Serial Observations on Left Ventricular Dysfunction in Acute Myocardial Infarction

II. Systolic Time Intervals in Power Failure

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SUMMARY

Systolic time intervals corrected for heart rate were studied serially in 50 patients with acute myocardial infarction (AMI) 1, 5, and 20 days after the onset of symptoms from simultaneously recorded kineto-, phono-, and electrocardiograms and carotid artery pulse tracings. Left ventricular ejection time (LVET), electromechanical systole (QA₂), and mechanical systole (M₁A₂) decreased from the first to the fifth day (P < 0.001) and increased again toward normal by the twentieth day. There was a clear correlation with the clinically assessed severity of AMI; the high prevalence of abnormal values indicated a frequent and early impairment of left ventricular performance. In severe power failure, deterioration was indicated by progressive shortening of the systolic time intervals, as contrasted to gradual improvement noted in uncomplicated infarctions. Similarly, LVET, QA₂, and M₁A₂ displayed a significant correlation with the grade of radiologically assessed pulmonary vascular congestion on the first day. A relationship between LVET, QA₂, and isovolumic contraction time (ICT)/LVET and the degree of paradoxical cardiac pulsation became apparent on the fifth day. LVET, M₁A₂, ICT/LVET, and preejection period (PEP)/LVET were more abnormal in the nine patients who died than in the 41 survivors (P < 0.05). PEP and ICT showed no correlation with clinically or radiologically assessed severity or prognosis of infarction, in contrast to findings in chronic heart failure. Decrease of contractility in AMI appears not to be directly reflected in preejection intervals. These findings indicate the value of externally measured left ventricular systolic time intervals, especially LVET, in assessment of the severity and prognosis of AMI.

Additional Indexing Words:
Carotid pulse Heart failure Kinetocardiography Phonocardiography

The potential value of the systolic time intervals in the assessment of left ventricular function in cardiac disease was elegantly demonstrated by Wiggers in 1921 and by Katz and Weil, and reemphasized by Blumberger. The relation of systolic time intervals to the hemodynamic events during ventricular contraction and ejection was later elucidated, so that their application to the noninvasive evaluation of left ventricular performance during myocardial infarction has become meaningful.

The mechanism of heart failure in acute myocardial infarction (AMI) differs from that in chronic congestive heart failure. Since

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studies reported on the systolic time intervals in the evaluation of AMI have been limited and the results seem to be somewhat conflicting,17–22 the present study was undertaken in 50 patients with AMI on the first, fifth, and twentieth day of their illness.

Patients and Methods

Fifty patients with AMI, consecutively admitted to the Intensive Care Unit of the University Central Hospital within 24 hr of the onset of symptoms, were included in the study. There were nine women and 41 men, ranging in age from 32 to 80 years, with a mean of 55.9 years. Transmural infarction by electrocardiographic criteria was present in 46 patients. The criteria applied in the diagnosis and in the evaluation of the clinical course have been described in detail in the first part of this study.16 Data on third and fourth heart sounds, arterial pressure, paradoxical cardiac pulsation, papillary muscle dysfunction, heart enlargement, and pulmonary vascular congestion discussed in the first part of this study were correlated with the systolic time intervals. The patients were divided according to clinical assessment of the severity of AMI into three groups as defined earlier:16 group 1, 15 patients with uncomplicated infarction; group 2, 23 patients with moderately extensive infarction; and group 3, 12 patients with severe infarction. Digoxin was administered to 39 patients because of definite signs of left ventricular failure, and catecholamines to four patients.

Examinations

Kinetocardiograms, electrocardiograms, phonocardiograms, and carotid artery tracings were simultaneously recorded with an Elema-Schönander EMT 439A transducer and a 6-channel direct-writing Mingograph 81 recorder on the first, fifth, and twentieth days at a paper speed of 50 mm/sec, and the systolic time intervals were measured. Although Spodick et al.23 have shown that measurement of ejection time does not significantly improve over the range of paper speed of 25 to 200 mm/sec, a check was made from measurements of various systolic time intervals obtained at 100 mm/sec. No differences were found. However, since changes in systolic time intervals are small and subject to random variability, close attention was paid to technical details during recording and measurement of the data. Heart sound vibrations for obtaining the durations of the time intervals were always simultaneously recorded with high-pass filters with nominal frequencies of 35, 70, and 250 Hz. Other details of the equipment are given in the first part of the study.16 The same investigator analyzed all studies. The patients were studied mainly in the morning in the fasting state except for the first tracing which was taken as soon as possible after admission. The recordings were obtained in expiratory apnea while the patient was in the supine position. In order to reduce measurement error and to avoid the secondary changes of prolonged apnea, we measured and averaged five consecutive cycles to the nearest 5 msec. The time intervals were defined as follows (fig. 1):

Electromechanical systole (QA2): from the onset of the QRS complex to the first high-frequency vibration of the aortic valve closure sound (A2).

