Clinical Evaluation of Left Ventricular Pressures in Myocardial Infarction

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SUMMARY
Left ventricular pressures were obtained in 14 patients with acute myocardial infarction, of whom seven survived. Calculated contractile element velocity (V_{CE}), left ventricular end-diastolic pressure, and maximum dp/dt were compared with other hemodynamic measurements of LV function. V_{CE} at 5 mm Hg (V_{CE5}) was calculated according to the Maxwell three-component model of muscle and used as an index of overall contractile state. There was considerable overlap between survivors and nonsurvivors in the levels of V_{CE5}, LVEDP, cardiac index, heart rate, and systemic vascular resistance. There was a reasonably good separation between survivors and nonsurvivors with measurements of mean arterial pressure, maximum LV dp/dt, mean isovolumic Δp/Δt, and stroke work. Changes in V_{CE5} were no more sensitive after five inotropic interventions than ventricular function curves in monitoring changes in contractile state. It is concluded that LV pressure measurements in acute myocardial infarction offer little further clinical information of importance beyond that which can be obtained from measurements of pulmonary capillary wedge pressure, arterial pressure, and cardiac output.

Additional Indexing Words:
Force-velocity relations
Ventricular function curve

SINCE acute myocardial infarction is primarily a disease of the left ventricle, the estimation of left ventricular function is of paramount importance in assessing the severity of circulatory impairment produced by the infarction. Standard technics for evaluating left ventricular function during diagnostic cardiac catheterization involve the measurement of left ventricular and arterial pressures in association with cardiac output. From these measurements and from ventriculograms, various indices have been derived for the description of left ventricular function.

The present study was undertaken to evaluate the usefulness of left ventricular pressure and derived indices in characterizing the degree of myocardial impairment, the effects of various therapeutic interventions, and the ultimate prognosis in a group of patients with acute myocardial infarction.

Methods
Fourteen patients admitted to the Myocardial Infarction Research Unit with an acute myocardial infarction confirmed by ECG and enzyme analysis underwent retrograde left ventricular catheterization after informed consent. There were 11 men and three women with an age range of 40 to 80 years. Seven patients died, while seven survived to leave the hospital. The clinical status of the subjects is summarized in Table I. Left
Table 1

Clinical Status of Subjects

<table>
<thead>
<tr>
<th>Pt</th>
<th>Age</th>
<th>Sex</th>
<th>Location MI</th>
<th>LV cath; hr after MI</th>
<th>Killip class</th>
<th>Died (time after LV cath)</th>
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<tr>
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<td>67</td>
<td>M</td>
<td>AS</td>
<td>18</td>
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<td>-</td>
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<tr>
<td>2</td>
<td>73</td>
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<td>AS</td>
<td>30</td>
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<tr>
<td>3</td>
<td>56</td>
<td>F</td>
<td>AL</td>
<td>Ind</td>
<td>IV</td>
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<tr>
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<td>45</td>
<td>M</td>
<td>ASL</td>
<td>12</td>
<td>II</td>
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<tr>
<td>5</td>
<td>56</td>
<td>M</td>
<td>I</td>
<td>14</td>
<td>II</td>
<td>-</td>
</tr>
<tr>
<td>6</td>
<td>74</td>
<td>F</td>
<td>AS</td>
<td>13</td>
<td>IV</td>
<td>+ (6 hr)</td>
</tr>
<tr>
<td>7</td>
<td>73</td>
<td>M</td>
<td>SE</td>
<td>20</td>
<td>III</td>
<td>-</td>
</tr>
<tr>
<td>8</td>
<td>64</td>
<td>F</td>
<td>AL</td>
<td>8</td>
<td>IV</td>
<td>+ (7 days)</td>
</tr>
<tr>
<td>9</td>
<td>66</td>
<td>M</td>
<td>SE</td>
<td>16</td>
<td>III</td>
<td>+ (3 days)</td>
</tr>
<tr>
<td>10</td>
<td>40</td>
<td>M</td>
<td>AS</td>
<td>20</td>
<td>I</td>
<td>-</td>
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<tr>
<td>11</td>
<td>80</td>
<td>M</td>
<td>I</td>
<td>6</td>
<td>IV</td>
<td>+ (5 days)</td>
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<tr>
<td>12</td>
<td>52</td>
<td>M</td>
<td>A</td>
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<td>II</td>
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<td>20</td>
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<td>Ind</td>
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</tbody>
</table>

Abbreviations: AS = anteroseptal; AL = anterolateral; I = inferior; SE = subendocardial; A = anterior; Ind = indefinite.

