Objective haemodynamic assessment after acute myocardial infarction¹

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Haemodynamic measurements were made in 84 patients with coronary heart disease soon after admission to a cardiac care unit. Patients without acute myocardial infarction had haemodynamic findings no different from those with definite infarction uncomplicated by congestive heart failure or cardiogenic shock. With these complications, however, haemodynamic derangements became more severe. Heart rate, cardiac and stroke volume index, peripheral vascular resistance, and left ventricular filling pressure all proved unreliable as single variables in predicting ultimate survival or death. However, left ventricular work index less than 1.75 kg m/min per m² was associated with very high mortality, and the value of this index predicted survival or death with more than 90 per cent accuracy.

In spite of major advances in the prevention and treatment of cardiac arrhythmia, 10 to 20 per cent of patients in hospital with acute myocardial infarction do not survive. Most succumb to cardiogenic shock or some other manifestation of 'pump failure'.

Established cardiogenic shock is usually associated with massive myocardial damage, involving 40 per cent or more of left ventricular mass (Page *et al.*, 1971; Harnarayan *et al.*, 1970; Scheidt *et al.*, 1971). It has been suggested that myocardial damage may occur in a 'stepwise' or progressive fashion. Extension of infarction is commonly observed at necropsy in patients who die with cardiogenic shock, though infrequently recognized clinically (Scheidt *et al.*, 1971). If the likelihood of progressive myocardial damage could be recognized early, attention could be focused clinically on efforts to limit loss of functioning heart muscle and prevent clinical deterioration.

The prospect of limiting myocardial damage by pharmacological or mechanical means is presently under active exploration. Improvement in electrocardiographic (Maroko *et al.*, 1971), haemodynamic (Franciosa *et al.*, 1972), and metabolic (Mueller *et al.*, 1972) correlates of myocardial damage has been demonstrated after the use of drugs which reduce contractility or aortic impedance ('afterload') and with the counterpulsating intra-aortic balloon.

These forms of therapy are experimental and neither the indications for their use nor the potential hazards are completely understood.

There is an urgent need for reliable and objective criteria which will identify patients at high risk for circulatory failure or death before clinical deterioration. Unconventional therapy may be justified in this group. We report herein studies directed toward establishing objective prognostic criteria in 84 patients with coronary heart disease.

Methods

All patients were admitted to the Warburg Cardiac Care Unit and studied in a specially equipped myocardial infarction research unit. The diagnosis of myocardial infarction was based on clinical, electrocardiographic, and laboratory data. Sixty-six patients had acute myocardial infarction with a typical clinical history and the development of either electrocardiographic Q waves or characteristic changes in serum activity of aspartate aminotransferase or creatine phosphokinase. Nine patients had a compatible history but equivocal electrocardiographic or serum enzyme changes and were classified as 'possible infarction'.

Nine patients admitted because of severe chest pain failed to show either electrocardiographic or serum enzyme changes and were considered not to have suffered an infarction. These patients were classified as 'no infarction' since the episode was considered to represent severe angina or 'coronary insufficiency'. The data from these patients were used as a 'control' and compared with the findings from patients with established infarctions. All patients were kept at complete rest in bed in

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the cardiac care unit for several days until acute myocardial infarction was diagnosed or excluded. During the first few days in hospital all three groups of patients were treated in the same manner.

Patients were grouped according to the following arbitrary clinical classification.

Class I: No evidence of congestive heart failure or shock.

Class II: Mild-moderate congestive heart failure (râles thought to be of cardiac origin heard over an area of less than half of both lung fields).

Class III: Severe congestive heart failure (râles heard over half or more of both lung fields: 'pulmonary oedema').

Class IV: Cardiogenic shock (intra-arterial systolic pressure less than 90 mmHg) with evidence of reduced cerebral or renal blood flow (urine flow < 20 ml/hr). Hypovolaemia was excluded by measurement of left ventricular filling pressure and/or expansion of intra-vascular volume when indicated.

Although uncertainties associated with clinical (Wolk, Scheidt, and Killip, 1972) and radiological (Harrison, Conte, and Heitzman, 1971) signs of congestive heart failure complicating acute myocardial infarction are well known, this classification is easily made at the bedside at the time of admission, correlates well with subsequent haemodynamic measurements (*vide infra*), and provides a rough estimate of expected hospital mortality (Killip and Kimball, 1967). It has been adopted as a standard classification by the myocardial infarction research units.