Mechanical systole (M1A2): from the onset of the first high-frequency vibrations of the first heart sound (M1) to A2.

Isovolumic contraction time (ICT): from the earliest visible ventricular systolic displacement (point Z) to point E of the kinetocardiogram.

Left ventricular ejection time (LVET): from point E to A2.

Isovolumic relaxation time (IRT): from A2 to the early diastolic dip in the kinetocardiogram initiating the rapid filling wave (point O).

Carotid left ventricular ejection time (LVET<sub>carotid</sub>): from the onset of the steep upstroke to the trough of the incisura of the carotid artery pulse tracing.

Preejection period (PEP): QA2 minus LVET<sub>carotid</sub>.

Because of the effect of heart rate on the systolic time intervals, individual values were

![Figure 1](https://circ.ahajournals.org/)

**Figure 1**

Precordial kinetocardiographic (KCG) and external carotid pulse (CP) recordings with simultaneous electrocardiographic (ECG) and phonocardiographic (PCG) references. The methods of measurement of the various systolic time intervals are indicated. Abbreviations: CU = carotid upstroke; CI = carotid incisura; others = see Methods.
corrected, for comparative purposes, to the standard heart rate (HR) of 70 cycles/min, by the following linear regression equations:

\[ QA_2 (\text{msec}) = 546 - 2.1 \times \text{HR} + 14 \pm 14 \text{ (sd)} \]

\[ M_1A_2 (\text{msec}) = 456 - 1.8 \times \text{HR} + 15 \pm 15 \]

\[ ICT (\text{msec}) = 65 - 0.17 \times \text{HR} + 12 \pm 12 \]

\[ LVET (\text{msec}) = 384 - 1.5 \times \text{HR} + 30 \pm 30 \]

\[ IRT (\text{msec}) = 203 - 0.85 \times \text{HR} + 29 \pm 29 \]

\[ PEP (\text{msec}) = 131 - 0.4 \times \text{HR} + 13 \pm 13 \]

\[ LVET_{carotid} (\text{msec}) = 413 - 1.7 \times \text{HR} + 10 \pm 10 \]

The equations for ICT, LVET, and IRT are based on our data on 139 healthy men 45 to 64 years of age, studied at rest and analyzed by kineto- and phonocardiography with criteria analogous to those employed in the present study. The equations for QA2, M1A2, PEP, and LVET_{carotid} are based on the data of Weissler et al. for 121 healthy males aged 19 to 65, and analyzed by phono- and electrocardiography and carotid artery tracing with criteria similar to those used in the present study. The ratios ICT/LVET and PEP/LVET_{carotid} were calculated from the rate-corrected values to avoid the effect of highly variable heart rate alone in AMI. The arterial pressure was measured by the usual auscultatory method.

Problems of Measurement

The Z point, indicating the initiation of the systolic ventricular movement following electrical activation, was easy to locate in the kinetocardiograms. Point Z occurred with narrow scatter about 40 msec after the onset of the QRS complex, and was thus in good agreement with the base-line suggestions of Davie et al. The method of El-Sherif et al. for determination of the Z point at the end of the a wave was found to give unreliable results. The recent data confirm that the initial systolic displacement in the apexcardiogram and in the KCG corresponds exactly with the first recordable pressure rise in the left ventricle recorded by catheter-tip micro-manometers and coincides also with the first visual evidence of left ventricular isovolumic contraction observed in high-speed cineradiography of ventricular radiopaque marker movement.

Definition of the E point was sometimes difficult because of several notches in the early isovolumic phase, although the E point of the kinetocardiogram is still a more accurate signal of the start of ventricular ejection than is the E crest recorded by the apexcardiogram. These notches occurred due to lack of the normal deep systolic retraction and were caused by ventricular asynchrony in the precordial displacement curves of the patients with AMI. This asynchrony was observed in all patients. Comparison of the tracings from different precordial points was helpful here, as was the relation of the E point to the first heart sound in simultaneous phonocardiograms. The E point never lagged behind the carotid upstroke, and, in this way, an unintended shortening of LVET and prolongation of ICT was avoided. The recordings were taken in the normal expiratory apnea. Thus the observed shortening of LVET and of other systolic time intervals related to stroke volume was not artificially induced by this methodologic influence which changes left ventricular filling. Care was taken to use the earliest point of ventricular depolarization for the measurements. We checked this point by comparing the onset of the QRS complex in the reference lead II to the onset of QRS in the other limb leads in simultaneous recordings and making any corrections required.

