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Intra aortic balloon pumping in myocardial infarction and unstable angina

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From 1972 to 1979 intra aortic balloon pumping (IABP) was attempted in 181 patients; catheter insertion failed in 13 (8%). More complications occurred with prolonged treatment but all three lethal complications (2%) were related to catheter insertion.

Seventy-six patients had clinical cardiogenic shock after myocardial infarction (CSMI). Haemodynamically, 23 were classified as preshock: 15 (66%) could be weaned, 12 (53%) survived over 3 months; whereas only 27/51 patients (51%) haemodynamically classified as shock could be weaned and 21 (40%) survived over 3 months.

Of forty-two patients with refractory angina at rest, 41 had prompt relief of pain after IABP, and subsequently underwent coronary artery bypasss grafting (CABG). Perioperative infarction rate was 8% (4/41), perioperative mortality was 7% (3/41). Total infarction rate was 11% (5/42), and total mortality 7% (3/41).

Pain relief was prompt in 14/17 patients (82%) with refractory angina after infarction. Pain persisted in three patients: all three sustained an infarction, one died. Two patients were excluded from surgery. Twelve patients underwent CABG; none died, none developed MI.

In eight patients persistence of pain suggested a slowly evolving MI, IABP abolished pain in seven.

Conclusion: IABP has demonstrated its efficacy both in pump failure and in refractory ischaemia. However, its use is not without risks.

The effect of intra aortic balloon pumping (IABP) is twofold[1]. The abrupt presystolic balloon deflation decreases afterload, while the post systolic balloon inflation enhances coronary perfusion pressure, which is the major determinant of coronary blood flow in the presence of critical stenosis[2, 3].

Reduction of afterload increases stroke volume and ejection fraction, and decreases wall tension and preload, and thus oxygen consumption. It has even been suggested, that counterpulsation may open dormant collateral channels[1], and that IABP increases flow to the subendocardium by augmenting existing collateral circulation[1].

Afterload reduction reduces oxygen demand; diastolic augmentation increases oxygen availability. The ratio of the Diastolic Pressure Time Index (DPTI) representing myocardial blood flow[1], and the Systolic Time Tension Index (TTI), which is related to myocardial oxygen consumption[1, 4] is termed the endocardial viability ratio (EVR)[1]. EVR has been regarded a reflection of this balance between oxygen availability and consumption.

The clinical use of IABP has been advocated for cardiogenic shock following acute myocardial infarction (CSMI) and severe left ventricular failure (LVF) after acute infarction[5–14], with and without mechanical defects, for angina pectoris at rest completely refractory to medical therapy[15, 16], for severe LVF and cardiopulmonary bypass dependence following open heart surgery[17], for LVF of varying aetiological background[18], for life threatening cardiac arrhythmias refractory to antiarrhythmic therapy, and as a supportive measure in critically ill cardiac patients undergoing major surgery[19].

Coronary care has entered the era of infarct size limitation. Recognizing the deleterious effect of a

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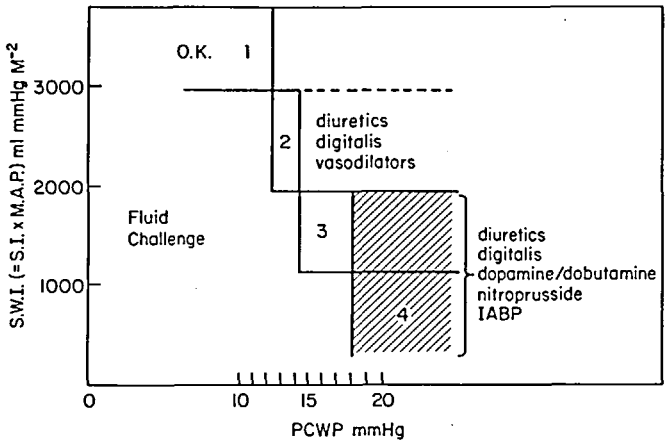

Figure 1 Myocardial Infarction Research Unit (MIRU) classification.

first transmural anterior myocardial infarction on left ventricular function, an interesting but uncontrolled pilot observation was recently published by Leinbach *et al.*[20, 21], who applied the IABP to patients with acute anterior infarctions without cardiogenic shock.

It is the purpose of this report to present the experience with IABP from the coronary care unit of the Thoraxcenter, Rotterdam, over the period 1972–1979.

The indications for IABP have been: CSMI and severe LVF following acute myocardial infarction, angina at rest completely refractory to medical therapy, post infarct (<2 weeks) angina at rest refractory to medical therapy and the suspicion of a slowly evolving myocardial infarction. The IABP was also used for other indications in our institution: cardiopulmonary bypass dependence following open heart surgery, intractable LVF in acute viral myocarditis or in septic shock and in patients with large ventricular aneurysms, in whom cardiac surgery was contemplated. These indications will not be discussed here.