Table 1 gives the distribution of the patients by clinical class at the time of the initial haemodynamic study.

At the onset of the study patients were selected because of the presence of complications such as heart failure, hypotension, or shock. Once the feasibility and safety of haemodynamic measurements was demonstrated, patients were selected for study randomly from new admissions. In all, 21 patients were randomly selected while 63 were studied because of complications. Patients with valvular heart disease, primary myocardial disease, or significant pulmonary disease are excluded from this report.

Physiological studies were initiated as soon after admission to the cardiac care unit as possible, beginning 4.5 hours to 21 days after the clinical 'moment of infarction' (median time 35 hours, Table 1). Subsequent studies were done at 12- to 24-hour intervals. Informed consent was obtained from all patients. Appropriate therapy was not interrupted during the course of the study.

Right and left heart catheterizations were performed with thin flexible nylon catheters¹ (outer diameter 1.4 mm, inner diameter 1.0 mm) approximately 105 cm long inserted by a modified Seldinger technique. The catheter is small enough for a 19 gauge thin wall Seldinger needle to be used for arterial puncture (Fig. 1).

TABLE 1 Description of patient groups

	Survivors	Deaths
Number	31	35
Time of study		
1) After infarct:		
median	24.5 hr	53 hr
range	7 hr to 21 dy	4.5 hr to 21 dy
2) Before death:		
median	—	37 hr
range		1 hr to 49 dy
Clinical class at time of initial haemodynamic study (patients):		
I	14	5
II	12	12
III	1	3
IV	4	15

The left ventricle is routinely entered without fluoroscopy and only occasional premature contractions occur upon passage into the ventricle.

During the past year a Swan-Ganz balloon-tipped flow-guided catheter has been used for right heart catheterization. Catheters were left in the pulmonary artery for as long as three days without complications. The left heart catheter was usually withdrawn into the ascending aorta after each set of measurements and the left ventricle re-entered for subsequent studies. In several patients, however, left ventricular pressure was continuously monitored for up to 45 hours without difficulty or arrhythmia. Pressures were recorded with Statham 23dB transducers and inscribed on an oscillographic recorder.²

Cardiac output was measured by dye dilution technique with injection of indocyanine green into the pulmonary artery. Arterial blood was sampled at the aortic root and withdrawn through a cuvette densitometer.³ Indicator dilution curves were replotted semilogarithmically by standard methods (Hamilton *et al.*, 1932).

External cardiac work index (CWI) in kg m/min per m² was calculated according to the formula:

$$CWI = (MSP - LVEDP) \times CI \times 1.055 \times 13.6 / 1000$$
 where MSP is the mean systolic pressure in mmHg, integrated over the total duration of left ventricular systole, LVEDP the left ventricular end-diastolic pressure in mmHg measured 0.04 sec after the onset of the QRS complex, CI the cardiac index in l./min per m², 1.055 the specific gravity of blood, and 13.6 the conversion factor for the specific gravity of mercury.

Stroke work index (SWI) was calculated from: $SWI = CWI \times 1000 / \text{heart rate}$.

Central blood volume was calculated from the standard formula (Luisada, 1959).

² Electronics for Medicine DR 20.

³ Waters cuvette XC 302, densitometer XP 302.

¹ Portland Plastics, Hythe, Kent, England.

FIG. 1 Nylon catheter and 19 gauge thin-wall Seldinger needle for arterial and left ventricular cannulation. For comparison, a standard 20 gauge needle is shown. The flexible catheter has remained in the left ventricle for continuous pressure monitoring for up to 45 hours.

Complications

In 257 haemodynamic studies there was one instance of ventricular fibrillation and one of sustained ventricular tachycardia associated with catheter passage through a cardiac chamber. Both were promptly terminated by praecordial DC shock. Atrial fibrillation was precipitated in one patient and atrial tachycardia in another. The former reverted to sinus rhythm after digitalis therapy and the latter terminated during tracheal suctioning. There were no adverse sequelae from any arrhythmias encountered. There were no complications from the arterial cannulations. Two episodes of phlebitis developed. Both occurred after a nylon catheter had been left in place for more than 72 hours. There was one instance of excessive bleeding around catheters in an anticoagulated patient, requiring administration of protamine and removal of the catheters. One perforation of a superficial basilic vein after passage of the flow-guided nylon catheter was recognized only by fluoroscopy and did not result in symptoms.

