Isometric Contraction Period of the Left Ventricle in Acute Myocardial Infarction

By Kiyoshi Inoue, M.D., George M. Young, M.D., Archibald L. Grierson, M.D., Harold Smulyan, M.D., and Robert H. Eich, M.D.

SUMMARY
The usefulness of nontraumatic methods for measuring the left ventricular isometric contraction period (ICP) in myocardial infarction (MI) has been evaluated. The ICP was measured in 13 normal men and 38 patients admitted to a coronary care unit with chest pain, including 18 with acute MI, 12 with old MI, and nine with chest pain of miscellaneous origin; one of these nine was also included in the group with acute myocardial infarction. ICP was determined by three different methods: (1) time between the onset of systolic wave (A) of apexcardiogram (ACG) and the beginning of upstroke (C) of carotid pulse tracing (CPT); (2) time between A and ejection crest (E) of the ACG; and (3) time between the initial low frequency, low amplitude vibration of the first heart sound, and the C of CPT. Statistically significant differences of ICP between the normal group and the three groups of patients were demonstrated only by method 1. Likewise, acute coronary ligation in seven dogs produced characteristic changes in ICP by method 1, which were associated with a reduction in the left ventricular (LV) stroke volume, LV dp/dt, and the aortic flow velocity. The ICP obtained by method 1 appears to be of value in the bedside evaluation of acute myocardial infarction.

Additional Indexing Words: Abnormal apexcardiogram Early systolic bulge Ill-defined ejection crest First heart sound

Attempts to quantitate circulatory function in acute myocardial infarction have been limited because of the transient nature of the hemodynamic changes and the fear of undertaking elaborate diagnostic procedures in patients with major illnesses. However, recent advances in technics for the recording of low frequency displacement curves from the chest wall have made available a safe and simple method to assess more accurately cardiac function.1-7

Since Libman and Sacks8 described an aneurysmal bulge occurring in association with myocardial infarction, Vakil,9 Harrison and Hughes,10 and Eddelman and his associates,11,12 using the kinetocardiogram, have emphasized the frequent appearance of abnormal precordial pulsations in patients with coronary artery disease. Recently, Lane and co-workers13 have published studies, on patients with coronary artery disease, of various types of systolic bulges shown by the apexcardiogram and compared these findings with myocardial asynergy as detected by cineventriculography.
In addition to the study of precordial movements, it has been appreciated that the duration of cardiac systolic and diastolic time intervals using noninvasive techniques is also of value in studying cardiac function.\textsuperscript{14, 15} Recently, Weissler and co-workers,\textsuperscript{16} using the phonocardiogram and electrocardiogram, suggested their use for measuring pre-ejection period to predict diminished cardiac output in patients with heart failure.

This study was designed to evaluate the usefulness of these cardiographic techniques for measuring the isometric contraction period of the left ventricle (ICP) in patients with acute or old myocardial infarction. ICP was measured by three different cardiographic methods. Each was evaluated, and the best was used to aid in assessing the severity of the cardiac dysfunction in patients.

**Methods**

The true isometric contraction period begins with the onset of muscle tension and ends with the onset of ejection. As shown in figure 1, this interval was estimated by three different cardiographic methods, using the phonocardiogram (PCG), apexcardiogram (ACG), and external carotid pulse tracing (CPT). The first, the A-C interval, is the time between the onset of the initial systolic wave (A point) of the ACG and the onset of the upstroke (C point) of the CPT. The latter was corrected by subtracting the lag in central pulse transmission time from the aortic root to the right common carotid artery. This pulse delay (PD) was measured as the interval between the onset of the high frequency vibrations of the aortic component (A\textsubscript{3}) of the second heart sound and the dicrotic notch (DN) of the CPT. The second method, the A-E interval, was the time between A and E points of the ACG. The E point was taken as the initial reduction in upstroke velocity of the primary systolic wave. The third, the I-C interval, was the period from the onset of the initial low frequency, low amplitude vibrations (I point) of the first heart sound and the C point of CPT minus PD. The left ventricular ejection time (ET) was also measured using the CPT, as the interval between the C point and the dicrotic notch. This was corrected for the heart rate by dividing by the square root of the duration of each cardiac cycle and expressed as the corrected left ventricular ejection time (CET). The evaluation of ICP was performed in three different groups. The first was in experimental myocardial infarction in dogs, the second in normal subjects, and the third in patients with coronary artery disease.