Methods

A three-lumen AVCOthane 40 or 20 cm^3 balloon catheter was introduced into the femoral artery with a side arm graft[22], or later through a transverse incision with sutures directly around the catheter, and connected with an AVCO 7 or 10 IABP console. All patients were monitored with Swan Ganz thermodilution catheters[23] and, in cases of hypertension or hypotension an arterial line in the left radial artery. All patients with indwelling catheters were anticoagulated with Heparin (20·000–30·000 units i.v. 24 h^{-1}).

DEFINITIONS AND APPROACH

(1) Cardiogenic shock and severe left ventricular failure following acute myocardial infarction

In 1972 we adopted the original Myocardial Infarction Research Unit (MIRU) classification for left ventricular function as suggested by Leinbach *et al.*[6] (Fig. 1). Originally, patients in CSMI MIRU class IV were candidates for treatment with IABP. However, it soon became apparent that the majority of patients with pulmonary capillary wedge pressures (PCWP) greater than 18 mmHg and stroke work index (S.W.I.=the product of stroke index, S.I., and mean arterial blood pressure, M.A.P.) between 1200 and 2000 ml mmHg M^{-2} rapidly progressed into MIRU class IV with medical therapy alone. Most progressed within one day, and required treatment with IABP. On the other hand, patients who had a PCWP lower than 18 mmHg associated with a S.W.I. below 2000 ml mmHg M^{-2} benefitted from medical therapy and from i.v. volume expanders, and did not require IABP.

These observations caused us to extend our indication for IABP (Fig. 1, hatched area), to all patients who had a PCWP greater than 18 mmHg and a S.W.I. lower than 2000 ml mmHg M^{-2}, together with the clinical signs of left ventricular failure (low blood pressure, cool clammy skin, mental dullness, pulmonary rales and urinary production <40 ml h^{-1}). Additional medical therapy in these patients and in those with shock

