Systolic Time Intervals and Left Ventricular Function in Acute Myocardial Infarction

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SUMMARY

Systolic time intervals (STI) were measured simultaneously with left ventricular pressure, right atrial pressure, and cardiac output in patients with acute myocardial infarction (AMI) within 24 hours of the onset of symptoms. Electromechanical systole (QS2) and left ventricular ejection time (LVET) were measured and preejection period (PEP) calculated and corrected for heart rate. LVET showed significant correlation with stroke volume ($r = 0.62$), confirming previous observations. Patients with significantly elevated left ventricular end-diastolic pressure (LVEDP) and clinical signs of congestive heart failure (CHF) usually exhibited a prolonged PEP when compared with normal subjects or patients with AMI who did not have signs of CHF. The normal PEP in the latter group might be due to adrenergic stimulation offsetting the effect of myocardial damage, although measurements of circulating catecholamines did not conclusively support this hypothesis. We conclude that STI are not reliable indices of left ventricular performance in AMI.

Additional Indexing Words:

Stroke volume Left ventricular end-diastolic pressure Congestive heart failure Catecholamines

THE measurement of systolic time intervals (STI) for the indirect assessment of cardiac function was used by Katz and Feil[1] and has recently been reintroduced by Weissler et al.[2-4] Characteristic changes of these measurements in patients with acute myocardial infarction (AMI) have been described by Diamant and Killip,[5] Toutouzas et al.,[6] and Bennett et al.[7] Observed changes have been correlated with clinical manifestations of the disease but not with direct measurements of left ventricular pressure. We have recently reported on the changes in left ventricular pressure measurements in patients with acute myocardial infarction[8] obtained by bedside catheterization[9] during the first 24 hours after the onset of symptoms. STI were obtained in some of these patients. The purpose of this report is to explore the relationship between systolic time intervals and direct hemodynamic measurements, including left ventricular pressures, obtained simultaneously.

Methods

The subjects were 24 patients with acute myocardial infarction confirmed by clinical, electrocardiographic, and enzymatic criteria. Studies were performed at the bedside within 24 hours of the initial symptoms. All studies were performed with the patient in supine position. None of the patients received digitalis or vasopressors, but all patients received either

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Supported in part by U. S. Public Health Service Research Grant HE 09785 from the National Heart and Lung Institute and a grant from the American Heart Association.

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Received August 2, 1971; revision accepted for publication October 13, 1971.

sedatives or analgesics, usually morphine, at some time prior to the study. On the basis of the clinical findings, the patients were subdivided into two groups as follows: Group 1 had electrocardiographic and enzymatic evidence of acute myocardial infarction without any clinical or radiologic evidence of left ventricular failure. The criteria for the clinical diagnosis of left ventricular failure was the presence of a ventricular diastolic gallop, persistent rales over the base of the lungs, and radiologic evidence of increased vascular markings or pulmonary edema. Group 2 patients all had left ventricular failure by the above criteria.

External Measurements

Lead 2 of the electrocardiogram, the phonocardiogram, and the carotid pulse wave were simultaneously recorded at a speed of 100 mm/sec on a Century or Sanborn recorder. Heart sounds were detected with a piezoelectric crystal microphone and filtered above 150 and below 50 Hz. The carotid pulse was recorded with a special pressure gauge[10] which was held under constant pressure of application with an adjustable clamp around the patient's neck. Measurements were taken over five to ten sequential heart beats and the results averaged. Two time intervals were measured: (1) electromechanical systole as the interval between the initial deflection of the Q wave to the second sound (QS2) and (2) left ventricular ejection time as the interval between the initial upstroke of the carotid pulse and incisura (LVET). Preejection period (PEP) was determined by subtracting LVET from QS2. Regression equations for systolic time intervals with respect to heart rate have been obtained in this laboratory from a group of 42 healthy individuals ranging in age from 25 to 75 years.

