Left Ventricular Function in Acute Myocardial Infarction

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ABSTRACT Left ventricular catheterization was carried out in 40 patients with acute myocardial infarction. Left ventricular end-diastolic pressure (LVEDP) was elevated in 85% of the patients studied. In 14 patients with apparently uncomplicated infarcts, LVEDP averaged 15 mm Hg, and cardiac index (2.98 liter/min/m^2), stroke volume (38.3 ml/m^2), and stroke work (49.2 g-m/m^2) were within normal limits. In 12 patients with clinical signs of left ventricular failure, LVEDP averaged 29.9 mm Hg, cardiac index was at the lower limit of normal (2.79 liter/min/m^2), but stroke volume (31.6 ml/m^2) and stroke work (37.3 g-m/m^2) were reduced. In 14 patients with clinical signs of shock, LVEDP averaged significantly lower than in the heart failure group (21.1 mm Hg), but cardiac index (1.59 liter/min/m^2), stroke volume (16.5 ml/m^2), and stroke work (11.1 g-m/m^2) were markedly reduced. A large presystolic atrial "kick" (average amplitude 9.5 mm Hg) was an important factor in the high LVEDP in the patients with heart failure but not in those with shock. The first derivative of left ventricular pressure was significantly lower in shock than in the nonshock group. Although right atrial pressure (RAP) and LVEDP were significantly correlated ($r = 0.49$), wide discrepancies in individual patients rendered the RAP an unreliable indicator of the magnitude of left ventricular filling pressure.

These data show the following: (*a*) LVEDP is usually elevated in acute myocardial infarction, even in absence of clinical heart failure; (*b*) cardiac output apparently is supported by increased LVEDP and compensatory tachycardia; (*c*) in patients with shock, left ventricular function usually is markedly impaired, but inadequate compensatory cardiac dilatation or tachycardia could contribute to the reduced cardiac output in some individuals; (*d*) lower LVEDP in shock than in heart failure may represent differences in left ventricular compliance.

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INTRODUCTION

Evaluation of cardiac function during acute myocardial infarction has previously been limited to the assessment of clinical and radiological signs (1), the measurement of cardiac output (2–5), right heart and pulmonary artery pressures (6, 7), and external indices of cardiac performance (8, 9). Although these studies have provided some insight into the severity of cardiac impairment which may accompany myocardial infarction, more precise quantitation of left ventricular dysfunction requires the direct measurement of left ventricular pressures (10, 11).

In recent years the mortality rate from arrhythmias in myocardial infarction has been markedly reduced, but mortality from the complications of pump failure has not been significantly altered (12, 13). The need for improved management of cardiac and circulatory failure after myocardial infarction has led to the introduction of new drugs (14, 15), mechanical cardiac assist devices (16), and aggressive surgical procedures (17) for the emergency treatment of myocardial infarction. The availability of these techniques has increased the requirement for an understanding of the functional disturbance associated with acute myocardial infarction and of means for detecting early signs of impending pump failure (18).

The development of a bedside method for catheterization of the left ventricle (19) has made it possible to evaluate left ventricular function with minimal disturbance in patients with acute myocardial infarction. The purpose of the present report is to describe the results of these studies in 40 patients with acute myocardial infarction, including 12 with clinical signs of left heart failure and 14 with shock.

METHODS

Subjects. Studies were performed in the Intensive Care Units of the Veterans Administration Hospital or Providence Hospital, Washington, D. C. Subjects studied were hospitalized with typical chest pain associated with electrocardiographic evidence of acute myocardial infarction. All were males except patients 9 and 14. All patients who survived over 24 hr exhibited elevated serum levels of glutamic oxaloacetic transaminase (SGOT). Patients with atypical symptoms or nonspecific electrocardiograms were excluded. In 38 subjects the ECG showed loss of forces indicative of transmural myocardial infarction, while in the other two the changes were typical of subendocardial infarction. Autopsies performed in 12 of the patients, who expired, confirmed the diagnosis of acute myocardial infarction.

14 patients had signs of shock at the time of study. Left ventricular function studies in this group were initiated in January, 1966. Shock was diagnosed by a fall in auscultatory systolic pressure to less than 90 mm Hg accompanied by one or more signs of inadequate peripheral blood flow, including oliguria (urine output less than 20 ml/hr), dulled sensorium, cool and clammy skin, and lactic acidosis. These patients were studied as soon as possible after the diagnosis of shock was made. The interval from onset of the acute myocardial infarction to study ranged from 2 hr to 36 days (Table II).

Studies in the 26 patients with acute myocardial infarction without shock were initiated in January, 1968. Patients were accepted for study only if the catheterization procedure could be accomplished within 24 hr from the onset of symptoms of myocardial infarction. Although studies were performed on a number of patients with no apparent complications, a particular attempt was made to include patients who exhibited some clinical signs of acute heart failure. Therefore, the subjects of this study do not represent a randomly selected group but rather a sample weighed in favor of more complicated cases. Patients were excluded if they had evidence of iliofemoral arterial disease, preexisting severe chronic heart failure, or other medical diseases (except hypertension) which might affect cardiac function.