Results

Clinical-haemodynamic correlation

Although there is a wide range of variability, the haemodynamic findings correlated well with the clinical classification of the patient (Fig. 2). Patients with moderate congestive heart failure, Class II, or

cardiogenic shock, Class IV, had progressively greater abnormalities than did those in Class I. Because of the unstable state and the need for urgent treatment there were too few studies of patients in pulmonary oedema, Class III, for meaningful analysis.

Haemodynamic measurements in the 9 patients with 'no infarction' are shown in Fig. 2 and are considered to represent a 'control' standard of comparison. In this group of patients with ischaemic heart disease but no evidence of overt infarction several haemodynamic abnormalities were apparent. Mean heart rate was 97 beats/min, mean cardiac index $2.7 \text{ l./min per m}^2$, mean stroke volume index $30 \text{ ml/beat per m}^2$, mean left ventricular end-diastolic pressure 11 mmHg , and mean cardiac work 6.5 kg m/min . Patients with established acute myocardial infarction and no clinical evidence of heart failure (Class I) did not, as a group, have haemodynamic findings significantly different from the 'no infarction' or 'control' patients.

Heart rates were higher, cardiac index, stroke volume index, cardiac work, and stroke work were lower in Class II as compared to Class I patients. Patients with cardiogenic shock (Class IV) showed the most pronounced haemodynamic derangements.

FIG. 2 Haemodynamics and clinical status after acute myocardial infarction. Class I (uncomplicated infarction) patients do not differ from 'control' patients with ischaemic heart disease but no infarction. Patients with heart failure (Class II) and shock (Class IV) show progressively more severe haemodynamic derangements. I = ISD.

Cardiac and stroke volume index were reduced to less than 50 per cent of normal. Cardiac and stroke work were significantly lower than in Group I ($P < 0.05$).

There is a wide range of left ventricular filling pressures in patients with acute myocardial infarction. Left ventricular end-diastolic pressure averaged 16 mmHg in Class I and rose to 25 mmHg in Class II patients. The variation in left ventricular end-diastolic pressure encountered in Class I patients, despite the absence of clinical evidence of heart failure, was wide, ranging from 4 to 31 mmHg (Fig. 3).

Left ventricular end-diastolic pressure in almost half the Class II patients equalled or exceeded 25 mmHg, a level exceeding pulmonary capillary oncotic pressure. Frank pulmonary oedema was not noted in these patients probably because mean left ventricular diastolic pressure was less than pulmonary capillary oncotic pressure, even though left ventricular end-diastolic pressure was higher.

Left ventricular end-diastolic pressure varied widely in Class IV patients, ranging from 8 to 45 mmHg. This wide range of pressure probably reflects the varying state of ventricular function encountered in cardiogenic shock. Some patients were moribund, others survived many hours or days. As stroke volume and arterial pressure fall, left ventricular end-diastolic pressure generally declines also, probably a reflection of the fact that the dying heart can neither fill nor empty.

Prognostic studies

Of the 66 patients with acute myocardial infarction,

31 survived to leave the hospital and 35 died. All but one of the 35 deaths were related to the cardiovascular system. Haemodynamic measurements obtained soon after admission proved valuable in predicting the eventual outcome after infarction.

FIG. 3 Left ventricular end-diastolic pressure (LVEDP) after acute myocardial infarction. It is frequently raised even in patients without clinical evidence of heart failure (Class I), and frequently equals or exceeds pulmonary capillary oncotic pressure in patients with mild or moderate heart failure (Class II) even when there is no clinical evidence of overt pulmonary oedema. Left ventricular end-diastolic pressure in shock is variable.

TABLE 2 Initial haemodynamic measurements

	Survivors	Deaths	P
Cardiac index (l./min per m ²)	2.5 ± 0.7	1.6 ± 0.7	<0.001
Stroke volume index (ml/beat per m ²)	32 ± 14	16 ± 6	<0.001
Peripheral vascular resistance (dynes-sec-cm ⁻⁵)	1700 ± 600	2200 ± 1300	NS
Cardiac work (kg m/min)	4.9 ± 2.4	2.2 ± 1.4	<0.001
Cardiac work index (kg m/min per m ²)	2.7 ± 1.2	1.3 ± 0.7	<0.001
Stroke work (gm m/beat)	62 ± 35	22 ± 12	<0.001
Stroke work index (gm m/beat per m ²)	32 ± 18	13 ± 6	<0.001
Heart rate (beats/min)	85 ± 21	102 ± 17	<0.05
Aortic pressure (mmHg)	124 ± 23/73 ± 13	108 ± 19/65 ± 13	NS
Pulmonary artery diastolic pressure (mmHg)	18 ± 13	24 ± 9	NS
Left ventricular end-diastolic pressure (mmHg)	20 ± 10	24 ± 7	NS
Right atrial pressure (mmHg)	8 ± 2	10 ± 4	NS

Mean ± 1 SD; NS, not significant.