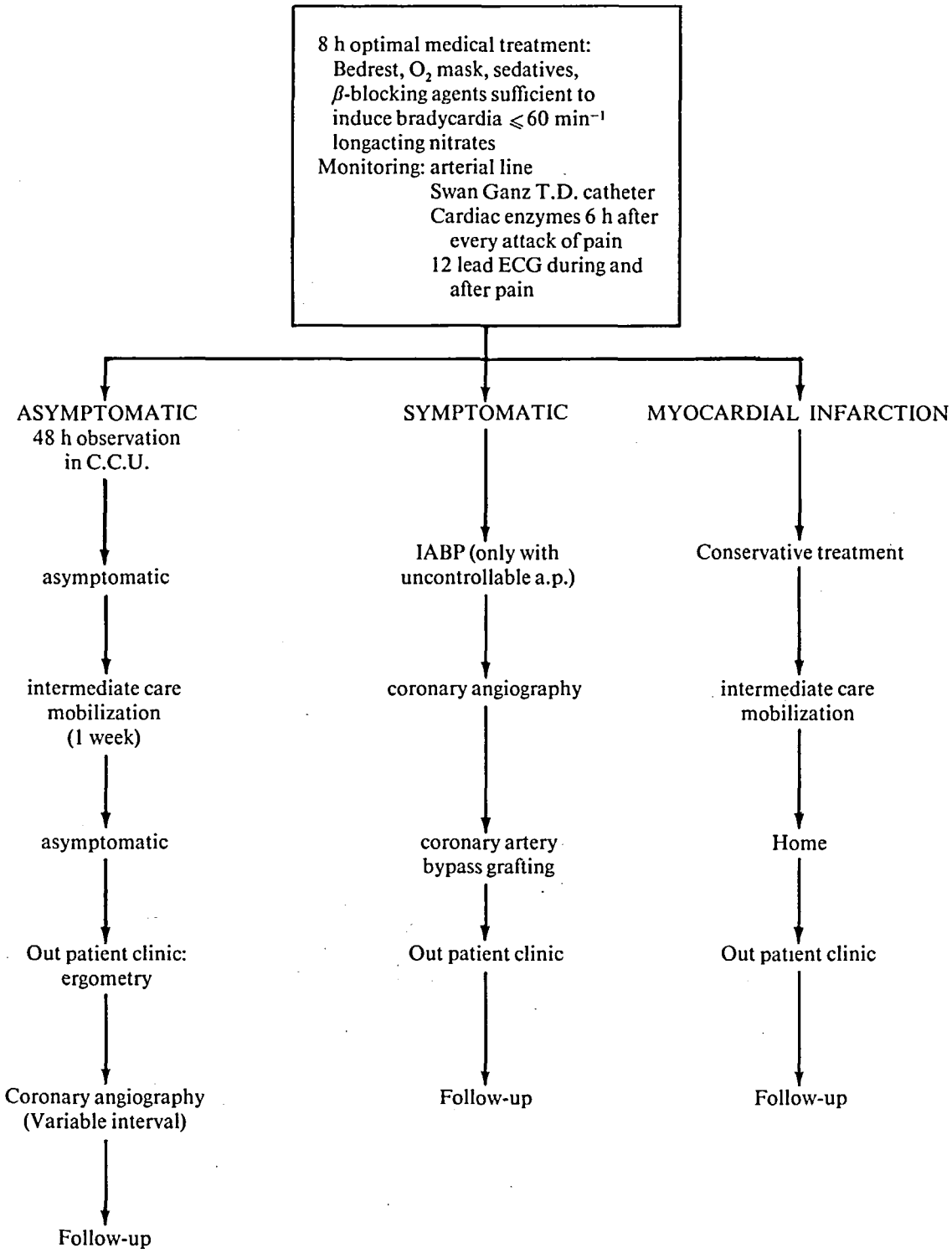

Figure 2 Approach in angina at rest.

(MIRU class IV) consisted of digitalis, diuretics, dopamine or dobutamine and nitroprusside. The therapy with IABP was maintained until a haemodynamically stable condition had been reached with the patient on oral medication alone.

After haemodynamic stability had been achieved, left ventricular angiography and coronary arteriography were performed. Surgery was undertaken only for reparable mechanical lesions.

The weaning from IABP consisted of a stepwise reduction in the ratio of assisted to not assisted beats, at 4 hourly intervals (1:2, 1:4, 1:8), with subsequent removal of IABP in the absence of haemodynamic deterioration.

(2) Unstable angina pectoris, with ischaemic attacks at rest (Fig. 2)

Patients with angina at rest with transient ST or T wave changes but without significant cardiac enzyme elevation or new Q wave formation, were kept at bedrest in the coronary care unit.

Haemodynamic monitoring during and after pain included heart rate, systemic blood pressure, pulmonary capillary wedge pressure or diastolic pulmonary artery pressure and cardiac output. During and following every attack of pain a twelve lead ECG was registered and, with persisting pain, repeated at 15 min intervals. Six hours after every attack of pain the cardiac enzymes (CPK, CPK-MB, α-HBDH) were determined. After a sedative, preferably diazepam (5–10 mg orally 8 h^{-1}), isosorbide dinitrate (5 mg sublingually every 2 h) was given and the i.v. administration of propranolol (1–10 mg, at $\leqslant$ 1 mg min^{-1}) started. Propranolol was given until a heart rate of $\leqslant$ 60 min^{-1} had been reached. An oral maintenance dosage (40–800 mg 24 h^{-1}) was then instituted. When propranolol was contra-indicated because of lung disease a more selective β-blocker might be chosen. In later cases we routinely added the new calcium antagonist, nifedipine, when β-blockers alone failed to control angina at rest.

If the patient was still symptomatic after 8 h of medical therapy, treatment was considered unsuccessful. For another 0–16 h intensive medical therapy was given but he was a candidate for emergency coronary angiography and coronary artery bypass grafting (CABG). When despite medical therapy including i.v. nitroglycerin breakthrough angina occurred, the IABP was introduced. Directly following CABG the IABP was removed unless there were indications for continuation. Patients with post infarct angina at rest were treated essentially in the same fashion. We did not recommend urgent coronary artery bypass grafting for patients cooled off by medical means, although many of these may require elective surgery sooner rather than later, depending on the extent of their coronary artery disease[25]. Patients who developed an acute infarction despite these measures were treated conservatively. We discontinued propranolol only if cardiac or pulmonary problems arose from its use.

(3) Slowly evolving myocardial infarction

Occasionally one is confronted with patients who have persisting ischaemic pain more than 12 h after their myocardial infarction, refractory to medical therapy, but without new ECG-changes. These patients may have mild left ventricular failure (MIRU class II) or frequent ventricular arrhythmias suggesting ventricular irritability due to prolonged ischaemia. In this group we considered the IABP the definitive therapeutic measure to reduce ischaemic pain, and we did not consider coronary artery bypass grafting.

Results (Table 1)

From September, 1972 to March 1979, 168 patients with unstable angina or myocardial infarction were treated with the IABP. In 13 more patients, introduction of the balloon catheter failed (8%). Of the total of 168 patients, 50 (29%) died during treatment, 16 died within three months (10%), and 102 (61%) survived over three months. Duration of treatment with IABP was less than five days in 87 patients, 5–10 days in 34 patients and exceeded ten days in 47 patients. Forty-six complications occurred in 168 patients: thrombocytopaenia, which was a common feature in patients pumped over five days, never caused trouble, and was therefore not considered to be a complication. Prolonged use of the IABP was associated with more complications. All lethal complications however, were related to balloon catheter insertion and no fatal complications resulted from prolonged treatment with the IABP (up to 46 days). In 3/168 (2%) patients death was attributable to the IABP: in two patients with CSMI, dying within 24 h, autopsy revealed dissection of the aorta. Trapping of gas in a kinked balloon may have contributed to death in one patient who died during CABG.