The regression equations for these normal subjects were as follows:

$$QS2 = -0.0020 \times \text{heart rate} + 0.522$$
$$LVET = -0.0016 \times \text{heart rate} + 0.394$$
$$PEP = -0.0004 \times \text{heart rate} + 0.126$$

The indices QS2I, LVETI, and PEPI were calculated by substituting the observed values for the constant in the regression equations. Percentage change was determined by the ratio between the observed and the predicted STI.

Hemodynamic Studies

The left heart was catheterized by a modified Seldinger technic,[9] while the right atrium was entered via an antecubital or femoral vein using a polyethylene catheter. Cardiac output was measured by the dye-dilution method. The methods employed in this study are described in detail elsewhere.[8] Blood samples for catecholamines were drawn from an antecubital vein and determined by a double-label isotope method.[11]

Results

Systolic time intervals (STI) are listed in table 1. In the group 1 patients who had no clinical evidence of left ventricular failure, the changes in PEP, LVET, and QS2 were variable. Although the mean values were reduced in comparison with normal subjects, the changes were not significant. The ratio PEP/LVET was unchanged from the expected normal value. In the patients with clinical evidence of left ventricular failure (group 2), PEP, PEPI, and PEP/LVET were significantly prolonged as compared with normal and group 1 subjects; QS2 was in the normal range, and LVET was shortened, although the latter reduction did not reach the level of statistical significance.

The relevant clinical and hemodynamic data as well as the catecholamine levels are presented in table 2. The mean age of the patients was significantly higher in group 2. LVEDP was significantly higher and SV significantly lower in group 2. The correlation between LVET and SVI ($r = 0.62$) is shown in figure 1. No significant correlation was found between STI and left ventricular end-diastolic pressure. Total catecholamines were more than five times the levels previously observed in normal subjects[11] in two patients out of 10 in group 1 and five out of nine in group 2. The mean changes in STI and in hemodynamic measurements between group 1 and 2 are shown in figures 2 and 3, respectively.

Discussion

Systolic time intervals have been correlated with hemodynamic measurement during positional changes[12] and pharmacologic manipulations.[13] Under the conditions of these acutely induced hemodynamic changes, STI correlated with the direct hemodynamic measurements. In congestive heart failure PEPI showed a highly significant correlation with clinical signs of failure, and LVET correlated with measured stroke volume.[14] Recently, the