Results

Deviations from Normal in the Total Series

The individual values for the systolic time intervals in relation to heart rate on the first, fifth, and twentieth days, as measured from kineto-, phono-, and electrocardiograms, are shown in figure 2. The values for QA2 were outside the normal range in 80% of the patients, for M1A2 in 82%, for ICT in 80%, for LVET in 66%, for LVET_{carotid} in 85%, and for IRT in 56%.

The mean values of all parameters measured, the individual values corrected for the standard heart rate of 70 cycles/min by respective regression equations, and the percentage deviations of the means from normal values are shown in table 1. The means of QA2, M1A2, LVET, LVET_{carotid}, ICT, and IRT were all highly significantly different from normal on the first, fifth, and twentieth days (P < 0.001 for each). ICT was prolonged, while the others were shortened. The PEP was the only systolic time interval that remained within the normal range throughout the study.

The mean values of all the systolic time intervals regularly deviated most from the normal levels on the fifth day from onset of AMI (table 1). QA2, M1A2, LVET, and LVET_{carotid} (P < 0.001 for each), and IRT, ICT/LVET (P < 0.01), and PEP/LVET (P < 0.05) were more abnormal on the fifth day when compared with the initial values. Further changes were insignificant. In contrast, PEP and ICT did not show any significant sequential changes throughout the study.
LV DYSFUNCTION IN ACUTE MI

Relation to Clinical Severity and Sequential Changes
When the sequential changes of the systolic time interval were analyzed in each of the three groups of patients with AMI the following main differences among the groups were observed. The greatest serial changes were encountered in group 2, while the changes relative to day 1 were less in groups 1 and 3.

The systolic time intervals did not differ significantly between groups 1 and 2. In these groups the greatest sequential changes between the first and fifth days occurred in QA2 (P < 0.001 for both groups), M1A2, LVET, and LVETcarotid (P < 0.01 for each in group 1, P < 0.001 for each in group 2) (table 2, fig. 3). These variables tended to return toward normal values later, and this change was strongest in the patients with uncomplicated infarctions (group 1).

In the patients of group 3 with severe power failure QA2 was initially much shortened as compared to the other groups (P < 0.05), and its further decrease in the course of time remained insignificant. However, LVET decreased progressively. The expected decrease from the first to the fifth day (P < 0.05) showed the same trend from the fifth to the twentieth day. After the values of the groups with milder forms of AMI reached a minimum on the fifth day, they tended to return to normal (table 2, fig. 3). In group 3 only, ICT and PEP decreased by the twentieth day. The values of group 3 differed significantly (P < 0.05) from those of group 1 for QA2, M1A2, LVET, and LVETcarotid, and from those of group 2 for QA2 and M1A2 on the fifth or the twentieth day, or both.

Relation to Prognosis
The mean values of the nine patients who died in the hospital, all due to hemodynamic deterioration (severe pulmonary edema or profound shock), were compared with the mean values of the 41 survivors (table 3, fig. 3). Already on the first day M1A2, LVET, and LVETcarotid were less and ICT/LVET and PEP/LVET were greater (P < 0.05 for all) in the patients who later died than in the survivors. On the twentieth day, or in tracings just preceding death, QA2, LVET, and LVETcarotid remained significantly shorter in those who died than in the survivors (P < 0.05 for each), while in the other measurements no significant differences were found.

Relation to Physical and Radiologic Findings
The patients with pansystolic paradoxical cardiac pulsations had on the fifth day

![Figure 3](http://circ.ahajournals.org/)

*Figure 3*
Deviations from normal of the means of heart-rate corrected PEP, ICT, LVET, and the values of PEP/LVET ratios in the three clinical groups of AMI and in the surviving and dying patients. The peak changes generally occurred on the 5th day. Note the overlapping and relatively stable values of PEP and ICT. LVET changes were closely similar when measured by carotid pulse or KCG, except for slight deviations found in patients just before death. The improvement in LVET in the course of time does not occur in severe infarctions. Symbols: see figure 2.

*Figure 2*
The durations of the individual systolic time intervals on the 1st, 5th, and 20th days in the three clinical groups of patients with AMI. The normal mean regression lines in relation to heart rate and 1 sd limits are given. Symbols: o = uncomplicated infarctions, group 1; □ = moderate infarctions, group 2; ● = severe infarctions, group 3.