**Dog Experiments**

Seven adult mongrel dogs, weighing from 18 to 29 kg were studied while under sodium pentobarbital (30 mg/kg) anesthesia. Respiration was maintained by a Harvard respirator. The chest was opened, and the heart was suspended in a pericardial cradle. Pressures in the central aorta and the left ventricle were measured using Statham strain gauges (P23Db) and large bore cannulae inserted through both common carotid arteries. An electromagnetic flowmeter (Statham model 4001), placed around the root of the aorta, sensed aortic flow velocity. This signal was measured simultaneously with phonocardiogram (PCG) from third left interspace along sternal border (3L) at low (L), medium (M), and high (H) frequency ranges, carotid pulse tracing (CPT), and electrocardiogram (ECG). ICP measured by the intervals between A and E, A and C, and I and C points. Dn = dicrotic notch.

![Figure 1](https://example.com/figure1.png)
integrated to give the stroke volume of the left ventricle. Left ventricular contractility was estimated by measuring the peak of the aortic flow velocity and the maximum rate of left ventricular pressure rise (LV dp/dt) using an RC circuit. A Sanborn model 21050-A pulse transducer microphone was placed directly on the surface of the left ventricular apical wall to obtain the ACG. An Electronics for Medicine model PS-1B sound transducer microphone was also placed on the wall of the left ventricular outflow area to sense the heart sounds for the PCG. Each microphone was connected to an Electronics for Medicine model TPD multifilter system phonocardiograph. Heart sounds at low and high frequency ranges, ACG, aortic stroke volume (AFV), left ventricular stroke volume (LVSV), LV dp/dt, and electrocardiogram (ECG) were recorded simultaneously using an Electronics for Medicine model DR-8 multichannel oscilloscopic photographic recorder at a paper speed of 200 mm/sec.

After the control tracing, a branch of the left anterior descending coronary artery was ligated, and repeat recordings were obtained at 30-min intervals for the 120 min following ligation. ICP was measured using the three methods described. In the dog studies, the C point was taken as the uncorrected onset of the upstroke of the central aortic pulse pressure curve. After each experiment, the heart was removed, and the gross specimen of the heart was examined to confirm the presence of infarcted myocardium.

**Human Study**

**Normal Subjects**

Thirteen normal men, aged 23 to 45 years, were studied. None had evidence of clinical heart disease. ECG, ACG, PCG, and CPT were simultaneously recorded at a paper speed of 100 mm/sec during relaxed mid-expiratory apnea. The PCG was obtained from one or two auscultatory areas by using simultaneous low and high-frequency ranges. The microphones (Electronics for Medicine model PS-1B) placed at the third intercostal space just to the left of the sternum or at the left ventricular apical region, or in both, were connected to the multifilter system phonocardiograph. To bring the heart closer to the chest wall, the ACG was recorded with patients in the left lateral decubitus position. The ACG pulse transducer microphone (Sanborn model 21050-A) was held in place at the point of maximum impulse by a rubber strap. The external carotid pulse tracing was obtained by an Electronics for Medicine model PS-1A pulse transducer microphone, held manually over the right common carotid artery.