LV pressures in AMI was accomplished from either the brachial or femoral artery without fluoroscopy by a technic previously described.7 The catheter was made of polyvinyl chloride (5 French) with a reverse S loop at its tip. It was attached via a Paley manifold to a Statham P23Db transducer. Intracardiac pressures were obtained following pullback of the catheter to the aorta. Cuff pressures were measured while the catheter was in the left ventricle. Lead 2 of the electrocardiogram, left ventricular pressure filtered above 10 Hz, and its electronically differentiated waveform (dp/dt) were recorded continuously on a Cleve Brush pressure ink recorder with selected beats at 100 mm/sec paper speed. Cardiac outputs were obtained in duplicate by injection of indocyanine green into the pulmonary artery from a previously placed balloon catheter,8 with sampling through the left ventricular catheter. Mean isovolumetric Δp/Δt (LV Δp/Δt) was calculated from developed left ventricular pressure (arterial diastolic – LV end-diastolic pressure) and isovolumic contraction time (ICT), i.e., developed pressure/ICT.9 In five patients the following interventions were carried out while the left ventricular catheter was in place: three patients received digoxin (0.01 mg/kg iv), one patient received norepinephrine bitartrate (Levophed 16 μg/min iv), and one patient received glucagon (5 mg iv).

The velocity of the contractile element was calculated according to the following derivations: Previous studies have suggested that the Maxwell three-component model of muscle is the most appropriate model to use at higher preloads in order to maintain a relatively constant V\text{max} with changes in preload.10, 11 In this model the parallel elastic is in parallel with both the contractile element and the series elastic and thus bears the total resting force. Thus, developed force is the appropriate force across the contractile element and series elastic during isovolumic contraction. The calculation of contractile element velocity (V\text{CE}) was derived as follows:

$$V_{CE} = \frac{d\sigma}{dt} = \frac{K\sigma + C}{R}$$

where \(\sigma\) is wall stress and K and C are the series elastic constants.12 If an isovolumic portion of systole is assumed, then wall stress (\(\sigma\)) is related to intraventricular pressure, P, by a constant, R, which includes all aspects of the LaPlace relation, \(\sigma = RP\). Substituting into the above equation, one obtains

$$V_{CE} = \frac{d\sigma}{dt} = \frac{K\sigma + C}{R}.$$  

From isolated muscle studies K = 32/muscle length, and C = 10 g/cm²/muscle length. The units of R are g/cm²/mm Hg, so that V\text{CE} is expressed in muscle length/sec. On the basis of data from a previous study of force-velocity relations,13 R averaged about 4 g/cm²/mm Hg. Thus, C/R is approximately 2.5 mm Hg/muscle length, and when developed pressure P becomes 1 mm Hg, KP is already 32 mm Hg/muscle length and is much greater than C/R. C/R is therefore small compared with KP at larger values of P and can be neglected. The modified
formula \( \frac{dp}{dt} \), however, approaches infinity as \( P \) approaches zero developed pressure (LVEDP), utilizing the Maxwell model. Thus, \( V_{\text{max}} \) approaches infinity and is indefinable as an index of contractile state. Therefore, the value of \( V_{\text{ce}} \) at 5 mm Hg (\( V_{\text{CE}} \)) was used as an approximation to \( V_{\text{max}} \). \( V_{\text{CE}} \) was calculated on three successive beats and averaged.

Error Analysis

The calculation of force-velocity relations from left ventricular pressure depends on the assumption that (1) there is an isovolumic portion of contraction, (2) the ventricle is contracting uniformly, and (3) the series elastic constants are the same for different hearts. Although these assumptions are violated in the setting of acute myocardial infarction, calculations of \( V_{\text{CE}} \) might provide some overall measure of the averages of the force-velocity relations of the left ventricle.