In subjects conforming with the above criteria, permission for catheterization was obtained from the patient and/or his family. The nature of the procedure and the possible complications were described in detail. Studies in nine patients were excluded either because the left ventricle could not be catheterized or because severe hypotension prevented measurements from being made in a control steady state free of vasoactive drug administration.

Most patients had been treated with narcotics and sedatives as indicated, and these were continued during the procedure if the clinical situation warranted their administration. Some of the patients had been on long-term digitalis therapy before their admission, and a few had been treated with a diuretic before study. Indicated drug therapy was not withheld from the patients in shock while preparations for study were being made. If a sympathomimetic amine had been administered, this was usually discontinued while the hemodynamic status was closely monitored. If blood pressure stabilized after the drug was stopped for at least 30 min, results at this time were used as control observations. If the drug could not be discontinued because of a potentially dangerous fall in blood pressure, therapy was promptly reinstituted, and data on the patient were not included in this analysis.

Procedures. The left ventricle was catheterized with a modified red Kifa catheter via the femoral artery using the Seldinger technique (19). The right atrium was entered with a PE 160 catheter either through the femoral or antecubital veins. Pressures were measured with Statham P23Db and P23BB strain gage transducers amplified and recorded on multichannel direct-writing recorders (Hewlett-Packard or Waters). Left ventricular end-diastolic pressure (LVEDP) was calculated at the base of the rapid ventricular pressure rise or 0.05 sec after the onset of the QRS deflection of the electrocardiogram. Left ventricular mean diastolic pressure was taken as the average diastolic pressure before atrial contraction. The maximum rate of rise of left ventricular pressure (dp/dt) was calculated from pressure waves recorded at 100 mm/sec paper speed after the catheter was thoroughly flushed. Under ideal laboratory conditions the frequency response of the catheter and recording system was flat ±5% to 15 cycles/sec.

Cardiac output was determined by the dye dilution method. Indocyanine green, 5 mg, was injected as a bolus into the right atrium while blood was withdrawn at a constant rate from the left ventricle or ascending aorta through a Gilford or Waters cuvette. Calculation was made from the dye curves by the standard Stewart-Hamilton method. Dye curves were obtained in duplicate or triplicate in most patients and were reproducible within 15%. Cardiac output was measured in eight patients in the shock group. In three the urgent clinical situation prevented performance of more than one control dye curve. In four of the patients with shock, dye curves from the right atrium to left ventricle exhibited normal contour and exponential disappearance with total dye passage time averaging 34 sec. In three patients the dye curves were considerably prolonged (passage time ranged from 72 to 186 sec), and although an exponential decline could be plotted, early recirculation probably led to an underestimation of cardiac output (patients 32–34). Mean transit time and central blood volume were not calculated in the patient with the most prolonged curve. In one patient with very prolonged transit time from the right atrium to left ventricle, dye curves were recorded from the femoral artery after injection into the left ventricle. These curves exhibited rapid transit and excellent reproducibility.

Left ventricular stroke work (LVSW) in g-m/m^2 was calculated from the formula:

$$\frac{(\text{MAP} - \text{LVEDP}) \times \text{SV} \times 13.6}{1000},$$

where MAP is mean aortic pressure, and SV is stroke volume corrected for body surface area (ml/m^2). Central blood volume (CBV) in ml/m^2 was calculated from the formula: $\frac{\text{MTT} \times \text{CI}}{60}$, where MTT is the mean transit time from the right atrium to the left ventricle or ascending aorta, and CI is the cardiac index in liter/min per m^2. Systemic vascular resistance (SVR) in dyne·sec·cm^{-5} was calculated from the formula: $\frac{(\text{MAP} - \text{RAP}) \times 1332 \times 60}{\text{CO}}$, where RAP is mean right atrial pressure in mm Hg. PO_2 was measured by a Clark electrode and serum glutamic oxaloacetic transaminase (SGOT) was determined by the AutoAnalyzer.

Complications. Left ventricular catheterization was successful in over 80% of the attempts. Fluoroscopy was employed in the early cases, but the ease of passage of the catheter obviated the need for this aid. Failure was attributable in most cases to local atherosclerotic disease in the femoral and iliac arteries. Occasionally, the catheter would coil in the ascending aorta and fail to negotiate the aortic valve.