Two more dissections were encountered: one patient with CSMI died of intractable arrhythmias nine days after insertion of the balloon catheter.

Table 1 Results IABP 1972–1979 (168 patients)

Indication	Duration IABP	Deaths on IABP	Deaths <3 months	Survival >3 months	Total
CSMI	<5 days	29	1	2	32
	5–10 days	4	3	8	15
	>10 days	1	5	23	29
	Total	34	9	33	76
Angina at rest despite medical treatment	<5 days	—	3	27	30
	5–10 days	—	—	8	8
	>10 days	—	—	4	4
	Total	—	3	39	42
Post infarct angina at rest despite medical treatment	<5 days	—	—	5	5
	5–10 days	—	1	5	6
	>10 days	1	—	5	6
	Total	1	1	15	17
Slowly evolving myocardial infarction	<5 days	—	—	1	1
	5–10 days	—	—	4	4
	>10 days	—	—	3	3
	Total	—	—	8	8
Postoperative CABG+ aneurysmectomy	<5 days	5	—	1	6
	5–10 days	—	—	—	—
	>10 days	—	—	3	3
	Total	5	—	4	9
Postoperative valve replacement	<5 days	5	1	2	8
	5–10 days	—	—	—	—
	>10 days	—	—	—	—
	Total	5	1	2	8
Miscellaneous	<5 days	3	1	1	5
	5–10 days	1	—	—	1
	>10 days	1	—	1	2
	Total	5	1	2	8
Total		50	15	103	168

Failures to insert balloon catheter: 13.

Autopsy showed entrance of the catheter into a false medial lumen, a few centimeters above the bifurcation. The catheter did not re-enter the aortic lumen; no loss of blood sufficient to account for death could be demonstrated. Angiography revealed an intimal flap in the descending thoracic aorta in one more patient analyzed after two years. Emboli in the femoral arteries could always be extracted with a size 6 Fogarty catheter.

CARDIOGENIC SHOCK AND SEVERE LEFT VENTRICULAR FAILURE (TABLE 2)

Seventy-six patients (34–71 years, mean 55 years; 64 males and 12 females) were treated with IABP. Sixty-two out of 76 (81%) had an acute anterior myocardial infarction, 11/76 (15%) had an acute inferior infarction, and 3/76 (4%) had ECG-changes suggesting both anterior and inferior infarction. Forty-two patients (55%) could be weaned but nine (12%) died within three months of intractable LVF or ventricular arrhythmias. Thirty-three patients (43%) survived over three months; 29 of these are still alive. The majority (21/29, 72%) require drugs, but lead active lives (New York Heart Association class I–II). Of the 76 patients, 23 had the clinical symptoms of shock, associated with the haemodynamic criteria of severe pump failure (PCWP $\geqslant$ 18 mmHg, and S.W.I. 1200–2000 ml mmHg M^{-2}, MIRU class III–

Table 2 Seventy-six patients (34–71 years, mean 55 years): 62 AMI; 3 AMI+PMI; 11 PMI

Total	Deaths on IABP	Deaths <3 months	Survivors >3 months
76 patients	34 patients	9 patients	33 patients
64 ♂	30 ♂	6 ♂	28 ♂
12 ♀	4 ♀	3 ♀	5 ♀
52 <60 years (68%)	20 <60 years (58%)	5 <60 years (55%)	27 <60 years (81%)
28 CAD >1 year (36%)	18 CAD >1 year (52%)	3 CAD >1 year (33%)	7 CAD >1 year (21%)
36 IABP delay ⩽6 h (47%)	8 IABP delay ⩽6 h (23%)	2 IABP delay ⩽6 h (22%)	26 IABP delay ⩽6 h (78%)

Table 3 Age, IABP delay, CAD >1 year

	IABP Delay						
	⩽6 h			>6 h			
Age	S	Deaths on IABP	Deaths on <3 months	S	Deaths on IABP	Deaths on <3 months	Total
<60 years	20(4)	4(3)	2	7(2)	16(5)	3	52(14)
>60 years	6(1)	4(3)			10(7)	4(3)	24(14)
Total	26(5)	8(6)	2	7(2)	26(12)	7(3)	76(28)

IV). Fifteen (66%) of these patients could be weaned and 12 (53%) survived over three months. In contrast, of the 53 patients who had clinical and haemodynamic cardiogenic shock (PCWP ⩾18 mmHg and S.W.I. <1200 ml mmHg M^{-2}, MIRU class IV), 27 (51%) could be weaned and only 21 (40%) survived over three months.

Since these two groups differed only with respect to S.W.I., they will not be separated in the subsequent description.