Table 1

Systolic Time Intervals, Indices, and Percentage Changes from Expected Values

Patients	PEP (sec)	QS2 (sec)	LVET (sec)	Heart rate (beats/min)	Indices				Changes (%)		
					PEPI	QS2I	LVETI	PEP/LVET	PEP	QS2	LVET
Group 1*											
1	0.090	0.390	0.300	62	0.115	0.514	0.399	0.300	−11	− 2	+ 2
2	0.088	0.324	0.236	81	0.120	0.486	0.366	0.373	− 6	−10	−11
3	0.070	0.330	0.260	75	0.100	0.480	0.380	0.269	−27	−11	− 5
4	0.085	0.395	0.310	67	0.112	0.529	0.417	0.274	−14	+ 2	+ 8
5	0.100	0.347	0.247	94	0.138	0.535	0.397	0.405	+14	+ 4	+ 1
6	0.091	0.332	0.241	74	0.121	0.480	0.359	0.377	− 5	−11	−15
7	0.097	0.371	0.274	63	0.122	0.497	0.375	0.354	− 9	− 6	− 6
8	0.093	0.404	0.311	58	0.116	0.520	0.404	0.299	−10	0	+ 3
9	0.119	0.364	0.245	93	0.156	0.550	0.394	0.486	+34	+ 8	− 1
10	0.087	0.322	0.235	75	0.117	0.472	0.355	0.370	− 9	−13	−14
11	0.077	0.293	0.216	113	0.122	0.519	0.397	0.356	− 5	− 1	+ 1
Mean	0.091	0.352	0.261	77.73	0.122	0.507	0.386	0.351	− 1.77	− 3.64	− 3.36
SD	0.013	0.036	0.033	16.58	0.010	0.024	0.017	0.063	15.96	7.00	7.47
Group 2†											
12	0.091	0.286	0.195	121	0.139	0.528	0.389	0.467	+17	+ 2	− 2
13	0.108	0.385	0.277	64	0.134	0.513	0.379	0.390	+ 8	− 2	− 5
14	0.098	0.322	0.224	105	0.140	0.532	0.392	0.437	+17	+ 3	− 1
15	0.131	0.358	0.227	85	0.165	0.528	0.363	0.577	+42	+ 2	−12
16	0.098	0.304	0.206	109	0.142	0.522	0.380	0.476	+20	0	− 6
17	0.105	0.272	0.161	118	0.152	0.508	0.356	0.623	+33	−5	−19
18	0.099	0.307	0.208	114	0.143	0.529	0.386	0.476	+24	+ 4	− 2
19	0.087	0.326	0.239	83	0.120	0.492	0.372	0.364	− 6	− 9	− 9
20	0.100	0.395	0.295	58	0.123	0.511	0.388	0.339	− 3	− 3	− 2
21	0.122	0.415	0.293	75	0.156	0.565	0.413	0.416	+27	+12	+ 7
22	0.067	0.329	0.252	83	0.100	0.495	0.385	0.266	−28	− 8	− 3
23	0.120	0.358	0.238	88	0.155	0.534	0.379	0.504	+32	+ 3	− 6
Mean	0.102	0.338	0.235	91.92	0.139	0.521	0.382	0.445	+15.80	− 0.8	− 5.00
SD	0.017	0.045	0.040	21.14	0.017	0.017	0.010	0.099	21.61	5.79	6.42
t‡	1.8217	0.8234	1.7370	1.7792	2.9361	1.6750	0.6595	2.6567	2.2460	1.3305	0.5646
P§	<0.05	NS	NS	<0.05	<0.01	NS	NS	<0.01	<0.02	NS	NS
Groups 1 and 2											
Mean	0.097	0.345	0.247	85.13	0.131	0.515	0.384	0.400	+ 5.870	− 1.783	− 4.217
SD	0.016	0.040	0.039	20.02	0.017	0.022	0.014	0.095	20.233	6.508	6.835

*Uncomplicated myocardial infarction.
†Myocardial infarction with congestive heart failure.
‡t-test between groups 1 and 2.
§One-tailed test.

Table 2

Age, Location of Infarction, Hemodynamic Data, and Circulating Catecholamines

Patients	Age (yr)	Location of MI	LVEDP (mm Hg)	RAP (mm Hg)	dp/dt (mm Hg/sec)	CI (liter/min/m²)	SVI (ml/beat/m²)	LVSWI (gm-m/m²)	Catecholamines (μg/liter)
Group 1									
1	38	P	15.0	4	1,360	—	—	—	0.40
2	56	A	18.5	8	2,608	2.54	28.25	40.5	2.61
3	49	A	22.5	16	2,176	—	—	—	0.51
4	45	P	13.0	4	2,070	—	—	—	0.31
5	51	P	10.0	4	1,960	—	—	—	0.45
6	33	P	17.0	12	2,496	3.45	39.15	54.7	3.08
7	39	A	7.5	4	2,556	3.60	52.92	67.8	0.44
8	54	P	16.5	10	1,530	2.70	48.15	37.5	0.44
9	47	A	18.5	7	2,258	2.85	30.00	47.5	—
10	62	P	14.0	8	2,375	3.17	45.00	15.9	0.61
11	47	P	9.0	3	1,697	2.41	24.00	23.3	0.22
Mean	47.36		14.68	7.3	2,099	2.96	38.20	41.0	0.907
SD	8.48		4.56	4.1	422	0.46	11.40	17.8	1.033
Group 2									
12	44	P	35.0	11	—	3.38	26.40	24.8	—
13	48	P	32.0	7	1,590	—	—	—	—
14	53	A	38.5	9	1,965	2.91	27.93	23.0	1.30
15	60	A	20.0	11	1,597	3.06	31.83	39.8	1.01
16	49	A	34.0	11	1,480	2.72	23.45	15.9	0.25
17	73	P	29.0	12	2,258	2.85	30.00	47.5	2.96
18	73	P	19.0	12	2,480	2.59	22.76	25.1	2.20
19	75	A	23.0	16	1,520	1.52	17.70	26.2	—
20	53	P	34.0	12	1,445	3.81	48.80	57.1	0.42
21	51	A	24.0	5	1,788	2.25	28.90	36.2	3.26
22	77	A	25.0	6.5	3,046	2.39	31.80	21.6	0.16
23	50	P	10.0	5	1,458	3.04	34.95	47.5	0.22
Mean	58.8		26.96	9.8	1,875	2.78	29.50	33.2	1.309
SD	12.2		8.28	3.3	520	0.60	8.00	13.3	1.216
t	2.5951		4.3443	1.6213	1.1072	0.6931	1.9413	1.0775	0.779
P	<0.01		<0.001	NS	NS	NS	<0.05	NS	NS
Groups 1 and 2									
Mean	53.35		21.087	8.587	1,987	2.847	32.888	36.217	1.097
SD	11.89		9.114	3.857	476	0.545	10.006	15.195	1.111