**Patients with Coronary Artery Disease**

A total of 38 patients admitted to a coronary care unit with acute chest pain were studied. The ages varied from 39 to 86 years, and there were 27 men and 11 women. All recordings were obtained within 72 hours of patient admission to the unit. Patients were classified into three groups: group I, 18 with acute myocardial infarction proved by diagnostic changes in ECG and serum enzyme studies; group II, 12 with history and ECG evidence of old myocardial infarction but no laboratory evidence suggesting acute involvement; and group III, nine with acute chest pain, who were proved not to have myocardial infarction; four of these had coronary insufficiency; three had small pulmonary emboli; one had an anxiety attack; and one had chest pain of undetermined origin. One patient of group III subsequently had an acute myocardial infarct and is also included in group I. The cardiographic recordings and the measurements of ICP were performed by the same technics as those used in normal subjects.

**Results**

**ICP in Experimental Myocardial Infarction in Dogs**

Frequently, occlusion of the coronary artery produced transient ventricular arrhythmias, but these usually subsided spontaneously within 10 min. One dog had a persistent ventricular tachycardia, which was successfully terminated by DC countershock. The parameters under study usually stabilized in 20 min following the occlusion, and control measurements were begun at this time.

The changes in ICP by the three different methods and the hemodynamic results are summarized in table I. Thirty minutes following coronary occlusion, a reduction in LV dp/dt of 27%, LVSV of 19%, and peak aortic flow velocity (AFV) of 32% was observed, which was associated with a slight prolongation of ICP (A-C and A-E). One hundred twenty minutes after coronary ligation LV dp/dt, LVSV, and AFV had fallen 45, 61, and 59%, respectively, associated with a 6 mm Hg rise in the left ventricular end-diastolic pressure (LVEDP). At this time, ICP was prolonged by 31 msec (A-C interval), 22 msec (A-E interval), and 6 msec (1-C interval). The changes in ICP by A-C and A-E intervals were roughly inversely proportional to the
changes in LVSV, AFV, and LV dp/dt, while those of I-C intervals were not related.

Figure 2 shows changes in the configuration of ACG as well as its temporal relationship to the electrical, acoustic, and hemodynamic events. As seen in the control tracing, the onset of both A and I points appeared in advance of the initial rise in intraventricular cavity pressure. The A point occurred 17 msec before the actual rise in intraventricular pressure, and I point was 10 msec in advance. The initial rapid and steep ascent of the ACG prior to ejection, turned into gradual slope at the E point which coincided with the onset of upstroke of the aortic pressure. The initial low frequency, low amplitude vibrations of the first heart sound developed into the high amplitude main vibration, which coincided with the peak of LV dp/dt curve, the E point of the ACG, and the onset of ejection. The ICP measurements in this control tracing, obtained by three different methods, were similar: 68 msec (A-C), 68 msec (A-E), and 65 msec (I-C). Sixty minutes following

Table 1

<table>
<thead>
<tr>
<th></th>
<th>Control values (mean ± ss)</th>
<th>Time (min) following coronary artery ligation (values: mean ± ss)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>30</td>
</tr>
<tr>
<td>HR</td>
<td>171.0 ± 6.0 (beats/min)</td>
<td>184 ± 7.2</td>
</tr>
<tr>
<td>MAP</td>
<td>79.0 ± 5.6 (mmHg)</td>
<td>76 ± 5.2</td>
</tr>
<tr>
<td>LVEDP</td>
<td>5.8 ± 0.8 (mmHg)</td>
<td>6.8 ± 0.6</td>
</tr>
<tr>
<td>LV dp/dt</td>
<td>100 (%)</td>
<td>73 ± 2.0</td>
</tr>
<tr>
<td>LVSV</td>
<td>100 (%)</td>
<td>81 ± 2.2</td>
</tr>
<tr>
<td>AFV</td>
<td>100 (%)</td>
<td>68 ± 3.1</td>
</tr>
<tr>
<td>A-C</td>
<td>69 ± 3.2 (msec)</td>
<td>73 ± 3.1</td>
</tr>
<tr>
<td>A-E</td>
<td>68 ± 4.2 (msec)</td>
<td>78 ± 5.5</td>
</tr>
<tr>
<td>I-C</td>
<td>64 ± 3.8 (msec)</td>
<td>55 ± 4.7</td>
</tr>
<tr>
<td>CET</td>
<td>7.1 ± 0.6 (msec)</td>
<td>7.7 ± 0.6</td>
</tr>
</tbody>
</table>

