		CAG × 3	Survival
22	58		CAG × 3	Death
24	52		CAG × 2	Death
		(ii) <i>Mechanical complication</i>		
1	56	Subacute heart rupture	Pericardiocentesis	Death
9	64	VSD	Infarctectomy; patch VSD; CAG × 1	Death
12	63	Subacute heart rupture	Pericardial drainage; infarctectomy	Survival
13	48	Haemopericardium	Pericardial drainage	Survival
14	52	VSD	Infarctectomy; patch VSD; CAG × 1	Survival
16	57	LV aneurysm	Excision LV aneurysm; CAG × 1 and endarterectomy	Survival
20	63	VSD	Infarctectomy; patch VSD	Death
23	57	Rupture papillary muscle	Replace mitral valve	Death
<i>Group B. 'Pre-shock'</i>				
2	45		CAG × 2	Survival

CAG = aorto-coronary bypass graft (1, 2, or 3 grafts.)

tained after counterpulsation was discontinued, and all patients left hospital with little or no evidence of cardiac failure on treatment. The 4 living patients have returned to previous activities.

Discussion

This paper reports the total experience in a general metropolitan hospital after initiation of a programme of counterpulsation (and cardiac surgery) for acute severe cardiac failure complicating myocardial infarction.

As reported by others (Motteram and Buchanan, 1967; Scheidt, Ascheim, and Killip, 1970; Mueller *et al.*, 1972) the mortality rate with conventional treatment was extremely high, there being only 2 survivors in 33 patients. This survival rate of 6 per cent contrasts with the survival rate of 36 per cent for patients with the clinical syndrome of cardiogenic shock and 47 per cent for the whole group with cardiac failure, treated with counterpulsation. These figures suggest that counterpulsation was an important therapeutic tool, and this is our interpretation. We must point out, however, that no attempt was made to randomize patient selection for counterpulsation, and we did decline to offer counterpulsation to patients over 65 years of age and to patients with severe pre-existing disease. Patients presenting when the device was already in use were the closest to a control group for the

patients treated with counterpulsation. In this group of 12 patients there was only one survivor.

Our results with counterpulsation are similar to those reported by Buckley and colleagues at the Massachusetts General Hospital (Leinbach *et al.*, 1973b). Like this group we have combined arterial counterpulsation with cardiac surgery, so that it is impossible to separate the individual value of each. Our survival rate for patients in shock is 36 per cent which is similar to that reported from Boston (Leinbach *et al.*, 1973b; M. J. Buckley, 1973, personal communication) and our excellent survival in non-shocked patients corresponds to similar results reported by the same group in patients with recurrent ischaemia after infarction (Leinbach *et al.*, 1973a; M. J. Buckley, 1973, personal communication). We have been able to show, however, that such results are possible outside a highly subsidized research unit. The results presented here are better than those reported by Scheidt and colleagues (1973) in another larger series. This probably is because of different criteria of acceptance, with counterpulsation being introduced at an earlier stage, and counterpulsation being combined with cardiac surgery.

With one exception we remain uncertain about the role of cardiac surgery with counterpulsation. Our experience includes 3 patients with haemopericardium (2 caused by subacute heart rupture) pre-

FIG. 4 Hospital course from the 4th until the 14th day of a patient with myocardial infarction complicated on the fifth day by acute haemopericardium which simulated cardiogenic shock. Lasix = frusemide, N. adren = noradrenaline, Adren = adrenaline, Iso = isoprenaline, P. dialysis = peritoneal dialysis, C'P, C'Pulse = counterpulsation.

senting as cardiogenic shock. We believe this must be considered a strong possibility especially in patients who deteriorate suddenly 48 hours or more after the first symptoms of infarction, with further pain and prominent right heart failure, even if other signs of tamponade are absent. We believe that open exploration, not pericardiocentesis, is the only logical definitive treatment of this condition. Krakauer *et al.* (1971) found heart rupture in 7 of 21 patients dying of cardiogenic shock despite counterpulsation and considered this to be a terminal event. We had no deaths caused by terminal heart rupture except for one patient whose hypotensive state was obviously caused by haemopericardium following an earlier non-lethal rupture into the pericardial sac. Our experience has shown that subacute heart rupture can be elusive of diagnosis and can mimic cardiogenic shock. Our 2 survivors

from haemopericardium had suffered relatively small infarcts and have now returned to all previous activities.

We have been somewhat disappointed with revascularization surgery when used alone, but were impressed that this was of value when combined with aneurysmectomy or closure of ventricular septal defect. We continue to use the same criteria for surgical acceptance as proposed by the Harvard group (Leinbach *et al.*, 1973b) but we are perhaps less optimistic about revascularization when the ventricle is dilated and a large component of the wall is completely akinetic.