Abbreviations: P = posterior and inferior; A = anterior; LVEDP = left ventricular end-diastolic pressure; RAP = right atrial pressure; CI = cardiac index; SVI = stroke volume index; LVSWI = left ventricular stroke-work index.

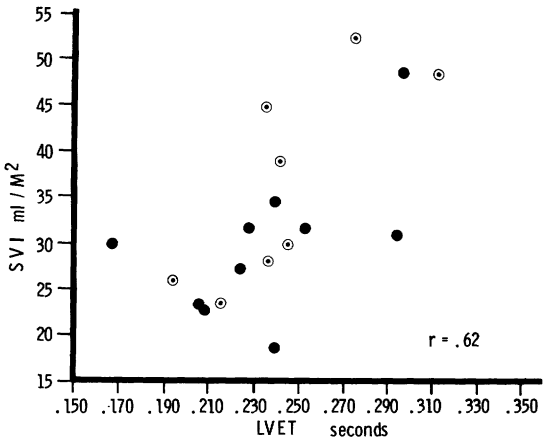

Figure 1

Chart showing left ventricular ejection time (LVET) on the abscissa and stroke volume index (SVI) on the ordinate. The open circles represent group 1 and the black circles group 2 patients.

PEP/LVET ratio has been suggested as a useful index of overall cardiac performance.[4]

In previous studies in patients with acute myocardial infarction, PEP was frequently found to be within normal limits, whereas QS2 and LVET were generally shortened.[5–7] The present investigation showed the same trends for the group as a whole. However, when the data are analyzed by subdividing the patients into subgroups by clinical criteria of presence or absence of left ventricular failure, significant differences in STI between these two classes of patients become apparent. PEP tended to be shortened in group 1 and significantly longer than normal in group 2. QS2 was somewhat shorter in group 1, whereas LVET was decreased in both groups, particularly in group 2 in comparison with normal subjects.

A variety of factors probably influence STI in the early stages of acute myocardial infarction. Stroke volume often is reduced in AMI,[8] and the correlation shown here between LVET and SVI ($r = 0.62$) suggests that LVET might be a useful noninvasive method for assessment of SVI, even in a situation where diverse factors are affecting both parameters.

It has been shown that PEP incorporates the isovolumic contraction time (ICT) of the ventricle,[14] the duration of which should be related to myocardial contractility. However, in AMI myocardial contractility is affected by two factors which are opposite in action: (1) sympathetic stimulation with a release of catecholamines, which tend to increase contractility and thereby decrease the duration of PEP, and (2) the effect of intrinsic muscle fiber damage, which decreases contractility