A catheter-tip transducer was not used in these patients with acute myocardial infarction. Previous studies, however, have suggested that there may be a little difference\(^1\) or only a small systematic difference\(^2\) between force-velocity relations calculated from a fluid-filled catheter attached to a transducer and a catheter-tip transducer. This difference is related to the underdamping typical of fluid-filled catheter-transducer systems. The error imposed in the calculation of \( V_{\text{max}} \) in the present study was estimated according to the method of Falsetti et al.\(^3\) The frequency response of the catheter-manifold-transducer system employed was analyzed by application of a square-wave pressure transient, equivalent to approximately 150 mm Hg, to the system with the catheter acting as a vent. The output of this response was amplified, electronically filtered above 10 Hz at 6 db per octave, and recorded at 100 mm/sec. The resonant frequency of the system was determined from the recorded waveform and found to range from 8 to 22 Hz. The fraction of critical damping ranged from 0.4 to 0.2, respectively. The error in calculated \( V_{\text{max}} \) for such a system is approximately \( \pm 15\% \), which compares favorably with the magnitude of error previously reported using fluid-filled catheter systems.\(^4\)

Results

The initial hemodynamic evaluations obtained on each patient after his arrival in the Myocardial Infarction Research Unit are illustrated in figure 1. For comparative purposes the patients are divided on the basis of their subsequent survival or nonsurvival. Although there was considerable overlap in heart rate, cardiac output, and systemic vascular resistance between the two groups, there was almost a complete separation in terms of mean arterial pressure. A similar separation was afforded by calculated stroke work. The nonsurvivors died of power failure and not arrhythmias.

Sample tracings of left ventricular pressure, dp/dt, and the electrocardiogram are illustrated in a representative patient in figure 2. Appropriate calculations of \( V_{\text{CE}} \) from three successive contractions in this patient are illustrated in figure 3, where each contraction is represented by a series of different symbols. Calculated \( V_{\text{CE}} \) is plotted as a function of developed pressure up to the opening of the aortic valve. \( V_{\text{CE}} \) was 1.47 muscle lengths/sec.

A summary of the left ventricular pressure measurements are indicated in figure 4. There was no difference in LV end-diastolic pressures between survivors and nonsurvivors. Left ventricular maximum dp/dt, however, showed almost a complete separation between survivors and nonsurvivors, with the level of separation at approximately 1,100 mm Hg/sec. There was a large overlap in \( V_{\text{CE}} \)

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**Figure 1**

Initial hemodynamic measurements (heart rate, mean BP, cardiac output, and systemic vascular resistance) obtained on admission. The small horizontal line is the average value for each group (survivors or nonsurvivors). The only statistically significant difference between the two groups occurred with mean BP (\( p < 0.05 \)).

Circulation, Volume XLV, February 1972
LV PRESSURES IN AMI

Figure 2

Representative tracings of the ECG, left ventricular (LV) pressure, and electronically differentiated LV dp/dt in patient 1. The end-diastolic pressure (EDP) is designated. The dots on the upstroke of the second ventricular pressure and dp/dt traces indicate the measured points used in the calculation of contractile element velocity (VCE). 

between the survivors and nonsurvivors, so that it was a poor prognostic indicator when compared with maximum LV dp/dt or mean arterial pressure.

The relationship between calculated VCE and left ventricular maximum dp/dt is illustrated in the upper panel of figure 5. Although there was a general relation between the two indices, VCE provided poor separation between survivors and nonsurvivors in contrast to maximum dp/dt. This probably reflects the dependence of maximum dp/dt on arterial pressure as well as on contractile state. The relationship between LV maximum dp/dt and mean isovolumic Δp/Δt is illustrated in figure 5, right. The good relation between the two suggests that mean isovolumic Δp/Δt can be used as an estimate of maximum dp/dt in patients in whom left ventricular pressures are not available.

Effects of Inotropic Interventions

Inotropic agents given to five patients while the LV catheter was in place allowed for a comparison of the sensitivity to changes in contractile state of indices calculated from LV pressure (VCE) and standard ventricular function curves (stroke volume or stroke work vs LV filling pressure). Three patients received digoxin 0.01 mg/kg while the left ventricular catheter was in place. One patient (no. 2) showed no detectable hemodynamic response in terms of changes in arterial pressure, cardiac output, left ventricular end-diastolic pressure, maximum dp/dt, or VCE. Patient 8 showed a good therapeutic response to digitalis, as illustrated by the measurements shown in figure 6. The pressure-velocity relation is plotted before and after digitalis therapy, while the other hemodynamic measurements are tabulated in the inset. The increased contractility of the ventricle was evidenced by the upward shift of the pressure-velocity relation and also by an increase in cardiac output, a decrease in heart rate, and a rise in maximum dp/dt despite a fall in the end-diastolic pressure. A third patient (no. 1),