FIG. 4 Initial haemodynamic measurements in survivors (open circles) and non-survivors (closed circles). Extensive overlap of values for cardiac index, stroke volume index, heart rate, peripheral vascular resistance, and left ventricular filling pressure renders use of these variables for prediction of survival unreliable.

To be useful prognostically, objective measurements in survivors and non-survivors should not only be significantly different when averaged by groups, but also demonstrate minimal overlap of individual values. A given patient can then be assigned with confidence to the group expected to survive or the group with a high probability of dying.

Patients who died had significantly lower cardiac and stroke volume indices, cardiac and stroke works, and significantly higher heart rates than those who survived (Table 2). Data from individual patients are shown in Fig. 4 and 5. There is considerable overlap between survivors and non-survivors with regard to heart rate, cardiac and stroke index, peri-

pheral vascular resistance, and left ventricular end-diastolic pressure. However, the degree of separation between the two groups for cardiac and stroke work is reasonably good. A cut-off point of 1.75 kg m/min per m² for cardiac work index was arbitrarily chosen as providing the best separation between survivors and non-survivors.

Thus, of 29 patients in whom death was predicted by the empirical criterion of cardiac work index less than 1.75 kg m/min per m², only 7 survived, including 2 patients treated with the intra-aortic counterpulsating balloon after haemodynamic evaluation. Of 29 patients in whom survival was predicted (cardiac work index > 1.75 kg m/min per m²), only 2 died: one death, moreover, was not due to a

FIG. 5 Initial haemodynamic measurements in survivors and non-survivors. Cardiac and stroke work index, derived variables reflecting left ventricular function, effect good separation between survivors and non-survivors. Cardiac work index less than 1.75 kg m/min per m^2 is associated with very high mortality.

primary cardiac cause. Excluding the patients who received mechanical circulatory assistance and the non-cardiovascular death, prediction of life or death from early measurement of cardiac work index using a cut-off value of 1.75 kg m/min per m^2 (or cardiac work of 3.0 kg m per min) is 90 per cent accurate (Table 3).

TABLE 3 Objective assessment of prognosis

No. of patients	Parameter	Cut-off value	False positive (death predicted but patient survived) survivors/total predictions (1)	False negative (survival predicted but patient died) deaths/total predictions (2)	Accuracy of prediction per cent (3)
58	Cardiac work	3.0 kg m/min	6/29	2/29	91
58	Cardiac work index	1.75 kg m/min per m^2	7/29	2/29	90
58	Stroke work index	20 g m/beat per m^2	8/29	2/29	88
58	Stroke work	30 g m/beat	7/29	4/29	86
61	Stroke volume index	20 ml/beat per m^2	5/29	7/32	85
62	Cardiac index	1.5 l./min per m^2	2/30	18/32	73
61	Left ventricular filling pressure	12 mmHg	18/29	3/32	70
61	Left ventricular filling pressure	23 mmHg	9/29	10/32	70
61	Heart rate	80 beats/min	18/30	4/31	67
57	Peripheral vascular resistance	1250 dynes-sec- cm^{-5}	23/30	4/27	56

Only patients with definite myocardial infarction are included.

1) Two patients who survived after intra-aortic balloon pumping, though death was predicted, are included in this column.

2) One patient who died of sepsis (non-cardiac death) is included in this column.

3) Balloon pump survivors and the non-cardiac death are excluded in calculating accuracy of prediction.

Discussion

The clinical status of a patient with acute myocardial infarction is a reflection of physiological and biochemical adjustments to the acute event. As shown in the present study, patients with uncomplicated infarction (Class I) have measured pressures and flows that are probably close to normal for patients of similar age with ischaemic heart disease but without acute infarction. Though there is much variation, left ventricular filling pressure in uncomplicated infarction is, in most cases, at the upper limits of normal.