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### Table 1

**Serial Rate-Corrected** Systolic Time Intervals (in msec) in 50 Patients with Acute Myocardial Infarction on the First, Fifth, and Twentieth Days

<table>
<thead>
<tr>
<th>Time</th>
<th>Group</th>
<th>N</th>
<th>Heart rate (beats/min)</th>
<th>QA₂</th>
<th>MA₂</th>
<th>LVET</th>
<th>LVET&lt;sub&gt;arterial&lt;/sub&gt;</th>
<th>ICT</th>
<th>ICT/LVET</th>
<th>PEP</th>
<th>PEP/LVET&lt;sub&gt;arterial&lt;/sub&gt;</th>
<th>IRT</th>
</tr>
</thead>
<tbody>
<tr>
<td>1st day</td>
<td>All patients</td>
<td>50</td>
<td>80 ± 15</td>
<td>379 ± 19</td>
<td>316 ± 27</td>
<td>257 ± 28</td>
<td>272 ± 27</td>
<td>78 ± 18</td>
<td>0.30 ± 0.09</td>
<td>106 ± 24</td>
<td>0.39 ± 0.12</td>
<td>112 ± 30</td>
</tr>
<tr>
<td></td>
<td>% deviation†</td>
<td></td>
<td>-5%</td>
<td>-4%</td>
<td>-8%</td>
<td>-7%</td>
<td>+44%</td>
<td>+50%</td>
<td>+3%</td>
<td>+14%</td>
<td>-22%</td>
<td></td>
</tr>
<tr>
<td>5th day</td>
<td>All patients</td>
<td>50</td>
<td>82 ± 18</td>
<td>363 ± 18</td>
<td>290 ± 23</td>
<td>237 ± 27</td>
<td>255 ± 24</td>
<td>81 ± 17</td>
<td>0.34 ± 0.08</td>
<td>108 ± 21</td>
<td>0.43 ± 0.11</td>
<td>123 ± 28</td>
</tr>
<tr>
<td></td>
<td>% deviation</td>
<td></td>
<td>-9%</td>
<td>-9%</td>
<td>-15%</td>
<td>-13%</td>
<td>+50%</td>
<td>+70%</td>
<td>+5%</td>
<td>+23%</td>
<td>-14%</td>
<td></td>
</tr>
<tr>
<td>20th day</td>
<td>All patients</td>
<td>45</td>
<td>68 ± 12</td>
<td>388 ± 31</td>
<td>306 ± 30</td>
<td>245 ± 29</td>
<td>260 ± 26</td>
<td>81 ± 16</td>
<td>0.33 ± 0.08</td>
<td>108 ± 23</td>
<td>0.42 ± 0.11</td>
<td>117 ± 25</td>
</tr>
<tr>
<td></td>
<td>% deviation</td>
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<td>-7%</td>
<td>-12%</td>
<td>-12%</td>
<td>+50%</td>
<td>+65%</td>
<td>+5%</td>
<td>+20%</td>
<td>-18%</td>
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<tr>
<td>Normal</td>
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<td>399 ± 14</td>
<td>330 ± 15</td>
<td>278 ± 30</td>
<td>294 ± 10</td>
<td>54 ± 12</td>
<td>0.20 ± 0.06</td>
<td>103 ± 13</td>
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<tr>
<td></td>
<td>sd in % of the mean</td>
<td>±3.5%</td>
<td>±4.5%</td>
<td>±11%</td>
<td>±3.5%</td>
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<td>±30%</td>
<td>±13%</td>
<td>±11%</td>
<td>±20%</td>
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*Heart rate 70 cycles/min, mean ± sd.
†Percentage deviation of the means from normal values.

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**Discussion**

Experimental and clinical observations have indicated that ICT and PEP are inversely related to the severity of the hemodynamic deterioration of the patients with acute myocardial infarction. The prevalence of abnormal LVET and ICT values were similar to those reported previously. In the present study on 50 consecutive patients with transmural acute myocardial infarction, the prevalence of abnormal LVET was inversely related to the severity of the hemodynamic deterioration of the patients with acute myocardial infarction. In general, the prevalence of abnormal values of the various systolic time intervals was not prolonged in relation to the severity of the hemodynamic deterioration of the patients with acute myocardial infarction. The prevalence of abnormal values of the various systolic time intervals was not prolonged in relation to the severity of the hemodynamic deterioration of the patients with acute myocardial infarction.