The following complications, which may have been related to the procedure, were observed: (*a*) *Ventricular tachycardia* in one case occurred several minutes after the catheter had been withdrawn from the left ventricle into the ascending aorta. The arrhythmia was terminated electrically and did not recur. The patient was subsequently discharged from the hospital. In two other cases a short run of ventricular tachycardia occurred when the ventricle was entered, and the procedure was abandoned. One of these patients was found to be severely hypokalemic at the time of study (serum K = 2.4 mEq/liter). Ventricular irritability was almost never observed after the catheter was positioned in the left ventricular cavity. Premature beats were more often associated with the right heart catheter if it was in the area of the tricuspid valve. These could be controlled by withdrawing the venous catheter a few centimeters. (*b*) *Atrial fibrillation* occurred in one case during the procedure and spontaneously reverted to sinus rhythm the following day. (*c*) Cerebral embolization occurred in one patient 16 hr after the procedure. The symptoms were mild hemiparesis and aphasia which cleared 2 days later. This patient succumbed to a second infarction on the 7th day of hospitalization, and the autopsy revealed large mural thrombi in the left ventricle. (*d*) Chest pain occurred during the procedure in several patients, but the causal relationship to the catheterization cannot be determined since all patients were studied early in the course of myocardial infarction when chest pain is a common symptom.

RESULTS

Clinical data. 26 patients with acute myocardial infarction without clinical signs of shock are described in Table I (group 1). 14 patients with clinical signs of shock are described in Table II (group 2). The mean age of group 1 patients was 51.6 yr, and of group 2, 63.4 yr ($P < 0.01$). None had cardiac murmurs at the time of study suggestive of mitral regurgitation. The incidence of previous myocardial infarctions in group 1 was 15.4% and in group 2, 35.7%. A history of hypertension was obtained in 54% of the patients in group 1 but only 14.3% in group 2. The mortality during hospitalization was 15.4% in group 1 and 85.7% in group 2. The location of the infarct defined by electrocardiogram and the incidence of preexisting angina pectoris or diabetes mellitus were not significantly different in the two groups.

Group 1 patients were subdivided on the basis of clinical and roentgenographic signs into two groups. Group 1a (patients 1–14, Table I) exhibited no signs of left ventricular failure. The lungs were clear to auscultation, and chest X-ray revealed no definite evidence of congestion. Group 1b (patients 15–26) presented with clinical evidence of heart failure manifested by persistent rales usually accompanied by X-ray evidence of pulmonary venous engorgement or interstitial edema. Several of these latter patients had no pulmonary symptoms, but most were aware of mild to moderate shortness of breath. A ventricular diastolic gallop was heard near the cardiac apex by at least two independent observers in all patients in group 1b but only two patients in group 1a. All group 1a patients recovered and were subsequently discharged from the hospital, whereas 33% of the group 1b patients succumbed during hospitalization. Of the four fatal cases, three had previous myocardial infarctions, and all four had a history of hypertension.

Serum transaminase (SGOT) was measured on admission and on successive days of hospitalization. In some patients who succumbed on the day of admission, only one or no blood samples were obtained. The peak SGOT value detected within the first 72 hr after onset of symptoms averaged 145 U (range 60–339) in group 1a, 186 U (range 100–360) in group 1b, and 532 U (range 15–2660) in group 2. SGOT returned to normal before the end of the 1st wk in all uncomplicated cases.

Hemodynamic studies. The hemodynamic data are presented in Tables III and IV for groups 1 and 2, respectively. The mean and standard deviation are indicated for group 1 and subgroups 1a and 1b in Table III and for group 2 in Table IV. The t values and probability (P) are shown in Table V and relevant correlation coefficients in Table VI.

Left ventricular end diastolic pressure (LVEDP) ranged from 6 to 40 mm Hg in group 1 and from 8 to 34 mm Hg in group 2. LVEDP was elevated (over 12 mm Hg) (20) in 10 of the 14 patients in group 1a who had no clinical signs of left ventricular failure. In group 1b LVEDP averaged 29.9 mm Hg and was higher than in group 1a (average 15.0 mm Hg) and also higher than in the shock group (average 21.1 mm Hg). Two of the four group 1a patients with normal LVEDP (patients 4 and 6) exhibited electrocardiographic evidence of subendocardial rather than transmural infarction. LVEDP was also normal in two group 2 patients, including one with a right ventricular infarction confirmed by autopsy. Left ventricular mean diastolic pressure (LVMDP) was significantly lower than LVEDP in group 1b (average difference 9.5 mm Hg), because of a significant atrial "kick." In group 1a LVEDP averaged only 3.6 mm Hg higher than LVMDP, and in the shock group the difference was only 2.9 mm Hg (Fig. 1). The magnitude of the atrial "kick" was significantly greater in group 1b than in the other groups ($P < 0.05$).

Mean right atrial pressure (RAP) was over 6 mm Hg in 10 of the 14 patients in group 1a, all of the patients in group 1b, and 10 of the 14 patients in shock. RAP was significantly higher in group 1b than in group 1a. RAP and LVEDP correlated better in shock ($r = 0.625$) than in the nonshock patients ($r = 0.460$) (Fig. 2). In three patients a normal RAP was associated with a normal LVEDP, but in the other five subjects, normal RAP coexisted with a modestly elevated LVEDP. In only two patients, (Nos. 7 and 39) was RAP higher than LVEDP. Both of these patients had electrocardiographic evidence

of diaphragmatic infarction, and patient 7 also had preexisting lung disease. Postmortem examination of patient 39 revealed extensive infarction of the right ventricle.

