Spontaneous Changes in Left Ventricular Function Over the First 24 Hours of Acute Myocardial Infarction: Implications for Evaluating Early Therapeutic Interventions

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SUMMARY **The spontaneous changes in left ventricular ejection fraction (LVEF) during the first 24 hours of a first transmural infarction were assessed in 34 patients by serial gated cardiac blood pool imaging. Major therapeutic interventions with a view to limit infarct size were not used. Four determinations of LVEF were performed. Study 1 was performed as soon as possible after admission to the hospital. Studies 2 and 3 were performed 2 and 4 hours, respectively, after study 1. Twenty-four patients (70%) had study 1 within 6 hours after the onset of acute chest pain and 10 had it 6–12 hours after the onset of chest pain. Study 4 was performed 24 hours after the onset of chest pain. Compared with study 1, 19 of 34 patients (56%) had spontaneous changes in LVEF in at least one of the subsequent studies, exceeding the expected variability in stable patients. The changes ranged from a 32% increase to 14% absolute decrease. LVEF improved in 11 patients and deteriorated in eight. These spontaneous changes in left ventricular performance indicate that a single assessment of LVEF during the early hours of transmural myocardial infarction may not properly characterize cardiac performance in an individual patient and may not be the most appropriate reference against which to compare subsequent evolution of left ventricular function. These data may have implications for studies of the effects of early therapeutic interventions on LVEF.**

IN EXPERIMENTAL MODELS of infarction, ischemic myocardium can be salvaged by interventions that improve the balance between myocardial oxygen supply and demand.[1–6] However, such clear-cut therapeutic benefit in human myocardial infarction has not been demonstrated.[7–13] One reason for this may be limitations in the techniques suitable for objective documentation of myocardial salvage in man. Recently, radionuclide techniques such as multigated cardiac blood pool imaging have been proposed for noninvasive assessment of global cardiac performance at the patient's bedside, thereby providing functional estimates of myocardial salvage. However, little is known about the spontaneous evolution of left ventricular (LV) performance during the early hours of acute infarction. It is during the first 6–12 hours that interventions probably will have their greatest therapeutic impact. In order for these measurements to be applied to this patient population, their variability during this period must be established. Thus, we performed serial assessment of LV ejection fraction (LVEF) using gated cardiac blood pool imaging during the first 24 hours of acute transmural myocardial infarction in a group of patients in whom major therapeutic interventions were not used. Our results indicate that substantial changes in global LV performance occur spontaneously during the early hours of infarction in a number of these patients.

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Methods

Patients

Thirty-four patients with their first transmural acute myocardial infarction were studied. Their mean age was 63 years (range 36–86 years). All had a typical history of myocardial infarction, and all demonstrated classic evolutionary electrocardiographic and serum-creatine kinase changes as defined by standard criteria.[14] In all instances, the first of the serial multigated equilibrium cardiac blood pool studies was performed at the bedside within 12 hours after the onset of chest pain. The patients entered in this study do not represent a consecutive series, but rather those who could be studied within the logistic constraints of our coronary care unit imaging program early enough in the course of the infarct. Patients with cardiogenic shock were excluded, as were patients with antecedent valvular or myocardial disease. Fifteen patients had a history of hypertension, but only eight were receiving antihypertensive medication at the time of admission (diuretics alone in four patients and diuretics in combination with a second drug in four). Two patients had a history of stable angina pectoris preceding the infarct, and both were treated with propranolol (maximal dose, 20 mg four times daily) and nitrates. Two patients were receiving procainamide for ventricular ectopy before admission. In all patients, preadmission medications were not continued. Before entering the study, written, informed consent was obtained in all patients.

Twenty-five patients had inferior wall myocardial infarction and nine had anterior wall infarction. This distribution approximates the anatomic distribution of acute myocardial infarction seen in our coronary care unit during the same period. Thirteen of the patients with inferior wall infarction had $>$ 1 mm of ST-segment depression in the precordial leads. This finding is of potential prognostic significance.[15]

The standard infarct treatment regimen instituted in

the coronary care unit was applied to all patients and consisted of morphine sulphate or mepiridine for relief of pain; prophylactic i.v. lidocaine (3 mg/min, after two appropriately spaced initial bolus injections); diazepam for sedation; heparin, 5000 U subcutaneously; oxygen by face mask or nasal cannula; and bedrest. No patient received any medication to limit infarct size, specifically, β-adrenergic blockers, slow-channel calcium antagonists, nitrates, or afterload reducers.