Figure 3

From three successive contractions of LV pressure (fig. 2), three pressure-velocity relations are plotted (with different symbols) for patient 1. Calculations were made appropriate to the three-component Maxwell model of muscle utilizing developed pressure (P) in the equation for VCE. VCE is plotted as a function of developed pressure up to the opening of the aortic valve.
who received digoxin (fig. 7), showed a reduction in LVEDP and cardiac output, a slight increase in arterial pressure and maximum dp/dt, and no change in V_{CE}. These findings suggest that the primary measurable effects of digitalis in this patient were (1) a peripheral vasoconstriction with a reduction in venous return and preload and (2) a slight arteriolar vasoconstriction. Patient 11, given 5 mg of glucagon iv, showed no change in cardiac output, end-diastolic pressure, maximum dp/dt, or in V_{CE} at 5 mm Hg. One critically ill patient (no. 6) was given an infusion of Levophed (16 μg/min), as illustrated in figure 8. Measurements of force-velocity relations showed a minimal rise in V_{CE} measured at 5 mm Hg from 0.98 to 1.19 muscle length/sec. This slight increase in contractility was also evidenced by a slight increase in maximum dp/dt at the same preload despite a fall in heart rate.

A comparison of V_{CE} with points on a ventricular function curve is illustrated in figure 9. For each patient the level of stroke work is plotted as a function of filling pressure, while the adjacent number indicates the magnitude of V_{CE}. The lack of correlation

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**Figure 4**

Measurements of left ventricular end-diastolic pressure (LVEDP), LV max dp/dt, and V_{CE} at 5 mm Hg. The small horizontal line represents the average value for each group (survivors or nonsurvivors). The only statistically significant difference between the two groups occurred with LV max dp/dt (P < 0.05).

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**Figure 5**

(Left) Relation between V_{CE} at 5 mm Hg developed pressure and LV max dp/dt. Although the two are related, LV max dp/dt provided a better separation between survivors and non-survivors than did V_{CE} at 5 mm Hg. (Right) Relation between LV Δp/Δt and LV max dp/dt. The Δp/Δt = (aortic diastolic pressure — LVEDP)/isovolumic contraction time.
Effects of digoxin (0.5 mg iv) on the pressure-velocity relations and hemodynamics of patient 8. Digitalis produced a shift upward in the pressure-velocity relation with an increase in $V_{CE}$ from 1.50 to 2.87 muscle length/sec. There was a corresponding increase in max LV dp/dt and cardiac output, together with a reduction in end-diastolic pressure.

between $V_{CE5}$ and the level of stroke work is evident. The level of stroke work, however, was similar to mean arterial pressure and maximum LV dp/dt in separating survivors from nonsurvivors.

Discussion

As in previous reports, we have found left ventricular catheterization at the bedside without fluoroscopy in acute myocardial infarction to be relatively safe and technically feasible. The important question, however, is whether or not the information gained from this procedure adds materially to our understanding of the functional change in cardiovascular performance or to the management of these patients.

One important measurement obtained during left ventricular catheterization is a measure of the preload or left ventricular end-diastolic pressure. This measurement helps to define the relative position of the ventricle on its Starling curve. In our unit patients with LV filling pressures lower than 15 mm Hg and low cardiac outputs are given appropriate fluid volume loading in an attempt to move the left ventricle to a more optimum position on its ventricular function curve. Conversely, patients with left ventricular end-diastolic pressures greater than 20 mm Hg are given diuretics to reduce the pulmonary venous pressures. With the recent development of a balloon-tip catheter, however, LV filling pressure is now available from measurements of pulmonary capillary wedge pressure, which are easily obtained by inflating the balloon while the catheter is positioned in a peripheral branch of the pulmonary artery. The good correlation between pulmonary capillary

Circulation, Volume XLV, February 1972
Dilatation of papillary myocardium shortens. Of gree developed, acute overall calculations can form contraction and pulmonary capillary to restricted appropriate traction from measurements and left ventricular catheterization. Therefore, left catheterization provides and left of a and left ventricular pressure17 suggests, the increase in max dp/dt and VCE2 at the same end-diastolic pressure.