Cardiac output was measured in 16 group 1 patients and 8 group 2 patients. In 14 patients in group 1, cardiac index (CI) was over 2.5 liter/min per m^2, whereas in group 2 only one patient had a CI in excess of 2.5 liter/min per m^2. Despite the clinical signs of heart failure, CI was not significantly lower in group 1b patients than in the uncomplicated group 1a patients. LVEDP did not correlate significantly with CI in group 1, but a significant negative correlation was observed in group 2 ($r = -0.81$) (Fig. 3). Group 1b patients exhibited significantly faster heart rates than group 1a, but no significant difference between groups 1 and 2 was observed. A low but significantly positive correlation coefficient was noted between LVEDP and heart rate in group 1 ($r = 0.41$), but in the shock group the correlation tended to be negative ($r = -0.30$). Despite the significant differences in RAP and LVEDP between the groups, calculated central blood volume (CBV) was practically identical for each group.

Stroke volume index (SV) was within the normal range (38.8 ml/m^2) in group 1a, but was moderately reduced in group 1b (26.3 ml/m^2) and markedly reduced in shock (16.5 ml/m^2). The differences between each group were significant. A significant negative correlation was observed between LVEDP and SV in group 1 ($r = -0.52$), and group 2 ($r = -0.66$). LVSW also fell progressively from group 1a to group 2. LVEDP and LVSW also correlated significantly in group 1 ($r = -0.68$) and group 2 ($r = -0.62$). The scatter (Fig. 4) shows little overlap between the groups.

The first derivative of left ventricular pressure (dp/dt) was significantly lower in group 2 than in group 1, and the dp/dt was directly correlated with LVEDP ($r = 0.60$). However, absolute values for dp/dt obtained

TABLE I

Clinical Data from Group 1 Patients (Nonshock)

Patient	Age	BSA	Hypertension	Diabetes	Previous myocardial infarction	Angina pectoris	Location of myocardial infarction	Mortality
		m^2						
1	38	2.02					P	
2	56	1.83					A	
3	45	1.87					P	
4	51	1.90	+				P	
5	33	2.13	+				P	
6	39	2.02					A	
7	54	1.79					P	
8	43	1.90					P	
9	36	1.73	+			+	P	
10	45	1.77					A	
11	47	1.84		+		+	A	
12	49	1.94					A	
13	50	2.42	+	+			P	
14	46	1.47					A	
15	48	1.80					P	
16	73	2.06	+			+	P	+
17	53	2.18	+				A	+
18	60	1.80				+	A	
19	49	2.11	+	+	+	+	A	
20	55	2.12	+	+	+	+	A	+
21	50	1.98			+	+	I	
22	73	1.68	+		+	+	P	+
23	44	1.88	+				P	
24	59	1.75	+			+	P	
25	51	1.90					A	
26	75	1.59	+			+	A	

BSA = body surface area; A = anterior wall myocardial infarction; I = indeterminate location; P = posterior or diaphragmatic wall infarction.

TABLE II

Clinical Data from Group 2 Patients (Shock)

Patient	Age	BSA	Time from onset	Hyper-tension	Diabetes	Previous myocardial infarction	Angina pectoris	Location of myocardia infarction	Mortality
		m^2	*hr*						
27	54	1.88	11				+	A	+
28	71	1.97	76		+			I	+
29	66	1.97	2	+		+		P	
30	54	1.59	12				+	I	+
31	53	1.78	120		+	+	+	A	+
32	61	1.80	48	+		+	+	A	+
33	72	1.51	36 days			?	+	A	+
34	79	1.86	72			+	+	P	+
35	61	2.03	72		+	+	+	P	+
36	60	1.59	7 days				+	A	+
37	72	2.04	24						+
38	58	1.60	12						
39	63	1.85	10 days					P	+
40	64	1.59	72				+	A	+

Abbreviations same as in Table I.

through this catheter system should be interpreted with caution, and comparison between patients may be unreliable.

Systemic vascular resistance (SVR) tended to be normal in group 1 patients but was often elevated in shock; however, the wide scatter of values in the group 2 patients prevented this difference from reaching statistical significance.

Arterial oxygen tension (PO_2) was measured only in those patients to whom O_2 was not administered or in whom it was elected to interrupt oxygen therapy. Oxygen administration was not discontinued in any of the group 2 patients. PO_2 was inversely correlated with LVEDP in the nonshock patients, being significantly higher in group 1a than in group 1b ($r = -0.59$) (Fig. 5).