In six patients with acute mechanical defects and intractable heart failure, surgery was performed. Only two of the six patients survived over one year. One required aneurysmectomy and closure of ventricular septal defect. The other patient had a papillary muscle rupture associated with acute inferior infarction and required mitral valve replacement. In both patients surgery could be postponed for six weeks after the acute event. In the four patients who died, surgery was performed at an earlier stage, all four required aneurysmectomy, and in two mitral regurgitation due to geometrical LV changes necessitated valve replacement.

Patients who survived over three months were generally young (27/33, 81% <60 years). Only a small minority (7/33, 21%) had a history of longstanding coronary artery disease, and the majority was treated very promptly with the IABP: 26/33 (78%) were treated with IABP within 6 h of onset of left ventricular failure (Table 2).

These apparent determinants of outcome: age, delay of therapy with the IABP and the presence of longstanding coronary artery disease (CAD >1 year), with documented previous infarctions and/or angina pectoris were related to survival (Table 3): of the patients younger than 60 years, 20/52 (38%) died during treatment with IABP, 5/52 (10%) died within three months, and 27/52 (52%) survived over three months. Of the patients older than 60 years, 14/24 (58%) died during therapy, 4/14 (28%) died within three months and only 6/24 (25%) survived over three months.

Of the 36 patients treated with the IABP within 6 h following onset of shock, eight (22%) died during therapy, two (6%) died within three months and 26 (72%) survived over three months.

Of the 40 patients in whom treatment with IABP was delayed for over 6 h, 26 (66%) died during treatment, seven (17%) died within three months and only seven (17%) survived over three months.

Twenty-eight patients had a longstanding history of coronary artery disease: 18/28 (64%) died during treatment, 3/28 (11%) died within three months, and 7/28 (25%) survived over three months.

The subset of patients younger than 60 years, with IABP instituted within 6 h had a favourable

Table 4 Angina at rest despite medical treatment

No. vessels obstructed	Total patients	IABP efficacy	MI on IABP	Deaths on IABP	Deaths on CABG	MI during CABG	Deaths during CABG	MI <1 month	Deaths <1 month
LMCA	1	1	—	—	1	—	—	—	—
3 V	19	18	1	—	18	2	1	2	2
2 V	14	14	—	—	14	1	—	—	—
1 V LAD	8	8	—	—	8	—	—	—	—
LCX	—	—	—	—	—	—	—	—	—
RCA	—	—	—	—	—	—	—	—	—
Total	42	41	1	—	41	3	1	2	2

Table 5 Post infarct angina at rest despite medical treatment

No. vessels obstructed	Total patients	IABP efficacy	MI on IABP	Deaths on IABP	Deaths on CABG	MI during CABG	Deaths during CABG	MI <1 month	Deaths <1 month
LMCA									
3 V	8	7	1	1	6	—	—	1	1
2 V	7	6	1	—	5	—	—	—	—
1 V LAD	2	1	1	—	1	—	—	—	—
LCx	—	—	—	—	—	—	—	—	—
RCA	—	—	—	—	—	—	—	—	—
Total	17	14	3	—	12	—	—	1	1

outcome. Of the 26 patients with this fortuitous combination 20 (76%) survived over three months, and of those dying during treatment 3/4 (75%) had longstanding coronary artery disease. All patients older than 60 years with an IABP delay exceeding 6 h died within three months. However 6/10 (60%) patients older than 60 years treated within 6 h survived over three months.

ANGINA AT REST REFRACTORY TO MAXIMAL MEDICAL THERAPY (TABLES 1, 4 AND 5)

Fifty-nine patients with this syndrome were treated with the IABP. Forty-two had angina at rest either of new onset or superimposed on previously stable angina. Seventeen patients had angina at rest, within two weeks following AMI (post infarct angina at rest). These will be discussed separately.

Pain relief was prompt in 41/42 (98%) patients (35–65 years, mean 62 years) after institution of IABP. In one patient, a 65 year old male, with longstanding three vessel CAD, symptoms worsened. This patient developed a non fatal AMI, and was no longer a candidate for CABG.

Cardiac catheterization showed left main coronary artery (LMCA) obstruction in one patient (3%), three vessel obstruction in 19 (46%), two vessel obstruction in 14 (34%) and single vessel obstruction (all proximal obstruction of the LAD) in eight patients (19%). Forty-two patients underwent CABG. Two patients (4%) had an AMI during surgery; in one of these infarction and subsequent death may have been attributable to trapping of gas in a kinked balloon (see complications, Table 8).

Myocardial infarction rate for the whole group was 11% (5/42): one patient had an AMI despite IABP, two patients (4%) had an AMI during CABG, and in two patients (4%) who died within 24 h, an AMI could not be excluded. Total mortality rate was 7% (3/42): one patient died during the surgical procedure (2%), two high risk patients (4%) with three vessel disease, poor left ventricular function and severe associated disease (diabetes mellitus) died within 24 h. Of the 38 patients who survived CABG, one had an AMI within three months, four had mild stable angina pectoris (NYHA class II). None died (follow-up three months–five years).