Imaging Protocol

All patients underwent bedside serial multigated equilibrium cardiac blood pool imaging studies. Study 1 was performed as soon as possible after arrival in the coronary care unit. Twenty-four (70%) of the 34 patients had the first multigated cardiac blood pool study performed within 6 hours (range 3–6 hours) after the onset of acute chest pain. In 10 patients, the study was performed 6–12 hours after the onset of chest pain. The mean (± SD) time after onset of chest pain for the first study was 6 ± 2 hours. There was no difference in the timing of study 1 after onset of chest pain in patients with inferior or anterior wall infarction (6 ± 3 vs 5 ± 2 hours, NS).

To assess LV ejection fraction (LVEF), study 1 was performed in the left anterior oblique position using a 10° caudal tilt; the obliquity chosen provided the best delineation of the septum. Studies 2 and 3 were performed 2 hours and 4 hours, respectively, after the first study. Study 4 was performed the next day, approximately 24 hours after the onset of acute infarction. At the time of each study, the patient's clinical status (Killip class[16]) was assessed and heart rate and blood pressure were recorded.

Multigated Equilibrium Cardiac Blood Pool Imaging

A mobile computerized scintillation camera (DYNAMO, Picker Corp, interfaced with a MUGACART Medical Data Systems) was used in 14 patients and a Sigma 420 camera interfaced with VIP 550 (Technicare) was used in 20 patients for bedside multigated equilibrium cardiac blood pool imaging. All studies in a given patient were obtained only with one system. For all studies, in vivo labeling of the patient's own red blood cells with technetium 99m-pertechnetate (25–30 mCi) was used according to standard techniques.[17] The energy window (20%) of the gamma camera was set symmetrically around the 140-keV photopeak. The scintillation data were acquired in gated frame mode using the R wave of the ECG as the synchronizing impulse and stored in computer core memory (64 × 64-word mode). The cardiac cycle was divided in 20–28 equal frames (each less than 40 msec in duration). A total of 175,000–250,000 counts/frame were accumulated in the whole field of view, resulting in 6000–20,000 counts in the left ventricle at end-diastole.

Assessment of LVEF

The processing of studies was performed using commercially available software. A variable region of interest was used. The edge of the left ventricle was defined as second derivative points on the count profile in each of the 28 frames using the MUGE program (Medical Data Systems). Using the QMICA program (Technicare), a varying region of interest was determined by changing the count threshold in four quadrants of the left ventricle so that these regions of interest by visual inspection accurately followed the outline of the contracting left ventricle throughout the cardiac cycle. LVEF was determined using background-corrected counts derived with varying regions of interest: (end-diastolic counts − end-systolic counts)/end-diastolic counts.

Definition of Significant Change in LVEF

Studies from this laboratory have documented the intrinsic variability of LVEF using the techniques of the present study in stable patients.[18] The mean variability of LVEF was significantly larger ($p < 0.01$) in patients with a normal LVEF (≥ 55%) than in those with an abnormal LVEF, when studies were performed twice on the same day and on different days. In 97% of the patients with abnormal LVEF, the variability was 5% (absolute) or smaller. However, 50% of the patients with normal LVEF exceeded this degree of variability and in 97% of the normal patients a variability as large as 12% occurred. This differential variability should be considered. Therefore, changes in LVEF greater than 5% in abnormal patients and greater than 12% in normal patients are significant changes.