DISCUSSION

The data obtained in the present study reveal that a disturbance in left ventricular function within the first 24 hr after an acute myocardial infarction is considerably more frequent than previous clinical observations have implied (21–23). Only 4 of 26 patients without shock had a normal LVEDP, and two of these had electrocardiographic evidence of subendocardial rather than transmural infarction. Not only was LVEDP frequently elevated in the absence of signs of left ventricular failure, but patients with only mild signs or symptoms of pulmonary congestion sometimes exhibited elevations in LVEDP to levels usually thought to be associated with significant pulmonary capillary transudation. Only one of seven patients with LVEDP from 25 to 40 mm Hg had clinical signs of pulmonary edema at the time of study.

It must be recognized that a high LVEDP does not necessarily signify left ventricular dilatation (24). Since angina (25) and experimental myocardial infarction (26) have been thought to produce changes in compliance, the elevated LVEDP in some of the patients in this series could be related to decreased distensibility of the ventricles during diastole. Such a reduction in compliance might help to explain the observation that the central blood volumes showed no correlation with LVEDP or signs of heart failure. These central blood volume measurements were calculated from mean transit times from the right atrium to the left ventricle or ascending aorta and, therefore, represent exclusively cardiac and pulmonary blood volume. If the ventricle were operating on a very steep pressure volume curve, increments in ventricular volume too small to be detected by this method could account for the elevated LVEDP. The pulmonary vascular bed also may be nondistensible in left heart failure (27, 28), and thus pulmonary blood volume might not be much larger in patients with higher LVEDP. Since the LVEDP was elevated in nearly all the patients in this series, it is possible that the pulmonary blood volume was uniformly expanded and, therefore, bore no relationship to the absolute level of the LVEDP.

Although a high LVEDP may not necessarily indicate left heart failure, patients with LVEDP over 19 mm Hg nearly always exhibited clinical or radiological signs of pulmonary congestion. LVEDP averaged 29.9 mm Hg in the patients with clinically diagnosed left ventricular failure compared with an average of 15.0 mm Hg in the patients without signs of heart failure. Cardiac index was not different in the two groups, but the heart rate was significantly faster and the stroke volume and stroke work lower in the patients with heart failure. Therefore, an increase in LVEDP and tachycardia apparently represent compensatory mechanisms by which cardiac output is maintained after severe myocardial infarction. A positive inotropic effect of reflex sympathoadrenal activation may also play a role in supporting cardiac performance (29). These observations suggest that the patients with clinical signs of heart failure had more severe impairment of left ventricular performance, but the data do not permit conclusions as to whether the cardiac dysfunction is due to: (*a*) a larger noncontracting area of myocardium; (*b*) paradoxical pulsations (dyskenesia) of the infarcted area; or (*c*) failure of the uninvolved myocardium to adequately compensate by increased fiber shortening (30).

Although cardiac output is usually markedly reduced in patients with myocardial infarction shock, the central

TABLE III
Hemodynamic Data for Group 1 Patients

Patient	MAP	LVEDP	LVMDP	RAP	CI	HR	SV	LVSW	dp/dt	CBV	SVR
	mm Hg	*mm Hg*	*mm Hg*	*mm Hg*	*liter/min per m²*	*beats/min*	*ml/beats per m²*	*g-m/m²*		*ml/m²*	*dynes·sec ·cm⁻⁵*
Group 1a											
1	96	15.0	12	4		62			1360		
2	124	18.5	8	8	2.54	90	28.2	40.5	2608	641	2094
3	87	13.0	9	4		75			2070		
4	116	10.0	8	4		67			1960		
5	120	17.0	12	12	3.45	88	39.1	54.7	2496	601	1177
6	102	7.5	6	4	3.60	68	52.9	68.0	2556	593	1078
7	83	6.0	6	12		66					
8	76	13.5	12	10		78					
9	75	16.5	13	10	2.70	56	48.1	38.3	1530	669	1115
10	94	12.0	10	9	2.94	72	40.9	45.6	2240	487	1305
11	135	18.5	15	7	2.85	95	30.0	47.5	2258	660	1954
12	73	22.5	16	16		64			2176		
13	98	20.5	18	12		120			1320		
14	145	20.0	15	12	2.81	96	29.2	49.6	2023	831	2432
Mean	101.7	15.0	11.4	8.8	2.98	78.4	38.3	49.2	2050	640.3	1590
SD±	23.0	5.0	3.8	3.8	0.39	17.5	9.8	10.0	440	104	533
Group 1b											
15	73	32.0	21	7		95			1590		
16	100	19.0	10	12	2.59	114	22.8	25.1	2480	531	1317
17	99	38.5	22	9	2.90	104	27.9	23.0	1965	685	1124
18	112	20.0	19	11	3.06	96	31.8	39.8	1597	635	1352
19	84	34.0	12	11	2.72	116	23.4	15.9	1480	606	1087
20	130	42.0	32	17		86					
21	99	22.0	19	9		78					
22	116.	29.0	20	12	2.54	104	24.4	28.9	1824	750	1993
23	104	35.0	26	11	3.38	128	26.4	24.8		659	1169
24	96	24.0	18	13	2.29	75	30.6	30.0	2000	498	1651
25	128	40.0	30	12	2.78	81	34.4	41.2	2250	921	1750
26	136	23.0	16	16	1.42	95	15.0	23.1	1520	476	4230
Mean	106.4	29.9	20.4	11.7	2.63	97.7	26.3	28.0	1870	640.1	1740
SD±	18.8	8.2	6.5	2.8	0.55	16.3	5.8	8.2	350	138.6	983
Group 1 (a and b)											
Mean	103.9	21.9	15.6	10.1	2.79	87.3	31.6	37.3	1960	640.2	1680
SD±	20.9	10.0	6.9	3.6	0.51	19.3	9.7	13.9	410	120.7	802