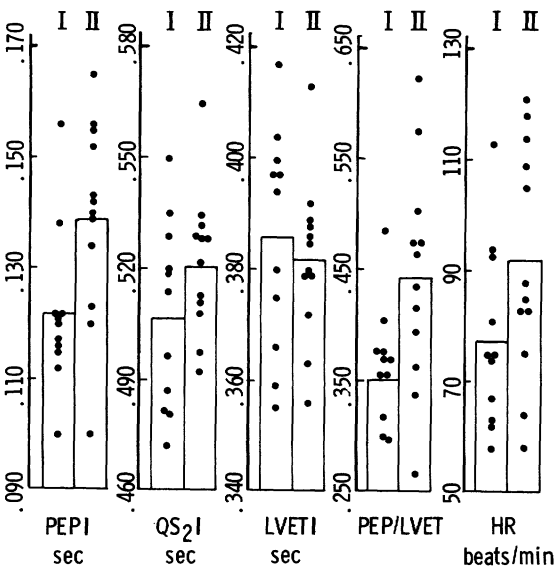

Figure 2

Bar graph comparing changes in mean systolic time intervals between group 1 and group 2 subjects. Individual values are indicated by dots.

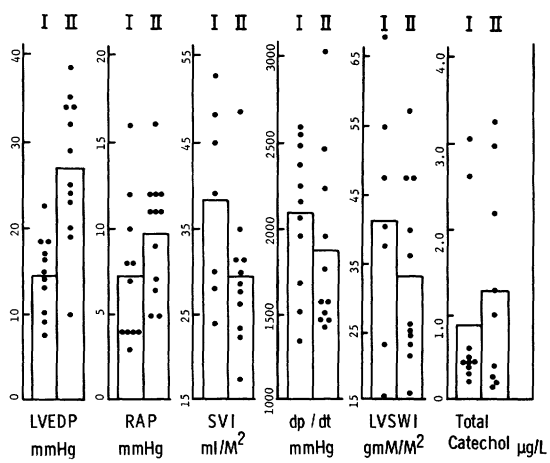

Figure 3

Bar graph comparing changes in mean hemodynamic measurements between group 1 and group 2 subjects.

and thus increases the duration of PEP. The moderate decrease in PEPI in group 1 patients who may have had less muscle fiber damage could be ascribed to predominance of the effects of increased sympathetic activity and circulating catecholamines. Group 2 patients, on the other hand, probably had more extensive muscle fiber damage, which resulted in cardiac failure despite increased catecholamine activity. The consequent prolongation of ICT would be expressed as an increase in PEP.

The present data show trends which support the above hypothesis. However, individual patients exhibit wide departure from the expected. For example, patients 5 and 9 had marked increase in PEP with only moderately increased LVEDP and no clinical signs of CHF. Circulating catecholamine levels did not correlate with the STI, although these values may not necessarily reflect the level of adrenergic activity of the myocardium.

The STI which showed the best agreement with the clinical findings was the PEP index. However, even the PEPI showed some overlap between the two groups. PEPI showed a more significant difference between group 1 and 2 than did PEP alone, probably because of the additive effect of the significant changes in both PEP and heart rate. We did not confirm that the PEP/LVET ratio was superior to PEPI.[4] Whereas STI in group 1 patients were similar to those seen in normal subjects under increased sympathetic stimulation, group 2 showed the characteristic pattern described for CHF. These data suggest that the STI are not sensitive indices for the diagnosis of infarction, but may aid in assessing myocardial competence once the diagnosis has been established by other means.

LVET may be useful as a crude estimate of stroke volume in acute myocardial infarction, although it appeared less reliable in the present series of patients with AMI than in patients with congestive heart failure due to other causes.[14] It should also be emphasized that indirect recording of a well-defined carotid pulse in a severely ill patient may involve considerable technical difficulties, and for that reason many patients had to be excluded from this study.

Once the diagnosis of AMI is established, the STI did not prove to be superior to physical examination in indicatng the presence of CHF in severe AMI. Although the STI exhibited a characteristic pattern for a group of patients, they had little predictive value in the individual case. The low correlation between STI and direct hemodynamic measurements appears to make application of this method in the diagnosis and evaluation of the patient with AMI of limited value.

Acknowledgment

The authors wish to thank Dr. B. Portnoy for his assistance in catecholamine determinations.

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Circulation. 1972;45:375-381
doi: 10.1161/01.CIR.45.2.375
Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231

Print ISSN: 0009-7322. Online ISSN: 1524-4539