Patient and control values of LVET and ICT were similar in patients with acute myocardial infarction and controls. The prevalence of abnormal LVET and ICT values were similar to those reported previously. In the present study on 50 consecutive patients with transmural acute myocardial infarction, the prevalence of abnormal LVET was inversely related to the severity of the hemodynamic deterioration of the patients with acute myocardial infarction.
Table 2

Serial Rate-Corrected* Systolic Time Intervals (in msec) in Three Clinical Subgroups of Patients with Acute Myocardial Infarction

<table>
<thead>
<tr>
<th>Time</th>
<th>Group</th>
<th>N</th>
<th>Heart rate (beats/min)</th>
<th>QA2</th>
<th>MA2</th>
<th>LVET</th>
<th>LVET_corrected</th>
<th>ICT</th>
<th>ICT/LVET</th>
<th>PEP</th>
<th>PEP/LVET_corrected</th>
<th>IRT</th>
</tr>
</thead>
<tbody>
<tr>
<td>1st day</td>
<td>Group 1</td>
<td>15</td>
<td>76 ± 11</td>
<td>383 ± 17</td>
<td>318 ± 21</td>
<td>264 ± 26</td>
<td>278 ± 29</td>
<td>79 ± 17</td>
<td>0.30 ± 0.09</td>
<td>105 ± 23</td>
<td>0.38 ± 0.11</td>
<td>125 ± 38</td>
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<td>-4%</td>
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<td>-5%</td>
<td>+46%</td>
<td>+50%</td>
<td>+2%</td>
<td>+9%</td>
<td>-13%</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Group 2</td>
<td>23</td>
<td>78 ± 17</td>
<td>379 ± 20</td>
<td>320 ± 26</td>
<td>250 ± 26</td>
<td>273 ± 24</td>
<td>75 ± 21</td>
<td>0.29 ± 0.10</td>
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</tr>
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<td></td>
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<td>+39%</td>
<td>+45%</td>
<td>+3%</td>
<td>+11%</td>
<td>-29%</td>
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<td>12</td>
<td>88 ± 16</td>
<td>366 ± 19</td>
<td>302 ± 31</td>
<td>245 ± 30</td>
<td>263 ± 28</td>
<td>80 ± 12</td>
<td>0.33 ± 0.07</td>
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<td>+75%</td>
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<td>259 ± 19</td>
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<td>+9%</td>
<td>+20%</td>
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<td>71 ± 12</td>
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<td>-22%</td>
<td>-19%</td>
<td>+31%</td>
<td>+65%</td>
<td>-4%</td>
<td>+20%</td>
<td>-27%</td>
<td></td>
</tr>
</tbody>
</table>

*Heart rate 70 cycles/min, mean ± sd.
†Percentage deviation of the means from normal values.
‡Includes data of two patients who died a few days before the twentieth day.
Table 3

Serial Rate-Corrected* Systolic Time Intervals (in msec) in Relation to Prognosis in Acute Myocardial Infarction