Table 6 Slowly evolving MI

Sex	Age	IABP efficacy resolution of pain: yes	IABP efficacy resolution of pain: no	Follow-up	Results
M	57		+	6 months	Death at 6 months
M	30	+		3 years	NYHA I
F	53	+		2 years	NYHA II (LVF)
M	52	+		2 years	NYHA I
M	56	+		6 months	NYHA II
M	57	+		2 years	NYHA I
M	56	+		?	?
M	41	+		2 years	NYHA I

POST INFARCTION ANGINA AT REST REFRACTORY TO MEDICAL THERAPY (TABLES 1 AND 5)

Within two weeks after sustaining AMI, 17 patients (27–62 years, mean 49 years) had angina at rest, refractory to medical therapy. After institution of IABP, pain relief was prompt in 14/17 (83%). Symptoms worsened despite IABP in three patients; all three sustained AMI, and were subsequently not operated on. One of these developed shock and died.

Two other patients were not operated on. One patient with severe distal CAD was judged inoperable. In another patient the IABP was removed when worsening renal function together with hematuria suggested renal infarction (see complications, Table 8).

Twelve patients underwent CABG. None died, none developed AMI, three have mild stable angina (follow-up three months–four years).

Total myocardial infarction rate was 24% (4/17). Three patients did not respond to IABP, all sustained an AMI, and one developed CSMI and died. He was a 53 year old male with several earlier infarctions and poor left ventricular function ($EF \leqslant 0{\cdot}30$) and an acute anterior infarction. The two other patients survived their AMI. One patient was considered inoperable and had a fatal AMI one month later. Total mortality rate was 12% (2/17). There was no surgical mortality in this group.

SLOWLY EVOLVING MYOCARDIAL INFARCTION (TABLES 1 AND 6)

Persistence of pain refractory to medical therapy over a period of 12 h after sustaining AMI, associated with refractory ventricular arrhythmias, and/or mild LVF, suggested a slowly evolving AMI in eight patients (30–57 years, mean 50 years). After IABP institution, pain relief associated with reduction in LVF and arrhythmias was prompt in 7/8 (88%). One patient not responding to IABP, remained limited by chronic LVF and died suddenly six months later.

Of the seven patients who did well on IABP, six were followed up for six months–three years: four are asymptomatic, one has stable angina pectoris requiring drugs, one has chronic mild LVF. No cardiac catheterization was performed in this group.

Discussion

CARDIOGENIC SHOCK

CSMI has a grave prognosis with medical therapy alone[25, 26]. IABP combined with cardiac surgery has been reported to reduce the mortality rate to 55–60%[25,27]. IABP alone has reduced the mortality rate to 70–84%[28, 29]. The remarkably good results at our institution[12–14] may be partially attributed to the extension of our indications from strict CSMI MIRU IV (MIRU IV) to severe LVF after AMI (MIRU III–IV). A better prognosis is expected related to a better haemodynamic status.

Our earlier experiences have indicated that severe LVF, so called 'preshock', is indeed a precursor to shock. The majority of patients presenting with a PCWP greater than 18 mmHg and a S.W.I. (S.I. × MAP) between 1200–2000 ml mmHg M^{-2} (MIRU III–IV) progressed rapidly into irreversible shock MIRU IV with medical therapy alone. This negative outcome of medical therapy alone is well demonstrated in the patients with CSMI III–IV who were candidates for the IABP, but in whom balloon catheter insertion failed (Table 7): 7/8 (88%) patients died within one month following medical therapy, and only one patient survived over one year, but was incapacitated by chronic LVF (NYHA III). All eight patients had a S.W.I.

Table 7 Failures to insert balloon catheter

Sex	Age	Indication IABP	Surgery	Follow-up	Result
M	59	CSMI III–IV	—	1 day	Death at 1 day
M	58	CSMI III–IV	—	20 days	Death at 20 days
M	51	CSMI III–IV	—	1 year	NYHA III, Death at 1 year
M	69	CSMI III–IV	—	15 days	Death at 15 days
F	44	CSMI III–IV	—	1 day	Death at 1 day
M	69	CSMI III–IV	—	1 day	Death at 1 day
M	65	CSMI III–IV	—	2 days	Death at 2 days
M	64	CSMI III–IV	—	3 days	Death at 2 days
F	57	Slowly evolving MI	—	2 years	Death at 2 years
M	61	p.o. CABG	—	1 day	Death at 1 day
M	60	post infarct a.p. at rest	+	3 years	NYHA I–II
M	61	miscellaneous	—	1 year	NYHA II
M	60	miscellaneous	—	1 year	?

exceeding 1200, but lower than 2000 ml mmHg M^{-2}. The limitations of medical therapy alone in severe LVF and shock after AMI are clear. Positive inotropic drugs enhance cardiac function but they act mainly upon normal areas of myocardium[31] and CSMI is characterized by a massive loss of myocardium. Post-mortem studies have documented a myocardial destruction of at least 40% of the left ventricle in patients dying in shock after AMI[26, 32]. This degree of myocardial loss reduces the extent of cardiac muscle responsive to inotropic agents. By increasing myocardial oxygen requirement, positive inotropic agents may even augment myocardial ischaemia. Positive inotropic agents however can restore normal blood pressure, and maintain an adequate aortic diastolic blood pressure, which is the major determinant of the major determinant of coronary blood flow in these extreme conditions[2, 3].