Reproducibility and Quality Control

Previous studies from this laboratory established the inter- and intraobserver variability of computer assessment of LVEF. Using the two computer softwares, mean intraobserver variability was comparable (1.4 ± 1.2% for MUGE and 2.0 ± 1.3% for QMICA, NS). The variability was not different for normal or abnormal LVEFs.[18] All 131 multigated equilibrium cardiac blood pool studies in the present investigation were reprocessed four times by one of us without knowledge of clinical data or sequence of studies. The LVEF values in the present study represent the mean of these four determinations. To exclude unintentional bias in processing, random studies were reprocessed after several weeks and compared with previously noted values. The variations (range 0–6%, mean 2.5 ± 1.7%) that occurred were within the previously assessed intraobserver variability.[18]

Statistical Analysis

Data are presented as the mean ± SD. Changes in LVEF and variabilities are expressed as absolute values of ejection fraction units; e.g., LVEF changing from 42% to 50% is an 8% change. An LVEF of 55% or more was considered normal. Since the variability of LVEF is dependent on baseline LVEF,[18] the results in patients with abnormal LVEF in study 1 were analyzed separately from those with normal LVEF. Serial data were analyzed by analysis of variance for repeated measures. Differences among defined groups were

compared using a t test. Data in individual patients were analyzed with the paired t test. The incidence of various factors were compared by a chi-square test. A p value < 0.05 was considered significant.

Results

Clinical Course

At the time of study 1, 26 patients were in Killip class I, six in class II and two in class III. Of the 34 study patients, two died shortly after study 3, within the first 24 hours. One patient died of cardiac rupture, the other of progressive pump failure. Of the surviving patients, one developed rupture of the ventricular septum between study 3 and 4 and underwent surgical repair 2 days later. None of the remaining patients had evidence of infarct extension (new chest pain, new electrocardiographic changes) during the 24-hour study period. Five patients manifested symptoms of left ventricular failure at the time of study 4. The clinical data of the whole group are summarized in table 1. Tables 2A–C give the data in individual patients.

Comparison of LVEF in Studies 1, 2, 3 and 4

Analysis of the data from individual patients revealed substantial spontaneous changes in LVEF over the 24-hour period. In the serial studies, both increases and decreases in LVEF were noted frequently. Compared with study 1, these changes ranged from a 32% absolute increase to 14% absolute decrease. The mean interstudy variation of LVEF was not related to the timing of the study during the 24-hour study period. The mean interstudy variability of LVEF in patients with acute myocardial infarction and initial abnormal LVEF (< 55%) was 5 ± 5% for studies performed on the first day (study 1 compared with studies 2 and 3), and was 9 ± 8% for studies performed on different days (study 1 compared with study 4). Both mean variabilities are significantly greater ($p < 0.02$) than those observed in a comparable group of stable patients.[18] In contrast, the mean interstudy variability of LVEF in patients with acute myocardial infarction and initial normal LVEF was 6 ± 5% for studies on the first day and 6 ± 4% for studies on different days. Neither value is significantly different from the mean variability observed in a comparable stable group of patients.[18]

Figure 1 displays the spontaneous changes in LVEF compared with study 1 in individual patients. In patients with initial LVEF < 55%, 16 of 27 patients (59%) demonstrated changes in LVEF > 5% on any of the serial studies (study 2 vs 1, 3 vs 1, or 4 vs 1). In 10 patients (37%), LVEF improved, in six patients (22%) it deteriorated and in 11 patients (41%) it did not change significantly. Three of 27 patients with an abnormal LVEF at study 1 had a normal LVEF at study 4. Of the seven patients with an LVEF ≥ 55% at the initial study, three had a change in LVEF of > 12%; in two patients LVEF decreased; in one patient it improved; and in four patients LVEF did not change significantly. One of the seven patients with a normal LVEF at study 1 had an abnormal LVEF at study 4. Thus, of 34 patients with acute transmural myocardial infarction, LVEF changed significantly in 19 patients (56%): 11 patients improved and eight deteriorated.

Analysis of variance of the serial studies in the overall group of 34 patients did not reveal a systematic change in LVEF during the 24-hour study period. The same was true when the patients were separated according to the location of infarction (table 1).

Changes in LVEF and Clinical Variables

In order to analyze whether the 11 patients who demonstrated improvement of LVEF (group A) could be distinguished from the eight patients who demonstrated deterioration of LVEF (group B), these two groups of patients were compared (tables 2A and 2B). At the time of study 1, the patients in group A had significantly lower mean LVEFs than the patients in group B, 37 ± 13% vs 48 ± 12% ($p < 0.05$). At the time of study 1, six of 11 patients in group A and seven of eight patients in group B were Killip class I (NS).