When the patient with acute myocardial infarction manifests clinical evidence of congestive heart failure (Class II), cardiac and stroke indices are reduced below age-matched controls, while left ventricular filling and right atrial pressures are raised (Wolk *et al.*, 1972). We have shown previously that arterial PO_2 is reduced and intrapulmonary shunting increased in Class II patients (Fillmore *et al.*, 1972).

Cardiogenic shock complicating acute myocardial infarction is associated with conspicuous physiological derangements. Cardiac index is less than one-third of normal, stroke index one-quarter normal, left ventricular end-diastolic pressure twice normal, and cardiac and stroke work greatly reduced. Severe hypoxia and large intrapulmonary shunts are also demonstrable with cardiogenic shock (Fillmore *et al.*, 1972).

It is not yet clear what portion of the initial myocardial injury in the patient with acute infarction is potentially reversible. In experimental myocardial

FIG. 6 Pressure, flow, and external cardiac work in survivors and non-survivors. The cardiac work index isopleth at $1.75 \text{ kg m/min per m}^2$ (dotted line) best separates survivors from non-survivors.

infarction, manipulation of intra- and extracardiac pressures, use of various pharmacological agents, and application of mechanical circulatory assistance have been shown to alter the area of apparent injury or death (Maroko *et al.*, 1971; Franciosa *et al.*, 1972; Mueller *et al.*, 1972; Maroko *et al.*, 1972). Biochemical and metabolic studies indicate that cellular death is the culmination of a gradual destructive process after deprivation of nutrient flow. Myocardial injury appears to be reversible despite relatively long periods of ischaemia (Brachfeld, 1969). These observations suggest the possibility that myocardial damage could be limited by appropriate clinical manipulation.

The great majority of patients in hospital with acute myocardial infarction do not suffer major complications and have a low mortality. Thus, the current mortality of patients without congestive heart failure or shock at the New York Hospital is 6 per cent. Mortality is increased in patients with clinical evidence of left ventricular dysfunction. With mild congestive heart failure, mortality is 17 per cent, with pulmonary oedema 38 per cent, and with cardiogenic shock 81 per cent (Killip and Kimball, 1967). Few therapeutic interventions, current and proposed, are without some risk, yet objective criteria for predicting the natural history of acute myocardial infarction are lacking.

There is a clear need for development of reliable prognostic criteria which can be applied to the individual with acute myocardial infarction. At-

tempts to grade severity and predict the outcome after infarction are not new. Early studies by Mintz and Katz (1947) and Rosenbaum and Levine (1941) were descriptive and related mortality to various features of history and physical examination. Schnur (1953) and Peel *et al.* (1962) constructed prognostic indices based on weighted clinical scores for historical data, associated diseases, physical findings, electrocardiographic abnormalities, rhythm disturbances, etc. Weil and Affi (1970) found arterial lactate to be an excellent indicator of the severity of acute circulatory failure and reported experimental findings that cumulative oxygen debt was related both to survival and blood lactate.

Scheidt, Ascheim, and Killip (1970) examined factors influencing survival in 75 patients with cardiogenic shock and noted that all patients with coma and total anuria died. Patients with rapid sinus tachycardia, atrioventricular block, pulmonary oedema, and severe refractory acidosis had mortality in excess of 90 per cent. Shubin *et al.* (1968) assessed the relative importance of various haemodynamic parameters in 20 patients with cardiogenic shock by discriminant function analysis. Measurements of flow, particularly cardiac index, were most reliable in predicting outcome, but observations in patients who died were made shortly before death and thus were unduly influenced by the near-agonal state. Parmley *et al.* (1972) did not find analysis of left ventricular function, including end-diastolic pressure, maximum dp/dt , and contractile element velocity (VCE) to be useful in separating survivors from non-survivors. Prakash *et al.* (1972) measured serum cortisol, plasma free fatty acids, and urinary catecholamines. Serum cortisol less than $20 \mu\text{g}/100\text{ml}$ within 24 hours of myocardial infarction identified survivors with good accuracy whereas the other biochemical determinations were prognostically less useful. Agress *et al.* (1972) used indirectly measured left ventricular filling pressure, systemic arterial pressure, and pre-ejection period to estimate a 'left ventricular contractility index': maximum LV dp/dt /left ventricular end-diastolic pressure. This 'contractility index' correlated well with directly measured stroke work and was useful in separating survivors from non-survivors.