Vadodilating agents[30, 32, 34], decrease oxygen requirements by reducing afterload and preload, but if cardiac output does not compensate for the decreased peripheral resistance, aortic diastolic blood pressure may drop beyond a critical level.

Our experience indicates that CSMI is principally a complication of acute anterior myocardial infarction. This observation may contrast with a number of studies that did not demonstrate a relation of shock to location of infarction[32, 35], but it is in agreement with recent investigations that demonstrated that impairment of pump function is greater and the occurrence of shock is more frequent in anterior than in inferior infarctions[29–31].

Anterior infarction is related to occlusive disease of the LAD, which in the majority of instances supplies a greater quantity of left ventricular myocardium than either the right or circumflex arteries. This explains the fact that anterior infarction is usually quantitatively greater than inferior[36, 37].

Shock complicating an acute inferior infarction is usually either associated with acute mechanical complications[31] (rupture of the posteromedial papillary muscle, which has a blood supply relatively less adequate than the anterior papillary muscle, or ventricular septal rupture) or with extension of infarction to the right ventricle[38, 38], causing predominant right ventricular infarction and failure, with a low input of the left ventricle, a mechanism fundamentally different from cardiogenic shock complicating acute anterior myocardial infarction, which is caused by extensive damage to the left ventricular free wall, apex and inter ventricular septum. This difference has practical consequences: if a patient is admitted with the clinical signs of cardiogenic shock and an acute anterior myocardial infarction, no time should be wasted and the IABP should be introduced as soon as possible. In the patient with an acute inferior infarction presenting with hypotension, oliguria and an altered sensorium, but without pulmonary congestion, haemodynamic measurements should be performed to exclude a right ventricular infarction, characterized by an elevated right atrial pressure, little generation of pressure in the right ventricle and a normal PCWP and a low C.O.

Table 8 Complications of IABP

Duration of IABP	<5 days	5–10 days	>10 days	Total
Total no. of patients	87	34	47	168
Infection of wound	2	3	6	11
Femoral artery bleeding	3	1	4	8
Local damage to artery requiring surgery		1	1	2
Ischaemia of leg-acute	1	2		3
Ischaemia of leg-chronic	1		2	3
Dissection of aorta	2	2		4
Kinking of balloon	1			1
Thromboembolism	3	4	6	13
Renal function impairment	1			1
Total no. of major complications	14	13	19	46
Thrombocytopaenia	2	15	37	54

Plasma infusions i.v. may increase the pressure gradient and passive pulmonary flow in this patient. IABP however reduces left ventricular afterload, and offers no benefit to such a patient[38, 39].

Our results are in agreement with those of others[31, 40] that there is a subset of patients with CSMI in whom prognosis appears to be more favourable. These are generally young patients, with their first anterior AMI, and prodromes of recent onset (<4 weeks). Coronary angiography frequently shows a proximal single occlusion of the LAD. At autopsy a proximal thrombotic occlusion of the LAD is found often with distal patency and no major arteriosclerotic changes in the other coronary vessels.

Patients with acute mechanical lesions comprise another subset. Rupture of a papillary muscle is relatively rare, and most commonly involves the posteromedial papillary muscle in inferior myocardial infarction[31]. The anterolateral papillary muscle may be involved in anterolateral infarction. In anterior infarction, dilation of the left ventricle with geometrical changes is probably a more important contributor to mitral regurgitation.

The clinical differentiation of mitral regurgitation from septal rupture is often difficult. Definitive diagnosis requires right heart catheterization with analysis of right atrial and ventricular blood oxygen content. Mitral regurgitation and septal rupture may occur simultaneously in the same patient[41]. Ventricular septal rupture usually involves the lower portion of the muscular septum and varies in size and number of perforations. Our current position is that surgery in CSMI should be limited to patients with definite mechanical lesions: mitral regurgitation due to papillary muscle rupture, ventricular septal ruptures, and circumscribed aneurysms. Our surgical experience is very limited (six patients). In fact only two patients survived: one had a ruptured papillary muscle and one a ventricular septal rupture, both being operated on well over six weeks after the acute event[13]. A third subgroup may be constituted by generally older patients with longstanding CAD and multiple vessel disease[31]. In this category, the onset of shock may be more gradual, possibly explaining the time delay in treatment with IABP so often encountered in these patients.