TABLE IV

Hemodynamic Data from Group 2 Patients

Patient	MAP	LVEDP	LVMDP	RAP	CI	HR	SV	LVSW	dp/dt	CBV	SVR
	mm Hg	*mm Hg*	*mm Hg*	*mm Hg*	*liter/min per m²*	*beats/min*	*ml/beats per m²*	*g-m/m²*		*ml/m²*	*dyne·sec ·cm⁻⁵*
27	57	33	33	25		80			564		
28	67	18	16	6		90					
29	70	34	27	18		102					
30	74	24	16	14		90			1280		
31	76	30	26	14		96			1080		
32	98	14	10	3	0.99	126	7.8	8.9	1320	810	4245
33	92	30	24	12	0.58	74	7.8	6.6	960		7356
34	54	24	24	5	0.98	120	8.2	3.3	448	607	2579
35	60	22	22	11	1.71	75	22.8	11.8	1280	543	1123
36	86	16	11	8	2.07	108	19.1	18.2	1700	697	1897
37	66	17	17	8	1.48	114	13.0	8.7	840	506	1537
38	70	8	5	6	2.68	108	24.8	20.1	2880		1196
39	36.	8	8	14	2.25	78	28.8	11.0	1280		586
40	70	18	16	13		116			540		
Mean	69.7	21.1	18.2	11.2	1.59	98.4	16.5	11.1	1180	637.4	2560
SD±	15.9	8.5	8.1	5.9	0.72	17.6	8.4	5.7	650	102.5	2241

venous pressure is not always elevated (2, 13, 14, 31–33). Therefore, it has not been clear whether shock represents a particularly severe degree of left ventricular failure or the sequelae of some other hemodynamic abnormality. In the present series of patients with shock a profound reduction of cardiac output, stroke volume and stroke work was usually accompanied by an elevated LVEDP indicative of severe impairment of cardiac function. Since all these indices of cardiac performance were inversely correlated with LVEDP in the shock group, it is apparent that the level of the LVEDP serves as a useful guide to the severity of pump failure in shock.

No data are available to quantitate either infarct size or ventricular contractility in these patients. The rise in SGOT can serve as only a rough guide to the extent of myocardial necrosis because of the random timing of sample collection and the transaminase contribution from other sources when there is circulatory congestion or shock. Although the rate of pressure generation by the left ventricle is a sensitive guide to its contractile force (34), the maximum dp/dt measured through a catheter system might yield unreliable results because of its limited frequency response. Furthermore, dp/dt is influenced by the level of LVEDP, the aortic pressure, and the heart rate (35). Nonetheless, the significantly lower dp/dt in the shock group when compared with the group 1 patients and the close negative correlation between LVEDP and dp/dt in shock is at least consistent with the other evidence suggesting that shock usually is characterized by a more severe degree of left ventricular failure. Whether these signs of ventricular dysfunction represent the direct consequences of an extensive myocardial infarction or the secondary effects of the shock on ventricular performance cannot be ascertained.

Despite the evidence for severe myocardial impairment in most patients with shock, two subjects with mild signs of shock exhibited a normal LVEDP, and the average LVEDP in the shock group was significantly lower than in the patients with heart failure who were maintaining apparently normal peripheral circulation. Thus, it is possible that inadequate left ventricular dilatation, perhaps related to reduced venous return, was a contributing factor to the reduction in cardiac output in

TABLE V

Significance of Differences in Hemodynamic Measurements between Patient Groups (P)

Group	MAP	LVEDP	LVMDP	RAP	CI	HR	SV	LVSW	dp/dt	CBV	SVR
1 vs. 2	<0.001	NS	NS	NS	<0.001	<0.1	0.001	<0.001	<0.001	NS	<0.02
1a vs. 1b	NS	<0.001	<0.001	<0.05	NS	<0.01	<0.01	<0.001	NS	NS	NS
1b vs. 2	<0.001	<0.02	NS	NS	<0.01	NS	<0.02	<0.001	<0.02	NS	NS

TABLE VI

Correlation Coefficients between LVEDP and Selected Hemodynamic Measurements

	Group		
	1	2	1 + 2
RAP	+0.46	+0.62	+0.49
CI	−0.15	−0.81	−0.03
HR	+0.41	−0.30	+0.21
SV	−0.52	−0.66	−0.24
LVSW	−0.68	−0.62	−0.19
dp/dt	−0.24	−0.66	−0.31

some of these patients. Previous studies have demonstrated that some patients with myocardial infarction shock improve after plasma volume expansion (36–39).