Abbreviations: HR = heart rate; MAP = mean arterial pressure; LVEDP = left ventricular end-diastolic pressure; LV dp/dt = maximum rate of left ventricular pressure rise; LVSV = left ventricular stroke volume; AFV = peak aortic flow velocity; CET = corrected left ventricular ejection time; A-C, A-E, and I-C = systolic time intervals, see text also.

Simultaneous electrocardiogram (ECG), phonocardiogram (PCG) with low (L) and high (H) frequency ranges, apexcardiogram (ACG), aortic (AO) and left ventricular (LV) pressures, and LV dp/dt in an open-chest dog. PCG and ACG were obtained directly from the left ventricular wall. The changes in ICP measured by A-C, A-E, and I-C intervals were shown at 60 and 120 min following coronary occlusion.

Circulation, Volume XLII, July 1970
ligation, the temporal relationship among the ACG, PCG, and pressures had greatly changed. The initial low frequency, low amplitude vibration of the first heart sound had disappeared, and the onset of the heart sound appeared 39 msec after the onset of rise in intraventricular pressure. This resulted in a greatly shortened I-C interval (38 msec). The E point of the ACG had become ill-defined, and E seemed to be located 3 msec before the onset of ejection (A-E, 87 msec). The A-C interval was 90 msec, which was definitely prolonged compared to the control value, and associated with a 43% reduction in LV dp/dt. After 120 min of occlusion, a large akinetic zone developed in the left ventricular apical myocardium and bulged out. The E point became more vague, which resulted in further difficulty in the measurement of the A-E interval. ICP, as measured by A-C method was 103 msec, by A-E, 88 msec, and by I-C, 68 msec. ICP by measuring A-C became definitely prolonged and was associated with a 45% reduction in LV dp/dt. The prominent a wave in the ACG and an exaggerated atrial sound were also associated with a rise in LVEDP. In all tracings, the A point preceded the onset of rise in intraventricular cavity pressure, and prolongation of the A-C interval paralleled reduction in LV dp/dt, AFV, and LVSV.

The ICP in Normal Subjects

The A and I points of the cardiographic tracing in seven subjects, and E and C points in five subjects occurred together. Three of the total 13 subjects had no difference between the A and the I, and the E and the C points, and had an identical ICP by the three methods of measurements. In the majority of the cases, however, there were small differences between these points. Figure 1 is an example in which A preceded I by 5 msec, and C appeared 19 msec in advance of the E point. For all 13 cases, A appeared 24 msec ± 2.4 (SE) after the onset of QRS in ECG, and preceded I point by 7 msec ± 1.2 (SE). The uncorrected C point preceded E by 15.4 msec ± 6.8 (SE). The results of measurements of ICP by these methods and CET are summarized in table 2. The mean value of the A-C interval, which was corrected by PD, was 50.2 msec ± 2.4 (SE), which was shorter than that of the A-E interval by 13.6 msec, and longer than that of I-C interval by 11.6 msec, which was also corrected by the PD. PD was 33.2 msec ± 4.8 (SE) in this group.

ICP in Patients with Coronary Artery Disease

All values for the total of 39 cases are presented in table 3.

Acute Myocardial Infarction (18 Cases)

There were six cases of anterior wall myocardial infarction (cases 1 to 6), five of inferior wall infarction (cases 7 to 11), four of lateral wall infarction (cases 12 to 15), one case of posterior wall damage (case 16), and two cases of combined wall damage (cases 17 and 18). Four patients died, and three had postmortem examinations. Eight patients had an A-C interval of more than 100 msec, including four who died and two with combined wall damage. The patient with the longest A-C interval had left bundle-branch block.