At the time of study 1, the mean heart rate was significantly higher in group A than in group B, 90 ± 21 vs 69 ± 16 beats/min ($p < 0.05$). The mean blood pressure was not different between the two groups, 101 ± 21 mm Hg vs 95 ± 11 mm Hg (NS). The mean rate-pressure product at the time of study 1 was significantly higher in group A than in group B, 92 ± 31 × 10^{-2} vs 65 ± 13 × 10^{-2} ($p < 0.05$). At the time of study 4, the mean rate-pressure product in group A had decreased significantly from the original value, to 75 ± 19 × 10^{-2} ($p < 0.01$). Although the mean heart rate did not change significantly, mean blood pressure decreased in group A, to 88 ± 6 mm Hg ($p < 0.05$). No such change was observed in group B. At the time

TABLE 1. *Killip Classification, Heart Rate, Mean Blood Pressure, Rate-Pressure Product and Left Ventricular Ejection Fraction in All Patients at the Time of Studies 1, 2, 3 and 4*

Study	n	Killip class I	II	III	$\overline{HR}$ (beats/min)	$\overline{BP}$ (mm Hg)	$\overline{RPP}$ (× 10^{-2})	LVEF (%) All	Inf	Ant
1	34	26	6	2	79 ± 19	100 ± 17	79 ± 25	43 ± 14	45 ± 15	36 ± 6
2	34	25	9		78 ± 19	96 ± 21	75 ± 21	44 ± 13	47 ± 12	34 ± 7
3	34	25	8	1	79 ± 17	97 ± 13	78 ± 19	44 ± 14	46 ± 15	36 ± 5
4	32	27	4	1	77 ± 17	92 ± 9	71 ± 16	47 ± 12	50 ± 11	37 ± 9

Data are mean ± SD.

By analysis of variance, there was no significant difference between studies in any variable.

Abbreviations: Ant = anterior wall infarct; $\overline{BP}$ = mean blood pressure; $\overline{HR}$ = mean heart rate; Inf = inferior wall infarct; LVEF = left ventricular ejection fraction; $\overline{RPP}$ = mean rate-pressure product.