ANGINA PECTORIS AT REST REFRACTORY TO MEDICAL THERAPY

A detailed descriptive diagnosis is preferable to a loose use of the term unstable angina or its many synonyms. The great diversity in definitions and therapeutic approaches makes it difficult to compare different studies on unstable angina pectoris. However, some facts clearly emerge from the studies performed in the past[42–45]. First, there is a sharp decrease in mortality and morbidity when comparing the studies done before 1970 with those done after 1970. While the earlier studies may represent the natural history of unstable angina pectoris, the later reflect the outcome of different therapeutic approaches. Second, subgroups at high risk of myocardial infarction and/or sudden death, are constituted (a) by patients with unstable angina pectoris, superimposed on stable angina pectoris, or previous infarction(s), (b) by patients who remain symptomatic after 48 h of

bedrest and maximal medical therapy[42, 44] and (c) by patients with post infarct refractory angina at rest[45, 46]. Third, obligatory surgery should be performed in patients with obstruction of the LMCA[47]. Fourth, patients with unstable angina and a very proximal LAD obstruction, and an as yet uncompromized myocardium seem to constitute a category, in whom surgery should be obligatory[47]. Fifth, both cardiac catheterization and CABG have a higher morbidity and mortality in unstable angina, when compared to stable angina[42, 43]. Sixth, surgical risk is related to cardiac function, as reflected by the ejection fraction and the number of vessels diseased[42]. Seventh, patients managed with medical therapy alone, have no higher mortality rate than those undergoing CABG. Eighth, the extent of coronary artery disease determines subsequent return of angina pectoris[24], and thus the need for elective CABG at a lesser risk than when under unstable conditions. Immediate surgery should therefore be restricted to patients not responding to medical therapy, and patients with LMCA and proximal LAD obstructions.

Our results indicate a high perioperative mortality in patients with refractory angina pectoris treated with the IABP (7%). However the series is small (41 patients), and it should be noted that death was attributable to intractable diabetes in two patients. One of these was also at high risk because of severe three vessel obstructive disease and poor left ventricular function. In one patient the IABP may have contributed to death. It should be stressed that no surgical mortality could be demonstrated in the 12 patients undergoing CABG for post infarct angina at rest. When considering the whole group, surgical mortality is 6% (3/53), still definitely higher than the mortality for patients undergoing CABG under more stable conditions (1%)[48].

The patients selected for IABP however were completely refractory to any form of medical therapy and had rapid progression of their symptoms[49], and they constitute a special 'high risk' group. We do not recommend a more aggressive and prophylactic use of the IABP, which may be unnecessarily hazardous. The introduction of new drugs may further diminish the need for IABP in these patients in future.

PERSISTING PAIN AFTER AMI, SUGGESTING SLOWLY EVOLVING INFARCTION

This series is rather small, there is no control group, and evidence that we actually reduced infarct size or the extent of ischaemic injury is lacking. Prolonged ischaemic pain, with hypotension and/or ventricular arrhythmias is an established indication for IABP[50]. However, in these reports the IABP served as a stabilizing device, prior to CABG, and was not the definitive method of therapy.

Our approach is unique in the sense that the IABP was used as a definitive therapy, for a fixed period of time (five days), without contemplating CABG. The abolition of pain suggests effective reduction of ischaemia, and we demonstrated that these patients could be weaned from the device without recurrence of symptoms, and had no myocardial infarctions or sudden death during a follow-up of six months–three years.

COMPLICATIONS OF IABP

Complications of IABP may be grave, and include arterial and aortic wall damage with or without gangrene of the leg distal to catheter insertion, embolization, thrombocytopaenia, balloon rupture, trapping of gas in a kinked balloon, and infection[13, 28, 51–55].

In our experience, prolonged use of IABP was associated with more complications[13], notably thromboembolic processes, vascular lesions and thrombocytopaenia. However, no relationship between fatal complications and duration of IABP was found. All three lethal complications were related to catheter insertion. Dissection of the aorta is the major complication of catheter insertion, and may go completely unnoticed, with effective intramural balloon pumping[28].

Conclusion

Cardiogenic shock and severe left ventricular failure, principally complicating acute anterior myocardial infarction, can be successfully treated with IABP alone. Delay of treatment is the major determinant of prognosis, which further depends on age and history of coronary artery disease. Surgery is indicated when mechanical defects exist, but has only limited success.

When medical therapy is ineffective, rapid institution of IABP ensures immediate pain relief to patients with angina pectoris at rest refractory to optimal medical therapy.

Even in patients with post infarct unstable angina subsequent coronary artery bypass grafting can be performed safely with good prognosis for survival and symptoms.

IABP abolishes symptoms when persistent pain, ventricular irritability and mild LVF suggest a slowly evolving myocardial infarction.

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