The systolic configuration of the ACG was altered in every case. Prominent a waves, as judged by the ratio to the total ACG...
Table 3

Apex cardiographic, Phonocardiographic, and Hemodynamic Measurements in 39 Patients with Chest Pain

<table>
<thead>
<tr>
<th>Case</th>
<th>Patient’s initials</th>
<th>Age (yr) &amp; sex</th>
<th>a (%)</th>
<th>ICP (msec)</th>
<th>ET (msec)</th>
<th>CET (msec)</th>
<th>HR (/min)</th>
<th>BP (mm Hg)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>A-C</td>
<td>A-E</td>
<td>I-C</td>
<td>ET</td>
<td>CET</td>
</tr>
<tr>
<td>1.</td>
<td>L.M.*</td>
<td>68 M</td>
<td>30</td>
<td>105</td>
<td>110</td>
<td>80</td>
<td>240</td>
<td>8.4</td>
</tr>
<tr>
<td>2.</td>
<td>B.L.</td>
<td>61 F</td>
<td>16</td>
<td>90</td>
<td>120</td>
<td>70</td>
<td>250</td>
<td>9.2</td>
</tr>
<tr>
<td>3.</td>
<td>W.D.</td>
<td>72 M</td>
<td>21</td>
<td>95</td>
<td>125</td>
<td>55</td>
<td>270</td>
<td>9.1</td>
</tr>
<tr>
<td>4.</td>
<td>C.J.†</td>
<td>45 M</td>
<td>38</td>
<td>120</td>
<td>90</td>
<td>50</td>
<td>110</td>
<td>6.3</td>
</tr>
<tr>
<td>5.</td>
<td>S.W.</td>
<td>62 M</td>
<td>26</td>
<td>80</td>
<td>100</td>
<td>45</td>
<td>160</td>
<td>6.8</td>
</tr>
<tr>
<td>6.</td>
<td>M.S.</td>
<td>84 F</td>
<td>10</td>
<td>90</td>
<td>140</td>
<td>70</td>
<td>250</td>
<td>9.6</td>
</tr>
<tr>
<td>7.</td>
<td>B.W.</td>
<td>64 M</td>
<td>20</td>
<td>85</td>
<td>100</td>
<td>65</td>
<td>260</td>
<td>9.8</td>
</tr>
<tr>
<td>8.</td>
<td>A.V.</td>
<td>48 F</td>
<td>23</td>
<td>80</td>
<td>55</td>
<td>55</td>
<td>220</td>
<td>8.3</td>
</tr>
<tr>
<td>9.</td>
<td>T.L.</td>
<td>52 F</td>
<td>-</td>
<td>90</td>
<td>120</td>
<td>50</td>
<td>200</td>
<td>8.6</td>
</tr>
<tr>
<td>10.</td>
<td>H.D.</td>
<td>50 M</td>
<td>14</td>
<td>130</td>
<td>130</td>
<td>55</td>
<td>220</td>
<td>8.7</td>
</tr>
<tr>
<td>11.</td>
<td>S.R.</td>
<td>43 M</td>
<td>9</td>
<td>80</td>
<td>90</td>
<td>65</td>
<td>200</td>
<td>7.7</td>
</tr>
<tr>
<td>12.</td>
<td>H.J.</td>
<td>70 M</td>
<td>30</td>
<td>90</td>
<td>120</td>
<td>65</td>
<td>210</td>
<td>8.1</td>
</tr>
<tr>
<td>13.</td>
<td>F.M.</td>
<td>56 M</td>
<td>9</td>
<td>90</td>
<td>85</td>
<td>45</td>
<td>220</td>
<td>9.5</td>
</tr>
<tr>
<td>14.</td>
<td>W.H.†</td>
<td>46 M</td>
<td>10</td>
<td>100</td>
<td>100</td>
<td>50</td>
<td>240</td>
<td>9.2</td>
</tr>
<tr>
<td>15.</td>
<td>L.M.†</td>
<td>81 M</td>
<td>26</td>
<td>115</td>
<td>60</td>
<td>50</td>
<td>160</td>
<td>6.2</td>
</tr>
<tr>
<td>16.</td>
<td>L.R.</td>
<td>86 F</td>
<td>15</td>
<td>105</td>
<td>140</td>
<td>35</td>
<td>240</td>
<td>8.1</td>
</tr>
<tr>
<td>17.</td>
<td>P.A.</td>
<td>68 F</td>
<td>18</td>
<td>115</td>
<td>120</td>
<td>40</td>
<td>260</td>
<td>9.0</td>
</tr>
<tr>
<td>18.</td>
<td>R.D.</td>
<td>79 F</td>
<td>10</td>
<td>110</td>
<td>90</td>
<td>60</td>
<td>230</td>
<td>8.9</td>
</tr>
</tbody>
</table>