We have no qualms about the safety of counterpulsation. The complications we have experienced have been relatively minor and there have been no long-term sequelae. Our most serious complication was a small aortic dissection, apparently induced by

TABLE 5 Hospital deaths

Case No.	Age (yr)	Pre-existing disease	Condition before C/P			Duration of C/P (hr)	Surgical procedure
			BP (mmHg) (kPa)	Urine (ml/hr)	Cerebral status		
1	56	—	80/70 <i>10.6/9.3</i>	< 10	Obtunded	11	Pericardial aspiration
2	49	Angina	65/50 <i>8.6/6.6</i>	Nil	Obtunded	13	Attempt aorto-coronary graft
3	63	Myocardial infarcts; peripheral vascular disease	100/60 <i>13.3/7.9</i>	7	Obtunded	46	Nil
4	48	Myocardial infarct	95/75 <i>12.6/9.9</i>	3	Obtunded	4	Nil
5	60	—	55/40 <i>7.3/5.3</i>	5	Obtunded	4	Nil
6	57	Cardiac failure; minor stroke; peripheral vascular disease	50/40 <i>6.6/5.3</i>	Nil	Obtunded	1	Nil
9	64	Hypertension	80/65 <i>10.6/8.6</i>	16	Clouded	170	Closure VSD
10	37	Angina	70/50 <i>9.3/6.6</i>	20	Clouded	43	Nil
11	64	Myocardial infarct; angina; diabetes mellitus	80/55 <i>10.6/7.3</i>	< 10	Clouded	78	Nil
15	63	—	90/70 <i>11.9/9.3</i>	20	Normal	50	Aorto-coronary bypass
17	55	Myocardial infarct; angina	100/70 <i>13.3/9.3</i>	25	Clouded	< 1	Nil
18	45	Angina pectoris; renal insufficiency; severe hypertension	60 systolic <i>7.9</i>	Nil	Obtunded	100	Nil
20	63	Myocardial infarct	75/60 <i>9.9/7.9</i>	20	Normal	113	Closure VSD
22	58	Hypertension; angina	70 systolic <i>9.3</i>	< 10	Normal	66	Aorto-coronary bypass
24	52	Myocardial infarct	90/60 <i>11.9/7.9</i>	< 10	Clouded	88	Aorto-coronary bypass
23	57	Hypertension	90/75 <i>11.9/9.9</i>	8	Clouded	11	Mitral valve replacement

● = complete occlusion.

+ = high grade obstruction.

Note: Figures in italics are blood pressure in kPa units.

Complication	Mode of death	Day of death	Necropsy findings coronary arteries			
			L main	L circ.	LAD	R
Haemopericardium; rupt. ventricle	Tamponade	2		+	●	+
Heart block	Power failure	2			●	●
Heart block	Power failure	3		+	●	+
—	Power failure	2		+	●	●
Heart block	Power failure	2		+	●	●
Heart block	Power failure	1			●	+
Breakdown of repair of VSD	Power failure; resp. infection	48			●	+
—	Power failure	3		+	●	+
—	Power failure	4		Necropsy refused		
—	Power failure	26		+	+	●
Nodal bradycardia, heart block (pacing unsuccessful)	Power failure; asystole	2		●	+	●
Renal failure	Power failure	5		+	●	
Renal failure postop. Digitalis toxicity	Power failure	6			●	+
	Unable to wean cardio- pulmonary bypass	4	+	+	+	+
Digitalis toxicity; unexpected vent. fibrillation	Power failure; ventricular fibrillation	5		+	+	●
Aortic dissection with cardio-pulmonary bypass; renal failure	Power failure; renal failure	6			+	+

TABLE 6 *Survivor status*

<i>Patient</i>	<i>Age (yr)</i>	<i>Duration C/P (hr)</i>	<i>Operative procedure</i>	<i>Cardiovascular status</i>	<i>Time of assessment (months after infarct)</i>
<i>Group A. Cardiogenic shock</i>					
(i) Power failure					
7	60	124	Nil	Disabled; death 12 months later, chronic cardiac failure	
8	46	180	Nil	Active; cardiac failure; moderately disabled	14
19	51	135	CAG × 3	Active; mild controlled cardiac failure	6
21	64	186	CAG × 3	Moderately disabled; cardiac failure	5
25	53	138	Nil	Active; mild controlled cardiac failure	2
(ii) Mechanical complication					
12	63	17	Pericardial drainage; infarctectomy	No disability, no therapy	11
13	48	27 + 93	Pericardial drainage	No disability, no therapy	11
14	52	108	Closure ventricular septal defect; infarctectomy; CAG × 1	Cardiac failure controlled, moderate disability	11
16	57	115	Excision aneurysm; CAG × 1	Active; no disability, mild controlled cardiac failure	9
<i>Group B. 'Pre-shock'</i>					
1	37	144	Nil	Mild angina; mild controlled cardiac failure; cardiac neurosis	8
2	45	132	CAG × 2	Active; mild controlled cardiac failure	6
3	64	138	Nil	Active; mild controlled cardiac failure	5
4	54	60	Nil	Sudden death, proven fresh infarct during smooth convalescence	1.5
5	46	186	Nil	Active; mild controlled cardiac failure	3

CAG = Aorto-coronary bypass graft.

abrasion of the aortic arch by the catheter tip during alteration in body position. This is preventable (O'Rourke and Shepherd, 1973) and we now deliberately locate the tip of the catheter at least 4 cm below the top of the arch. The published reports record only two lethal complications with intra-aortic balloon counterpulsation. Neither of these should recur with the experience that has already been gained.

The results presented here support the view that counterpulsation and cardiac surgery have a definite role in management of the mechanical complications of myocardial infarction. The precise role of each remains to be delineated.

It appears to be a typical example of medical conservatism that, where a mass of theoretical, experimental, and clinical evidence points to counterpulsation having an important preventive role in limiting infarct size, its use to date has been directed almost exclusively to patients with widespread established necrosis. We consider this illogi-

cal, especially when the procedure can be applied safely and relatively simply.

We believe that the role of counterpulsation in preventing death and limiting disability after infarction will best be demonstrated in a controlled trial with patients less ill than any reported here. We believe that such a trial is now justifiable.

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