<table>
<thead>
<tr>
<th>Time</th>
<th>Prognosis</th>
<th>N</th>
<th>Heart rate (beats/min)</th>
<th>QA,</th>
<th>MA,</th>
<th>LVET</th>
<th>LVETcoroltd</th>
<th>ICT</th>
<th>ICT/LVET</th>
<th>PEP</th>
<th>PEP/LVETcoroltd</th>
<th>IRT</th>
</tr>
</thead>
<tbody>
<tr>
<td>1st day</td>
<td>Survivors</td>
<td>41</td>
<td>77 ± 14</td>
<td>380 ± 17</td>
<td>320 ± 23</td>
<td>262 ± 24</td>
<td>277 ± 25</td>
<td>76 ± 19</td>
<td>0.29 ± 0.10</td>
<td>103 ± 24</td>
<td>0.37 ± 0.12</td>
<td>111 ± 28</td>
</tr>
<tr>
<td>% deviation†</td>
<td></td>
<td></td>
<td>-5%</td>
<td>-3%</td>
<td>-6%</td>
<td>-6%</td>
<td>+41%</td>
<td>+45%</td>
<td>0%</td>
<td>+6%</td>
<td>-22%</td>
<td></td>
</tr>
<tr>
<td>Dying</td>
<td></td>
<td>9</td>
<td>91 ± 16</td>
<td>369 ± 19</td>
<td>293 ± 28</td>
<td>236 ± 25</td>
<td>250 ± 22</td>
<td>85 ± 9</td>
<td>0.36 ± 0.07</td>
<td>119 ± 23</td>
<td>0.48 ± 0.12</td>
<td>115 ± 42</td>
</tr>
<tr>
<td>% deviation</td>
<td></td>
<td></td>
<td>-8%</td>
<td>-11%</td>
<td>-15%</td>
<td>-15%</td>
<td>+57%</td>
<td>+80%</td>
<td>+16%</td>
<td>+37%</td>
<td>-20%</td>
<td></td>
</tr>
<tr>
<td>5th day</td>
<td>Survivors</td>
<td>41</td>
<td>78 ± 15</td>
<td>364 ± 16</td>
<td>300 ± 19</td>
<td>239 ± 25</td>
<td>259 ± 21</td>
<td>81 ± 17</td>
<td>0.34 ± 0.08</td>
<td>105 ± 21</td>
<td>0.41 ± 0.11</td>
<td>129 ± 26</td>
</tr>
<tr>
<td>% deviation</td>
<td></td>
<td></td>
<td>-9%</td>
<td>-9%</td>
<td>-14%</td>
<td>-12%</td>
<td>+50%</td>
<td>+70%</td>
<td>+2%</td>
<td>+17%</td>
<td>-10%</td>
<td></td>
</tr>
<tr>
<td>Dying</td>
<td></td>
<td>9</td>
<td>98 ± 19</td>
<td>355 ± 20</td>
<td>284 ± 25</td>
<td>224 ± 21</td>
<td>232 ± 23</td>
<td>79 ± 16</td>
<td>0.35 ± 0.10</td>
<td>123 ± 22</td>
<td>0.53 ± 0.12</td>
<td>118 ± 30</td>
</tr>
<tr>
<td>% deviation</td>
<td></td>
<td></td>
<td>-11%</td>
<td>-14%</td>
<td>-21%</td>
<td>-21%</td>
<td>+46%</td>
<td>+75%</td>
<td>+19%</td>
<td>+51%</td>
<td>-17%</td>
<td></td>
</tr>
<tr>
<td>20th day</td>
<td>Survivors</td>
<td>41</td>
<td>67 ± 10</td>
<td>373 ± 44</td>
<td>312 ± 24</td>
<td>251 ± 19</td>
<td>266 ± 21</td>
<td>81 ± 16</td>
<td>0.32 ± 0.07</td>
<td>106 ± 23</td>
<td>0.40 ± 0.11</td>
<td>119 ± 23</td>
</tr>
<tr>
<td>% deviation</td>
<td></td>
<td></td>
<td>-7%</td>
<td>-5%</td>
<td>-10%</td>
<td>-10%</td>
<td>+50%</td>
<td>+60%</td>
<td>+3%</td>
<td>+14%</td>
<td>-17%</td>
<td></td>
</tr>
<tr>
<td>Dying</td>
<td></td>
<td>7</td>
<td>78 ± 27</td>
<td>320 ± 31</td>
<td>255 ± 41</td>
<td>191 ± 34</td>
<td>227 ± 26</td>
<td>71 ± 15</td>
<td>0.37 ± 0.09</td>
<td>93 ± 24</td>
<td>0.41 ± 0.08</td>
<td>84 ± 49</td>
</tr>
<tr>
<td>% deviation</td>
<td></td>
<td></td>
<td>-20%</td>
<td>-23%</td>
<td>-46%</td>
<td>-23%</td>
<td>+31%</td>
<td>+85%</td>
<td>-9%</td>
<td>+17%</td>
<td>(N = 2)</td>
<td></td>
</tr>
</tbody>
</table>

*Heart rate 70 cycles/min, mean ± sd.
†Percentage deviation of the means from normal values.

Table 4

Rate-Corrected* Systolic Time Intervals (in msec) in Relation to Pulmonary Vascular Congestion on the First Day of Acute Myocardial Infarction