Group I (acute myocardial infarction; 18 cases)

Group II (old myocardial infarction; 12 cases)

Group III (no myocardial infarction; 9 cases)

* Died
† Postmortem examination.
‡ Same patient as case 1; first admission.

Abbreviations: a (%) = ratio of amplitude of a wave of apex cardiogram to total complex; ICP = isometric contraction period; ET = left ventricular systolic ejection time; other abbreviations, same as previous tables.

complex,18,17 were observed in 12 cases. All patients demonstrated a systolic bulge in early, middle, or late systole. These abnormal ACG tracings frequently had an ill-defined E point, making the measurements of the A-E interval unreliable. As seen in figure 3, the initial peak of the systolic wave could be taken as the E point, and the subsequent wave as a.
systolic bulge (B). This E point, however, preceded C by approximately 35 msec, which is the reverse of the ACG-E and the CPT-C points in the normal group. Presumably, this initial peak is another bulge, which appeared in the pre-ejection phase. In the majority of these cases, the intensity of the first heart sound was also attenuated, and the initial low frequency components were absent, resulting in an ill-defined I point. Figure 4 is an example.

Old Myocardial Infarction (12 Cases)

In 10 patients (cases 19 to 28), including three (cases 19, 23, and 24) with radiologic evidence of left ventricular aneurysm, systolic bulges had appeared in early, middle, or late systole. Figure 5 is an example of one patient with radiologic evidence of a left ventricular apical aneurysm and a sustained systolic bulge contiguous with an ill-defined E point.

The vague E point was observed in these 10 cases with apparently abnormal systolic configurations. This also resulted in the unreliability of the technic for measuring the ICP by the A-E method.

No Myocardial Infarction (Nine Cases)

Four cases (cases 31 to 34) had transient ST-T changes in the ECG, but had normal serial cardiac enzyme studies. Another three cases (cases 35 to 37) were suspected of having small pulmonary emboli, and in the remaining two cases (cases 38 and 39), the genesis of the transient chest pain was unclear. None had an abnormal bulge, except a prominent late systolic shoulder (SS), which was also seen in some of the normal subjects. An example of this is shown in figure 6. This patient had chest pain of uncertain origin with ECG evidence of left ventricular hypertrophy. Another patient (case 37) first entered the
hospital with chest pain, felt to be due to a small pulmonary embolism from other clinical data. At that time he had almost normal values of ICP by the three different methods. Ten days later, acute myocardial infarction developed, and this episode was associated with rapid prolongation of ICP (case 1). This is the only case which is used in two different groups.