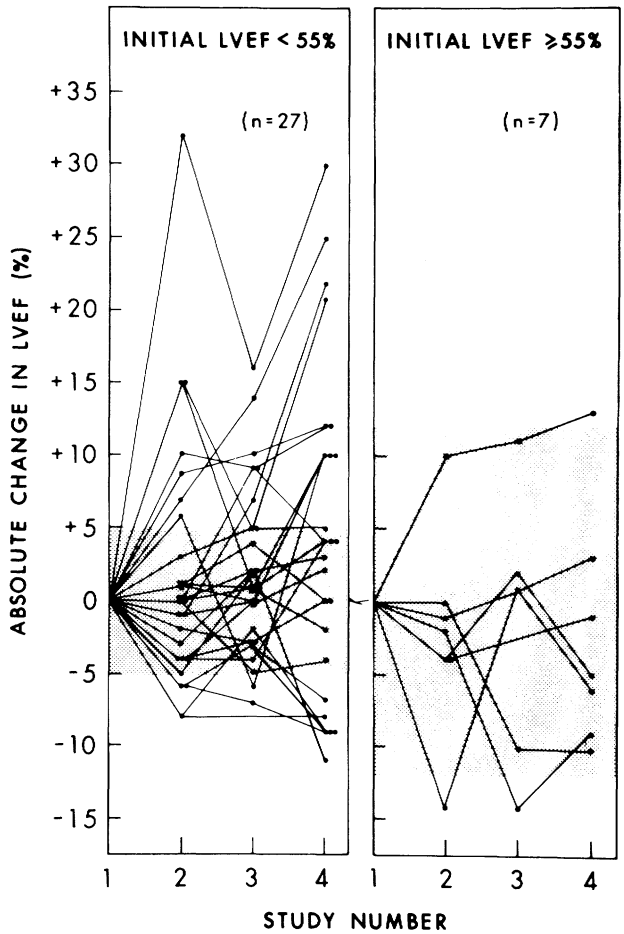

FIGURE 1. *Spontaneous change in left ventricular ejection fraction (LVEF) in individual patients in studies 2, 3 and 4 compared with study 1. The shaded zones indicate the anticipated variability in either direction of LVEF observed previously in 97% of stable patients without acute infarction.[18] In patients with abnormal LVEF (< 55%) changes >5% (absolute), and in patients with normal LVEF (≥ 55%) changes > 12% (absolute) are considered significant and nonrandom changes. Sixteen of the patients with abnormal LVEF and three with normal LVEF had significant changes in LVEF compared with study 1 during the first 24 hours of acute myocardial infarction.*

of study 4, the mean rate-pressure products in groups A and B were not different.

In individual patients, there was no correlation between either direction or magnitude of changes in heart rate ($r = -0.08$), mean blood pressure ($r = -0.09$) or rate-pressure product ($r = -0.17$) and changes of LVEF (tables 2A and B). There was no significant difference in peak serum enzyme levels or the distribution of infarct location between groups A and B. The incidence of precordial ST-segment depression in patients with inferior wall infarcts was not different in the two groups (four of nine patients in group A and three of five in group B).

Discussion

This study demonstrates that 56% of patients with acute myocardial infarction may have significant spontaneous changes of LVEF during the early hours of infarction, in the absence of major therapeutic interventions. These absolute changes, which are most evident in patients with abnormal baseline function, occurred in either direction, and in the present study ranged from an increase of 32% to a decrease of 14%. These changes are greater than the variability of LVEF observed in stable patients.[18] These data may have implications for studies evaluating the effects of early therapeutic interventions on LV performance.[7-13, 19, 20] For example, the beneficial effects of selective intracoronary thrombolysis and subsequent improvement of LVEF recently have been reported.[9-13] However, these changes also may reflect, in part, spontaneous variation in patients with acute infarction. Our data emphasize that such clinical studies must be performed in a sufficiently large number of patients and involve contrast of treatment results to data in an appropriately matched control group of untreated patients.[12]

In the majority of the patients in the present study,

TABLE 2A. *Serial Left Ventricular Ejection Fraction, Rate-Pressure Product and Killip Classification in the 11 Patients Who Had Improvement of Ejection Fraction Compared with Study 1*

Pt		Study 1	Study 2	Study 3	Study 4	Hours post-MI Study 1	Location MI
1	LVEF	33	40	47	58	11	Inferior
	RPP	80	74	79	82		
	K	III	II	II	II		
2	LVEF	36	42	30	46	12	Inferior
	RPP	77	121	108	86		
	K	I	I	II	I		
3	LVEF	33	48	34	43	6	Inferior
	RPP	69	74	74	56		
	K	I	I	I	II		
4	LVEF	37	69	53	67	7	Inferior
	RPP	95	103	98	71		
	K	II	II	II	I		
5	LVEF	14	29	19	35	4	Inferior
	RPP	163	107	139	106		
	K	II	I	II	I		
6	LVEF	46	56	55	58	5	Inferior
	RPP	79	72	67	70		
	K	I	I	I	I		
7	LVEF	36	45	46	48	5	Inferior
	RPP	96	80	81	67		
	K	II	I	I	I		
8	LVEF	47	47	56	51	4	Inferior
	RPP	82	82	90	50		
	K	I	I	I	I		
9	LVEF	61	71	72	74	5	Inferior
	RPP	93	84	83	83		
	K	I	I	I	I		
10	LVEF	42	43	43	52	4	Inferior
	RPP	48	56	61	50		
	K	I	I	I	I		
11	LVEF	21	20	27	43	6	Anterior
	RPP	131	121	100	105		
	K	II	II	III	III		

LVEF is given as percent.

Abbreviations: LVEF = left ventricular ejection fraction; K = Killip classification; MI = myocardial infarction; RPP = rate-pressure product.