<table>
<thead>
<tr>
<th>Grade of pulmonary vascular congestion</th>
<th>N</th>
<th>Heart rate (beats/min)</th>
<th>QA,</th>
<th>MA,</th>
<th>LVET</th>
<th>LVETcoroltd</th>
<th>ICT</th>
<th>ICT/LVET</th>
<th>PEP</th>
<th>PEP/LVETcoroltd</th>
<th>IRT</th>
</tr>
</thead>
<tbody>
<tr>
<td>Grade 0</td>
<td>4</td>
<td>78 ± 15</td>
<td>370 ± 19</td>
<td>307 ± 16</td>
<td>256 ± 14</td>
<td>286 ± 30</td>
<td>76 ± 13</td>
<td>0.29 ± 0.09</td>
<td>84 ± 20</td>
<td>0.29 ± 0.10</td>
<td>108 ± 19</td>
</tr>
<tr>
<td>% deviation†</td>
<td></td>
<td>-7%</td>
<td>-7%</td>
<td>-8%</td>
<td>-3%</td>
<td>+41%</td>
<td>+45%</td>
<td>-10%</td>
<td>-17%</td>
<td>-24%</td>
<td></td>
</tr>
<tr>
<td>Grade 1</td>
<td>24</td>
<td>77 ± 14</td>
<td>382 ± 16</td>
<td>322 ± 24</td>
<td>264 ± 26</td>
<td>274 ± 25</td>
<td>76 ± 24</td>
<td>0.29 ± 0.09</td>
<td>108 ± 22</td>
<td>0.40 ± 0.12</td>
<td>108 ± 37</td>
</tr>
<tr>
<td>% deviation</td>
<td></td>
<td>-4%</td>
<td>-2%</td>
<td>-5%</td>
<td>-7%</td>
<td>+41%</td>
<td>+45%</td>
<td>+5%</td>
<td>+14%</td>
<td>-24%</td>
<td></td>
</tr>
<tr>
<td>Grade 2</td>
<td>10</td>
<td>83 ± 20</td>
<td>387 ± 24</td>
<td>319 ± 30</td>
<td>257 ± 32</td>
<td>278 ± 23</td>
<td>80 ± 31</td>
<td>0.31 ± 0.09</td>
<td>108 ± 29</td>
<td>0.40 ± 0.14</td>
<td>121 ± 23</td>
</tr>
<tr>
<td>% deviation</td>
<td></td>
<td>-3%</td>
<td>-3%</td>
<td>-8%</td>
<td>-5%</td>
<td>+48%</td>
<td>+55%</td>
<td>+5%</td>
<td>+14%</td>
<td>-15%</td>
<td></td>
</tr>
<tr>
<td>Grade 3</td>
<td>7</td>
<td>86 ± 16</td>
<td>355 ± 19</td>
<td>288 ± 26</td>
<td>231 ± 24</td>
<td>246 ± 21</td>
<td>80 ± 28</td>
<td>0.34 ± 0.10</td>
<td>110 ± 24</td>
<td>0.45 ± 0.12</td>
<td>115 ± 16</td>
</tr>
<tr>
<td>% deviation</td>
<td></td>
<td>-11%</td>
<td>-13%</td>
<td>-17%</td>
<td>-16%</td>
<td>+48%</td>
<td>+70%</td>
<td>+7%</td>
<td>+29%</td>
<td>-20%</td>
<td></td>
</tr>
</tbody>
</table>

*Heart rate 70 cycles/min, mean ± sd.
†Percentage deviation of the means from normal values.
the leveling trend in the groups with milder forms. These results are in contrast to the progressive prolongation of preejection intervals reported by others\textsuperscript{14, 15} in patients with advancing degrees of chronic congestive heart failure.

In experimental myocardial ischemia early deterioration of ventricular contractility has been found to occur within seconds after coronary occlusion.\textsuperscript{32, 33} However, major adjustments of preload and afterload, which regularly occur in AMI, may disturb the above relationship profoundly. In severe circulatory impairment such as was found in the patients of group 3 with AMI a decrease in stroke volume could be expected to prolong ICT and PEP\textsuperscript{12} along with the reduced contractility. However, direct inotropic actions of a high catecholamine level described in these patients\textsuperscript{34, 35} and of digitalis,\textsuperscript{5, 36} and a decrease in afterload by lowered aortic diastolic pressure or an increase in preload by markedly elevated left ventricular end-diastolic pressure would counteract these changes on PEP and ICT. It therefore seems likely that the usual positive correlation of isovolumic contraction indices, such as PEP and ICT, to chronic heart failure is reversed when power failure of acute myocardial infarction occurs. Decrease in myocardial contractile performance under these circumstances will not directly be reflected in the various preejection time intervals.