Comparison of ICP Between Normals and Three Groups of Patients

The ranges as well as the mean values of ICP obtained in patients with coronary artery disease by the three different methods are summarized in table 4. The variations of ICP by these different methods and in these different groups of patients were not related to the level of the arterial diastolic pressure nor to the length of the cardiac cycle. In comparing the results of table 4 with those of table 2, all ICP measurements by the three methods in myocardial infarction (groups I and II) were statistically significantly prolonged from those of normals with a P value of <0.001. The difference between the normal group and group III was not statistically significant. As seen in table 4, the A-C interval is the only ICP measurement which statistically separates group I from each of the other two groups. ICPs measured by the A-E and I-C intervals in acute and old myocardial infarction groups were longer than those of nonmyocardial infarction (group III). However, these methods did not separate the acute and old myocardial infarction groups. The corrected ejection time was slightly shortened in group I and slightly prolonged in group II, but these differences were not statistically significant from those observed in the normal group and group III.
ISOMETRIC CONTRACTION PERIOD

**Figure 5**
Old myocardial infarction (case 23, group II in table 3) with radiologic evidence of apical aneurysm. Note prolongation of A-E and normal A-C and I-C intervals, and the sustained systolic bulge contiguous with E point. Symbols same as in preceding figures.

**Table 4**
Comparison of ICP Among Three Groups of Patients With Chest Pain

<table>
<thead>
<tr>
<th>Group</th>
<th>A-C (msec)</th>
<th>A-E (msec)</th>
<th>I-C (msec)</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Range</td>
<td>Mean</td>
<td>SD</td>
</tr>
<tr>
<td></td>
<td>80-130</td>
<td>98.5</td>
<td>14.1</td>
</tr>
<tr>
<td>(18 cases)</td>
<td>55-140</td>
<td>101.0</td>
<td>27.4</td>
</tr>
<tr>
<td></td>
<td>35-80</td>
<td>55.8</td>
<td>11.0</td>
</tr>
<tr>
<td>II</td>
<td>Range</td>
<td>Mean</td>
<td>SD</td>
</tr>
<tr>
<td></td>
<td>55-90</td>
<td>72.5</td>
<td>7.3</td>
</tr>
<tr>
<td>(12 cases)</td>
<td>80-125</td>
<td>97.9</td>
<td>16.1</td>
</tr>
<tr>
<td></td>
<td>30-75</td>
<td>52.3</td>
<td>10.3</td>
</tr>
<tr>
<td>III</td>
<td>Range</td>
<td>Mean</td>
<td>SD</td>
</tr>
<tr>
<td></td>
<td>45-80</td>
<td>59.2</td>
<td>12.1</td>
</tr>
<tr>
<td>(9 cases)</td>
<td>65-95</td>
<td>77.8</td>
<td>7.2</td>
</tr>
<tr>
<td></td>
<td>25-55</td>
<td>41.1</td>
<td>12.8</td>
</tr>
</tbody>
</table>

Abbreviations: Same as in previous tables.

**Discussion**
The ICP as measured by the cardiographic method offers a nontraumatic means for estimating the velocity of isometric contraction of the myocardium and thus indirectly evaluating contractility. The clinical application of these indirect measurements of the ICP has been studied in the evaluation of the coronary artery disease with variable results. Benchimol and Dimond and Lane and associates, using A-E interval
of the ACG, and Davie and associates\textsuperscript{21} and Agress and Wegner,\textsuperscript{22} using the kinetocardiogram, have observed prolongation of the ICP in coronary artery disease. Using these methods, Benchimol and Dimond\textsuperscript{23} and Harrison and co-workers,\textsuperscript{24} however, have also reported a prolongation of the ICP in normal older subjects. Variability of these results has led to some concern about the clinical usefulness of nontraumatic methods for the measurement of ICP. To evaluate the variability of the ACG method, the accuracy of the A-E interval was compared in our study with two other polygraphic technics as an estimate of "true" ICP.