Contractile element velocity can be obtained from measurements of left ventricular pressure and dp/dt. Because of the nonuniform contraction pattern of the left ventricle in acute myocardial infarction, however, such calculations can represent, at best, only an overall average. In particular, as pressure is developed, the infarcted segment undergoes paradoxic systolic expansion, while the normal myocardium shortens. Furthermore, any degree of mitral regurgitation secondary to papillary muscle dysfunction or left ventricular dilatation permits muscle shortening. Thus, without an isovolumic period the calculation of VCE from pressure data alone becomes only an approximation.

Previously published studies of Vmax in the intact heart have employed a two-component model of muscle14,15 which employs total LV pressure (LVEDP + developed pressure) in the calculation of VCE. If LVEDP is small, the differences between muscle models are negligible. Thus, the lower range of normal subjects in our laboratory, which is about 1.5 muscle lengths/sec using the three-component model (unpublished data), compares favorably with data from normal subjects obtained with the two-component model.14,15 As LVEDP rises, however, the difference between data calculated by the two different models becomes apparent. Thus, calculations of Vmax

Figure 8
Effects of Levophed (16μg/min iv) on the pressure-velocity relation in patient 6. A slight increase in contractile state is suggested by the increase in max dp/dt and VCE5 at the same end-diastolic pressure.

![Graph showing pressure-velocity relationship](image)

Figure 9
Initial levels of stroke work are plotted relative to end-diastolic pressure as representative points on a ventricular function curve for each patient. Near each point the numerical value of VCE5 is indicated. Although the levels of stroke work almost completely separate survivors from nonsurvivors (P < 0.05), there is no relationship between the levels of stroke work and VCE5. The effects of digitalis (Dig) on two patients (figs. 6 and 7) are illustrated by the pairs of points connected by an arrow.

Circulation, Volume XLV, February 1972
employing the two-component model in patients with heart disease\textsuperscript{4} revealed smaller values of $V_{\text{max}}$ than the values of $V_{\text{CRS}}$ in the present study. About two thirds of the patients in the present study (including nonsurvivors) had values within the normal range. This finding may reflect intense sympathetic stimulation in some patients which raises their overall contractile state to the normal range.

The general relationship between left ventricular maximum $dp/dt$ and $V_{\text{CRS}}$ suggests that LV max $dp/dt$ may be the more useful index of overall contractile state since it is more readily obtained. However, maximum $dp/dt$ is also influenced by mitral regurgitation, acute ventricular aneurysm, and the level of arterial pressure. With hypotension, for example, LV max $dp/dt$ may be apparently reduced since the aortic valve may open before maximum $dp/dt$ would ordinarily have been reached. This may be the reason why left ventricular maximum $dp/dt$ was a better prognostic index than $V_{\text{CRS}}$ since it was also influenced by arterial pressure, which was an excellent prognostic index in this study. The correlation between LV max $dp/dt$ and isovolumic $\Delta p/\Delta t$ may allow for estimation of contractile state in the absence of direct measurement of left ventricular pressure. Since preejection period (PEP) correlates very closely with ICT, one may estimate LV max $dp/dt$ by calculation of mean electromechanical $\Delta p/\Delta t$ (arterial diastolic pressure pulmonary artery wedge pressure/PEP).\textsuperscript{9}

Another potential value of $V_{\text{CRS}}$ would be as a sensitive index to changes in contractile state. Previous animal studies, for example, have suggested that $V_{\text{max}}$ is slightly more sensitive to changes in contractile state than standard ventricular function curves.\textsuperscript{18} In the present study the sensitivity of $V_{\text{CRS}}$ to inotropic interventions appeared to be about the same as the hemodynamic measurements which describe a ventricular function curve.

It would appear, therefore, that the measurement of left ventricular pressure may provide little more information of clinical value than can now be obtained with standard hemodynamic technics employing the pulmonary artery balloon catheter and measurements of cardiac output. In selected cases left ventricular catheterization may be indicated if appropriate measurements of left ventricular filling pressure cannot be otherwise obtained from pulmonary pressures or when precise measurements of the magnitude of the "a" wave are desired. Furthermore, in selected cases with acute myocardial infarction, when early coronary arteriography and ventriculography are undertaken prior to surgical intervention,\textsuperscript{19} left ventricular pressures are routinely obtained. Further evaluation of data from such sources may provide additional information relative to the importance of measurement of the function of the left ventricle directly.

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