The observed slight prolongation of kinetocardiographically determined ICT in the presence of unaltered PEP (as calculated by both carotid pulse and KCG methods) may be due to methodologic difficulties in the measurement, as the noninvasive methods of determining ICT are as yet rather inaccurate. Also lack of knowledge of dynamic behavior of the electromechanical lag in AMI may be a factor. This discrepancy in changes between ICT and PEP amounted to no more than 6 to 26 msec. Externally measured PEP will reflect true PEP more accurately than externally measured ICT reflects true ICT.\textsuperscript{8, 27, 37} In our study the prolongation of ICT occurred mainly in the earliest part of ICT, i.e., in the interval from Z point to M\textsubscript{1} (one-half to two-thirds of the total prolongation), although this subdivision of ICT into its components remains somewhat questionable because of the difficulty in determining the M\textsubscript{1} component of the first heart sound.\textsuperscript{8, 28} This would particularly apply to AMI where contractility changes are common. Although the Z point in the apexcardiogram and KCG reflects the start of true isovolumic contraction accurately and the E point in the KCG marks the start of ejection, small errors may occur in the determination of these points in the precordial displacement curves particularly when large a waves are present. Also the Z point in ventricular dilatation and asynergy in AMI may be recorded earlier than in the normal subject, in whom the heart is somewhat distant from the chest wall and contracts in synergy. Despite all these possible sources of error the basic information found in the present study is not altered, and neither ICT nor PEP proved to be useful for grading either the hemodynamic severity or the prognosis in AMI.

The lack of change in PEP differs from two reports of prolonged PEP in AMI\textsuperscript{17, 20} but confirms the earlier findings of Halpern et al.\textsuperscript{21} and Nagy.\textsuperscript{22} In AMI the normal value of PEP would, in fact, suggest impaired contractility when viewed in the hemodynamic setting discussed above.

**Left Ventricular Ejection Time, Electromechanical Systole, and Mechanical Systole**

Experimental and clinical evidence have been presented to show that LVET, when rate-corrected, is primarily and closely related to stroke volume, while the effects of myocardial contractility and aortic impedance also play a role.\textsuperscript{4, 5, 9, 12} Therefore, the finding of a decreased LVET in the present study, with parallel changes in QA\textsubscript{2} and M\textsubscript{1}A\textsubscript{2}, suggests that stroke volume is frequently reduced in the early phase of AMI. This is also known to occur from direct estimations of cardiac output in AMI.\textsuperscript{38} Moreover, the time of maximum decrease of LVET on the fifth day

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observed in the present study agrees well with time of maximum reduction of stroke volume in AMI in serial studies reported by Nager et al.\textsuperscript{39} The hemodynamic disorder persists in milder degree even 3 weeks after infarction, as indicated by abnormal systolic time intervals.

A formula for prediction of stroke volume from LVET and heart rate has been developed by Harley et al.\textsuperscript{12} based on an accurate study of beat-to-beat relationships \( (r = 0.95) \). When their formula \( SV = 0.501 \text{LVET} + 0.130 \text{HR} - 67.2 \) was applied to the present data, stroke volumes were obtained which fell in the expected range. For example, the calculated mean stroke volume was 22 ml on the twentieth day in the moribund patients, while on the same day in the survivors it was 69 ml. The corresponding cardiac outputs were 1.7 and 4.6 liters. The smallest forward stroke volume of 12 ml occurred on the day of death in the patient with papillary muscle rupture. As such figures were not checked by actual output determinations they may only be attractive postulates. Especially in severe AMI, considerable increases in aortic impedance may occur which would prolong LVET\textsuperscript{11} while simultaneously endogenous or exogenous catecholamines and digitalis will shorten LVET,\textsuperscript{4, 5, 36, 40} with unknown net result. Therefore, estimations of stroke volume from LVET are subject to errors in AMI, the magnitudes of which are so far undetermined.

Of the systolic time intervals we studied it was clear, however, that the degree of shortening of LVET most often correlates inversely with the clinically assessed severity of AMI, the degree of radiologic pulmonary vascular congestion, the degree of ventricular asynergy, and prognosis. The association of shortened LVET with signs of cardiac pump failure supports the idea that, in AMI, LVET largely reflects stroke volume. Shubin et al. have demonstrated that stroke volume is the hemodynamic parameter most closely related to severity of cardiogenic shock.\textsuperscript{41} Remarkably, LVET shortened progressively in the patients dying from pump failure without showing the usual tendency of later improvement in sequential determinations. Thus, LVET would appear to be a very convenient parameter for the serial monitoring of left ventricular pump function in coronary care units.

Shortening of QA\textsubscript{2} in AMI is in marked contrast to its normal duration in chronic heart failure,\textsuperscript{14, 15} where LVET still decreases. It has been suggested that catecholamines\textsuperscript{42} along with the reduced stroke volume\textsuperscript{12} are responsible for this feature of heart failure in AMI.

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Serial Observations on Left Ventricular Dysfunction in Acute Myocardial Infarction: II. Systolic Time Intervals in Power Failure
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