In our dog studies, the heart sounds and the ACG were obtained directly from the ventricular wall. In these preparations, the initial low frequency, low amplitude vibrations of the first heart sound (I point), established in the control tracing, tended to disappear following coronary ligation. This could be explained by reduction in the intensity of the first heart sound. This attenuation is in turn due to reduction in dp/dt\textsuperscript{25} and in acceleration and deceleration of blood flow,\textsuperscript{26} factors responsible for first heart sound production. The E point, ACG representation of the onset of left ventricular ejection, was clearly definable in the control tracings. This point, however, was often obscure following coronary ligation. As shown by Rushmer and associates\textsuperscript{27} and Gorlin and associates,\textsuperscript{28, 29} sudden ischemia of a portion of the myocardium creates a local akinesis, which subsequently causes an abnormality in force, speed, and sequence of ventricular contraction. This distortion of ventricular motion has also been shown to alter greatly the systolic wave form of the ACG.\textsuperscript{13, 30} Therefore, the abnormal ACG that follows coronary artery ligation may be produced, at least in part, by a different mechanism from that which occurs in the normal ventricle.\textsuperscript{31} It is even possible that the entire abnormal systolic wave of the ACG is due to one or more abnormal bulges. These bulges make accurate localization of the ACG-E point difficult and explain the difficulties in detecting a precise E point from the ACG.

Therefore, the onset and the terminal of ICP, correctly measured by I and E points in the control tracing, may not be valid when applied to experimental myocardial infarction in dogs. On the other hand, the location of the A point in the ACG could always be established in these dog studies despite the presence of experimental myocardial infarction. Furthermore, this A point occurred 17 msec before the onset of left ventricular pressure rise, 25 msec ± 1.9 (se) after the onset of the ECG QRS complex, and was the first detectable mechanical event of systole. Dieudonne\textsuperscript{32} has shown also that intramural pressure of the left ventricle increased 20 msec in advance of the initial rise in intracavitary pressure. Therefore, it seems reasonable to define the onset of mechanical systole by the A point of the ACG. Using only the A-C interval in the dog preparations, a good correlation between ICP and the hemodynamic changes in LV dp/dt, AFV, LSV, and LVEDP could be demonstrated.

These three different technics for measuring ICP were also applied to both normal subjects and patients with coronary artery disease. In patients, the end of ICP was determined indirectly by using the external carotid pulse tracing. The obvious delay between aortic valve opening and onset of upstroke of the CPT (C point) is then corrected by subtracting the PD interval. It has been shown that the interval between the actual aortic valve opening and the CPT-C point is less than that occurring between aortic valve closure and the dicrotic notch of the carotid pulse.\textsuperscript{3} While it may not be completely accurate to correct the C point by the PD, this correction is probably proportional to the actual C point delay. Therefore, the PD correction of the C point was used in this study in order that the ICP of normals might be compared with that of patients with coronary artery disease.

The ranges as well as the mean values of the normalized ICP have been reported by Spodick and Kumar,\textsuperscript{33} Oreskov,\textsuperscript{34} and Tafur and co-workers\textsuperscript{3} who used several different cardiographic technics. Our results in the normal group were similar, and A-C, I-C, and
A-E intervals in our studies were in close agreement with each other. In subjects with acute myocardial infarction, however, only the A-C interval provided a useful measurement of ICP, as both the I and E points became ill-defined in the majority of subjects, while the A point in ACG which occurred 24 msec ± 2.4 (se) after the onset of the QRS complex in normals, could always be established in patients with myocardial infarction. It is of interest that measurement of ICP using the A-C interval could statistically separate the groups with acute myocardial infarction from the other patients with coronary artery disease. In one patient, who developed an acute myocardial infarction during observation, a prolongation of ICP occurred. In addition, the greatest prolongation of ICP by measuring A-C intervals was mainly observed in those with severe disease, that is, combined wall damage and in those who died.

With nontraumatic methods, it must be emphasized that the evaluation of the mechanical events of the left ventricle is not always exact. This is due in part to difficulty in relating these intervals to precise cardiac events, during which the coupling with the thoracic wall is always undetermined but critical. Nonetheless, this study suggests that indirect measurement of ICP using the A-C interval has value in the clinical evaluation of patients with acute myocardial infarction. Its potential uses might include assessment of the time course of the severity of the cardiodynamic changes in this disease.

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Circulation. 1970;42:79-90
doi: 10.1161/01.CIR.42.1.79
Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
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