Another possible explanation for the lower LVEDP in shock when compared with the heart failure group is a relatively increased ventricular compliance in shock. In the group with heart failure atrial contraction was associated with a prominent presystolic rise in left ventricular pressure averaging 9.5 mm Hg, whereas in the shock group the atrial "kick" was significantly smaller although the mean ventricular diastolic pressures before atrial systole were similar. It is possible that the absence of a significant atrial "kick" in shock merely reflects a smaller atrial stroke volume, but the findings also could be explained by differences in ventricular compliance. Either the infarcted left ventricle may be relatively non-

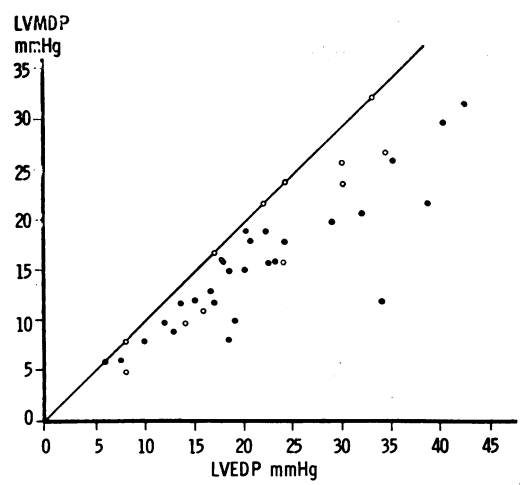

FIGURE 1 The relationship between left ventricular end-diastolic pressure (LVEDP) and left ventricular mean diastolic pressure (LVMDP). The closed circles represent patients from group 1, the open circles patients from group 2. The diagonal line represents the line of identity. At higher pressures patients with shock exhibit a smaller difference between LVMDP and LVEDP than in the nonshock group.

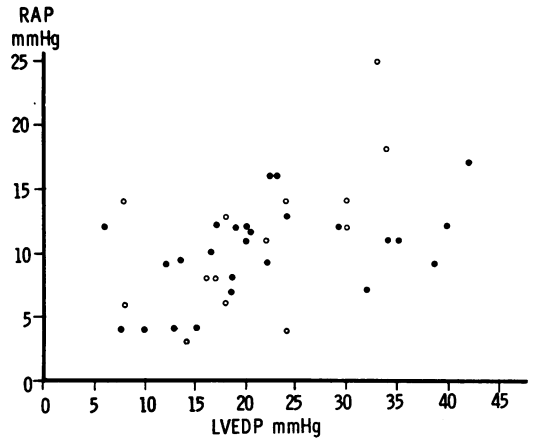

FIGURE 2 The relationship between left ventricular end-diastolic pressure (LVEDP) and right atrial pressure (RAP). Symbols same as in Fig. 1. Although correlation is significant ($r = 0.49$), a wide scatter is observed.

compliant in the absence of shock, or shock is associated with increased compliance due to generalized myocardial hypoxia or to large dyskinetic segments of the myocardium (35). The results also might have been influenced by the fact that nine of the patients with shock were studied more than 24 hr after the onset of their myocardial infarction, whereas the nonshock group all were studied in the first day.

In several respects the shock group did not exhibit a homogenous hemodynamic pattern. Tachycardia, which appeared to be a predictable compensatory mechanism in the nonshock group with reduced stroke volume, was not uniformly present in shock. Indeed, heart rate tended to be negatively correlated with LVEDP in shock, and the low cardiac output in some patients could be at-

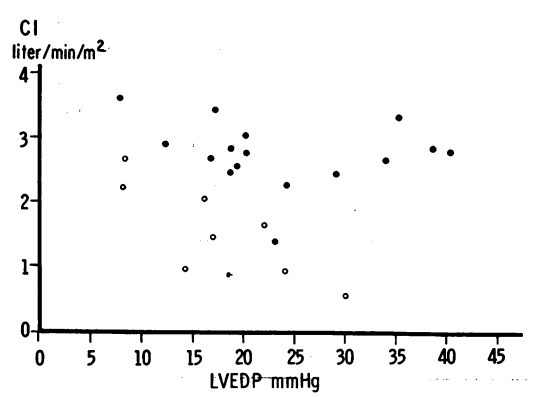

FIGURE 3 The relationship between left ventricular end-diastolic pressure (LVEDP) and cardiac index (CI). Symbols same as in Fig. 1. CI is maintained at normal levels in group 1 despite elevated LVEDP, whereas CI is inversely related to LVEDP in the shock group ($r = -0.81$).

tributed in part to the failure of the heart to increase its rate appropriately. However, the persistence of a slow heart rate in the face of a falling stroke volume in shock may represent an intrinsic protective device by which the ischemic heart defends itself against a tachycardia-induced increase in oxygen demand (40).