TABLE 2B. *Serial Left Ventricular Ejection Fraction, Rate-Pressure Product and Killip Classification in the Eight Patients Who Had Deterioration of Ejection Fraction Compared with Study 1*

Pt		Study 1	Study 2	Study 3	Study 4	Hours post-MI Study 1	Location MI
12	LVEF	47	43	—	39	7	Inferior
	RPP	37	35	—	36		
	K	I	I	I	II		
13	LVEF	51	45	48	42	5	Inferior
	RPP	61	69	74	70		
	K	I	I	I	I		
14	LVEF	46	40	39	37	6	Inferior
	RPP	75	73	89	77		
	K	I	I	I	I		
15	LVEF	60	58	46	51	4	Inferior
	RPP	66	75	69	47		
	K	I	I	I	I		
16	LVEF	67	53	68	61	5	Inferior
	RPP	66	53	60	60		
	K	I	I	I	I		
17	LVEF	39	31	37	30	9	Anterior
	RPP	58	64	64	72		
	K	I	I	I	I		
18	LVEF	37	32	39	26	3	Anterior
	RPP	80	71	80	58		
	K	II	II	II	II		
19	LVEF	33	29	30	26	7	Anterior
	RPP	76	46	57	71		
	K	I	I	II	I		

LVEF is given as percent.

Abbreviations: LVEF = left ventricular ejection fraction; K = Killip classification; MI = myocardial infarction; RPP = rate-pressure product.

we could not explain the spontaneous variation of LVEF. In patient 5, hypertension was treated medically with hydralazine hydrochloride and a decrease in blood pressure was associated with an increase of LVEF. Patients 1 and 29, who were in Killip class III on admission, were treated with furosemide and morphine sulphate, but only one demonstrated an immediate improvement of LVEF. None of the other patients received major medical treatment that could explain changes in LVEF. No patient received sublingual nitroglycerin for recurrent chest pain. Theoretically, changes in LVEF can be caused by a number of mechanisms. Improvement of LVEF can be expected with a decrease in LV afterload or a decrease of myocardial oxygen need. The patients who demonstrated improvement of LVEF spontaneously also demonstrated a significant decrease of mean rate-pressure product, mainly due to a decrease of mean blood pressure, over the course of study. However, there were individual patients in whom no change was apparent, despite improvement in LV function. When individual patients were analyzed, no correlation existed between either direction or magnitude of change in heart rate, mean blood pressure, rate-pressure product and change of LVEF.

Changes in LV preload also may affect LV pump function; such changes would have gone undetected in the present study because LV volumes were not assessed. However, patients with improved LV function received treatment no different from that of other patients, particularly with respect to morphine sulphate, a drug with potential for reducing preload.

Improved LV function also could be the result of resolving ischemia. Salvagable myocardium is present in experimental models up to 6 hours after infarc-

TABLE 2C. *Serial Left Ventricular Ejection Fraction, Rate-Pressure Product and Killip Classification in the 15 Patients Who Did Not Demonstrate Significant Changes in Ejection Fraction Compared with Study 1*

Pt		Study 1	Study 2	Study 3	Study 4	Hours post-MI Study 1	Location MI
20	LVEF	44	44	45	42	4	Inferior
	RPP	95	92	106	94		
	K	I	I	I	I		
21	LVEF	49	46	51	*	8	Inferior
	RPP	95	95	92			
	K	I	II	I			
22	LVEF	44	45	45	48	12	Inferior
	RPP	88	85	80	61		
	K	I	I	I	I		
23	LVEF	39	39	41	42	6	Inferior
	RPP	64	84	87	80		
	K	I	I	I	I		
24	LVEF	47	45	44	47	8	Inferior
	RPP	75	64	63	75		
	K	I	II	I	I		
25	LVEF	16	17	16	*	8	Inferior
	RPP	58	58	59			
	K	I	I	I			
26	LVEF	56	52	—	55	4	Inferior
	RPP	119	100	—	100		
	K	I	I	I	I		
27	LVEF	72	68	74	67	5	Inferior
	RPP	48	54	44	50		
	K	I	I	I	I		
28	LVEF	64	63	—	67	5	Inferior
	RPP	58	55	52	62		
	K	I	I	I	I		
29	LVEF	29	32	34	34	6	Inferior
	RPP	107	89	74	76		
	K	III	II	II	I		
30	LVEF	69	—	59	59	5	Inferior
	RPP	80	—	90	69		
	K	I	I	I	I		
31	LVEF	39	39	34	35	4	Anterior
	RPP	65	60	68	65		
	K	I	II	I	I		
32	LVEF	35	36	39	35	4	Anterior
	RPP	97	88	88	79		
	K	II	II	II	I		
33	LVEF	41	37	37	45	4	Anterior
	RPP	64	56	64	74		
	K	I	I	I	I		
34	LVEF	39	40	39	41	5	Anterior
	RPP	63	60	60	74		
	K	I	I	I	I		

LVEF is given as percent.