The present study presents haemodynamic data which provide an estimation of mortality soon after the onset of symptoms. No single measurement of pressure or flow was useful for predicting death or survival. However, a combination of variables reflecting left ventricular function provided a sharp prognostic discrimination. Thus calculation of cardiac work, derived from measurement of cardiac output, mean systolic pressure generated by the left ventricle, and left ventricular end-diastolic pressure,

was most useful in separating survivors from non-survivors. It should be noted that the term 'cardiac work' as here defined represents only external, or pressure-flow work, and does not reflect total work performed by the left ventricle. Without measurements of ventricular volume, wall tension, a major determinant of total myocardial work, cannot be calculated. Regardless of physiological meaning, external cardiac work, derived from easily obtainable measurements, appears to be empirically useful.

Thus cardiac work index determined at the initial haemodynamic study exceeded $1.75 \text{ kg m/min per m}^2$ in 22 of 29 survivors but failed to reach that level in 27 of the 29 patients who died. 'False positive' results, that is, cardiac work index less than $1.75 \text{ kg m/min per m}^2$, were obtained in 7 patients in whom death was predicted but the patient survived. Two of these patients were treated with the intra-aortic balloon pump which produced obvious clinical improvement: we believe they would almost certainly have died but for the mechanical circulatory assistance. Two of the survivors were severely incapacitated after discharge (New York Heart Association functional class IV); one of these was subsequently admitted to hospital several times with severe cardiac failure and died during attempted coronary bypass surgery less than three months after the initial infarction. A fifth patient sustained two further myocardial infarctions within the year after discharge. The sixth patient was studied 5 hours after a prolonged cardiorespiratory arrest that occurred just before admission to hospital. He was comatose and had aspiration pneumonia at the time of study. In the remaining patient the initial pronounced depression of cardiac work with survival is unexplained and he is now well.

'False negative' results, that is, cardiac work index more than 1.75 kg m/min , were obtained in 2 patients in whom survival was predicted but the patients died. One patient aspirated during a cardiac arrest just before admission to the hospital and died of sepsis one month after infarction. This was considered a non-cardiovascular death. The second patient was recovering from his initial infarction when he sustained a clinical and electrocardiographic extension of infarction on the seventh day after admission. Cardiac work index measured after the extension of infarction was $1.1 \text{ kg m/min per m}^2$ and the patient subsequently died with cardiogenic shock.

Comparison of several measured or derived physiological variables shows the superiority of cardiac work as a useful prognostic index for patients in hospital with acute myocardial infarction (Table 3). The method of calculating work is pertinent, since use of peak systolic pressure or omission of

left ventricular end-diastolic pressure in the formula failed to yield prognostic discrimination. Fig. 6 graphically illustrates the discrimination achieved by using cardiac work, rather than either flow or pressure measurements alone.

Our findings are supported by haemodynamic studies obtained within 24 hours of acute myocardial infarction in the 9 myocardial infarction research units. Pooled data from 308 patients indicate that cardiac work gives excellent separation between hospital survivors and non-survivors (Myocardial Infarction Research Unit Annual Report, 1971).

To be useful, a prognostic index should be derived from measurements obtained shortly after the initial medical evaluation and should not be skewed by data obtained preterminally. The results of the present study do not reflect preterminal derangements since the initial evaluation occurred well before death in most of the subjects (median time of study 37 hours before death in those who died). The results are also not simply reflections of the physiological derangements of cardiogenic shock, since only 12 of 29 separate determinations of cardiac work in non-survivors were done while patients were in shock.

Current evidence suggests that patients with large initial infarcts are subject to a vicious circle of decreased myocardial perfusion and increased myocardial work after the initial injury, leading to extension of infarction and still further reduction in myocardial performance (Scheidt *et al.*, 1971). The early haemodynamic measurements in the present study which revealed low cardiac work presumably reflect a large initial insult before clinical deterioration. It appears that an initial low cardiac work, even in the individual who is holding his own clinically, identifies the patient who is likely to suffer further myocardial damage, develop cardiogenic shock, and have a high mortality.

Conclusions

Haemodynamic measurements are simply and safely obtained soon after acute myocardial infarction, and are useful for prognostic purposes. No single haemodynamic variable appears as reliable for predicting survival as a combination reflecting left ventricular function. Magnitude of cardiac work, calculated from cardiac output, mean systolic pressure, and left ventricular end-diastolic pressure, was 90 per cent accurate in separating survivors from non-survivors. Early identification of the patient at high risk for circulatory failure or death may speed application of therapy, perhaps unconventional or hazardous, to limit myocardial damage and prevent clinical deterioration.

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