An increase in peripheral resistance, which might be expected as a compensatory response to a fall in cardiac output, was not uniformly present in the hypotensive patients with shock. Although vasoconstriction may have occurred in some vascular beds in these patients, the fact that total calculated resistance did not rise in all patients suggests that large vascular beds either failed to constrict or were actually dilated. Whether this failure of peripheral resistance to increase in some patients is an important pathogenetic factor in the shock (41), or merely represents a natural variation in the intensity of sympathetic reflex vasoconstrictor activity (42) cannot be determined. However, since heart rate and peripheral resistance both may fail to react appropriately in shock, it is possible that acidosis (43) or cerebral hypoxia (44) may be responsible for inhibiting normal cardiovascular reactivity.

The reliability of right atrial or central venous pressure (CVP) as an index of the severity of left ventricular failure in myocardial infarction is a subject of considerable clinical importance. RAP was elevated in 29 of the 34 patients with abnormal LVEDP in this series, and RAP was elevated in all but one patient with an LVEDP over 19 mm Hg. However, the correlation between RAP and LVEDP was not close ($r = 0.495$). In two patients the right atrial pressure was higher than the LVEDP, whereas in the others LVEDP surpassed RAP by from 2 to 29.5 mm Hg. Therefore, the absolute level

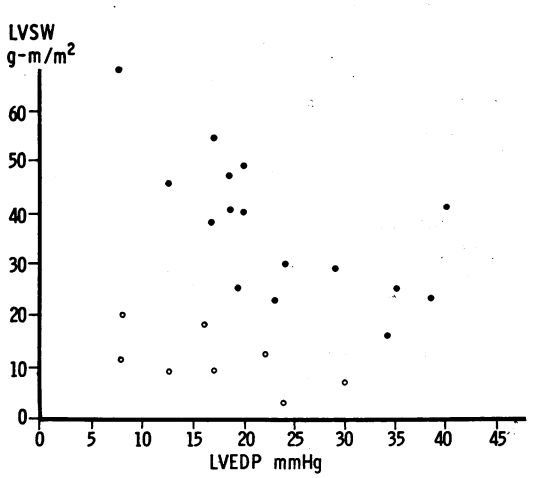

FIGURE 4 The relationship between left ventricular end-diastolic pressure (LVEDP) and left ventricular stroke work index (LVSW). Symbols same as in Fig. 1. A significant negative correlation is observed in both groups.

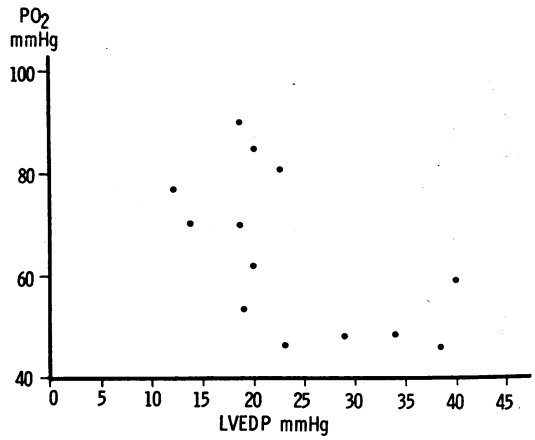

FIGURE 5 The relationship between left ventricular end-diastolic pressure and arterial oxygen tension (PO_2) ($r = -0.59$).

of the CVP may be of some value in detecting the presence of an elevated LVEDP, but it is not a dependable guide to the magnitude of the increase in left ventricular filling pressure.

Changes in CVP during volume expansion have previously been shown to serve as a fairly accurate reflection of changes in LVEDP in myocardial infarction shock (35). This relationship might not hold in the absence of shock, however, if left ventricular compliance were reduced. In the present series RAP was better correlated with LVEDP in shock than in the nonshock group. If a high LVEDP in patients with a nearly normal RAP is indicative of reduced left ventricular distensibility, it is possible that the RAP might actually be a more reliable guide to chamber dilatation.

The low arterial oxygen tension observed in acute myocardial infarction has been subjected to extensive study (45, 46). Although it is likely that arterial hypoxemia is of multiple etiologies in these patients, the significant negative correlation noted between PO_2 and LVEDP indicates that pulmonary venous hypertension is an important contributing factor. Since the high LVEDP often was not accompanied by signs of pulmonary congestion, these observations may help to explain the dilemma of other investigators who have observed hypoxemia in the absence of clinical signs of left ventricular failure (47).

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