*Patient death.

Abbreviations: LVEF = left ventricular ejection fraction; K = Killip classification; MI = myocardial infarction; RPP = rate-pressure product.

tion.[21, 22] Myocardial perfusion defects demonstrated with thallium-201 during the acute phase of infarction may decrease in size during the subacute phase, possibly by reperfusion of initially ischemic myocardium.[23, 24] DeWood et al.[25] recently described a decreasing incidence of complete coronary occlusion with time in patients studied angiographically during the acute phase of myocardial infarction. Spontaneous thrombolysis or relief of superimposed coronary spasm[26] may explain these angiographic observations. These data may explain in part the changes in LV function demonstrated in our study. In addition, the development of collaterals may play a role in improved performance. The deterioration of LV function in individual patients is consistent with clinicopathologic data showing the progressive nature of acute myocardial infarction.[27] However, none of the patients in the present study had clinical evidence of persisting ischemia or infarct extension.[28] Recently, Hutchins and Bulkley[29] and Eaton et al.[30] pointed out that worsening of LV function can be reflected in two pathologic patterns: infarct extension by development of new areas of necrosis or infarct expansion by acute dilatation of the infarct without new necrosis. Our study focused on serial assessment of global LVEF. Complete analysis of regional wall motion would require serial multiple view studies.[31] This was performed in only 17 of 34 patients, too few for comprehensive analysis. Deterioration of regional wall motion, which may represent infarct expansion, was noted in only two patients; whereas regional wall motion improved in eight patients and remained unchanged in seven.[32]

Pantridge et al.[33] and Mueller et al.[34] observed transiently increased sympathetic stimulation, reflected in tachycardia and/or hypertension, in one-third of their patients early after their infarct. This may be not only secondary to pain and anxiety, but may occur as a consequence of neurogenic reflexes arising from the infarcted and adjacent myocardium. Such sympathetic stimulation also could result in an improved performance during the earliest measurements, predominantly through improved function in noninfarcted zones. Over time, as sympathetic stimulation decreases, LV function also may decrease, resulting in an apparent deterioration in performance.

Changes in LV function in patients with acute myocardial infarction have been reported.[35–46] Most studies have compared hemodynamic values obtained during the first days of the infarct to those obtained at later follow-up. These studies generally have indicated that improvement in cardiac performance may occur gradually in the majority of the surviving patients. In contrast, recent studies involving serial radionuclide assessment of LVEF during the subacute phase of infarction in stable patients have shown only minimal variation of LVEF.[47–49] Thus far, no study has focused on LV function during the first 24 hours of infarction, which is the time frame within which most acute therapeutic interventions should be used. Although in the present study significant changes in LVEF occurred in individual patients, consistent with previous studies,[47–49] mean LVEF for the overall group did not change (table 1).

Our results also may have relevance with regard to the use of LVEF as suggested by Shah et al.[15, 50] These investigators reported that a LVEF of 30% or less obtained during the first 24 hours of acute infarction was of prognostic value in predicting a high risk of hospital morbidity and mortality from pump failure. In the present study, four patients (nos 5, 11, 25 and 29) had LVEFs lower than 30% at the time of study 1. In three of these patients, LVEF had improved at the time of study 4 to greater than 30%. Therefore, for this specific application, it may be better to measure LVEF after the first 24 hours of infarction, when major changes are less likely to occur.

In conclusion, a single assessment of LVEF during the first 24 hours of acute myocardial infarction may not characterize LV performance properly in an individual patient. Dynamic and apparently unpredictable changes in LV function during the early hours of infarction hinder the validity of evaluating the effects of therapeutic interventions by comparing posttherapy data to a single baseline value.

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