Effect of Coronary Artery Disease and Acute Myocardial Infarction on Left Ventricular Compliance in Man

By George Diamond, M.D., and James S. Forrester, M.D.

SUMMARY
The evaluation of left ventricular (LV) compliance by use of the pressure-volume (P-V) relationship encounters several serious difficulties. Since the P-V relationship is curvilinear, it is difficult to quantitate. Furthermore, alterations of resting heart size and geometry also produce marked changes in the P-V curve. The first derivative of the P-V relationship, however, is a precisely linear function expressed by the formula \( \frac{dP}{dV} = aP + b \). The slope of this linear function, \( a \), termed the passive elastic modulus, has been shown to be independent of initial volume and primarily and predominantly determined by changes in the stiffness of the myocardium. Myocardial wall stiffness was evaluated in three groups of subjects during LV catheterization. In 13 normal subjects \( a = 0.005 \); in 13 with coronary artery disease \( a = 0.011 \); and in 12 with acute infarction \( a = 0.045 \). The differences in stiffness among the groups were highly significant \( (P < 0.005) \).

It was concluded that a measurable change in ventricular compliance occurs with the development of coronary artery disease and that a further increase in wall stiffness occurs with the development of acute infarction. The magnitude of increase in LV wall stiffness correlated directly with immediate prognosis: 87% of those subjects with a \( \Delta P/\Delta V \) greater than 0.5 mm Hg/cc died of power failure during the acute stage of their illness. These alterations in compliance may invalidate certain traditional concepts of LV function and heart failure.

Additional Indexing Words:
Heart failure
Pressure-volume relationship
Left ventricular end-diastolic pressure
Left ventricular wall stiffness

Recent studies have suggested that decreased left ventricular (LV) compliance accompanies both coronary artery disease and acute myocardial infarction.\(^1\)\(^-\)\(^6\) The implications of these findings are of considerable clinical importance since compliance is one of the major determinants of left ventricular diastolic pressure\(^7\) and of the relationship between right- and left-heart filling pressures.\(^8\)

Attempts to evaluate ventricular compliance in man, however, have encountered several serious difficulties resulting from the curvilinear nature of the pressure-volume (P-V) relationship, the difficulty in generating in vivo P-V data, and the absence of a quantitative means of expressing LV wall stiffness. Since the P-V relationship is determined by ventricular size and geometry as well as by wall stiffness,\(^9\) ventricular wall...
stiffness cannot be characterized solely from isolated measurements of pressure and volume. Recent studies on canine hearts, however, have led to the development of a quantitative index of ventricular wall stiffness—the passive elastic modulus—that is independent of ventricular volume and is only slightly affected by geometry." The purpose of the present study was to adapt this index of wall stiffness to the intact heart in man and to quantitate alterations of LV compliance in subjects with coronary artery disease and acute myocardial infarction.

**Methods**

**Data Acquisition**

Thirty-eight subjects ranging in age from 37 to 80 years were studied (table 1). Twenty-six subjects were studied in the catheterization laboratory during evaluation of coronary artery disease. In addition, 12 subjects were studied in the Myocardial Infarction Research Unit (MIRU) from 2 to 25 hours following acute myocardial infarction documented by historical, biochemical, and electrocardiographic criteria.

In all subjects left-heart catheterization was performed via a brachial or femoral artery. Valvular insufficiency and intracardiac shunts were excluded by angiography in the catheterization laboratory subjects, and by physical examination and indicator-dilution curve analysis in MIRU subjects. Indocyanine-green cardiac output determinations were performed in duplicate by injection into the pulmonary artery and sampling from a peripheral artery. The standard deviation about the mean of duplicate cardiac output determinations was 8%. LV pressure was recorded at 100 mm/sec paper speed and high gain on a Clevite brush recorder. The frequency response of the catheter-transducer systems used was flat ± 5% to 10–15 Hz. At the heart rates encountered, this frequency response corresponded to the third harmonic of the waveforms under study. Catheterization results are shown in table 2.

On the basis of diagnosis, three groups were established:

- **Group I**: 13 subjects with no hemodynamic or angiographic evidence of cardiac disease.
- **Group II**: 13 subjects with significant angiographic coronary artery disease.
- **Group III**: 12 subjects with acute myocardial infarction.

**Definitions**

The number of terms in the literature relating to ventricular compliance has been a source of confusion, so much so that "compliance" itself is not clearly defined. Therefore, terms will be used throughout this report only in relation to the following definitions.

- **Compliance**—a general term relating to the overall passive diastolic pressure-volume characteristics of the ventricle.
Table 2

**Catheterization Data**

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<tr>
<th>Patient</th>
<th>Ventriculogram</th>
<th>Coronary angiogram</th>
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<th>EDP</th>
<th>CO</th>
<th>HR</th>
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</table>

**Group I**

| 14      | N              | LAD 90% narrowed    | 0    | 9    | 5.32 | 106  | 0.180 |
| 15      | Akinesis       | LAD, LCA occluded   | 8    | 25   | 3.20 | 65   | 0.346 |
| 16      | N              | LAD 90% narrowed    | 0    | 5    | 5.37 | 58   | 0.054 |
| 17      | Akinesis       | LAD, LCA occluded   | 10   | 20   | 2.90 | 67   | 0.231 |
| 18      | Hypokinesis    | LAD, RCA occluded   | 1    | 10   | 4.80 | 100  | 0.188 |
| 19      | N              | RCA, LCA 50% narrowed| 0    | 9    | 7.65 | 77   | 0.090 |
| 20      | Akinesis       | LAD occluded;       | 3    | 20   | 4.50 | 86   | 0.324 |
|         |                | RCA, LAD, LCA       |      |      |      |      |       |
|         |                | 75% narrowed        | 3    | 9    | 5.20 | 85   | 0.098 |
| 21      | N              | LAD, RCA occluded   | 5    | 12   | 4.40 | 83   | 0.132 |
| 22      | Akinesis       | LCA 75% narrowed    | 2    | 18   | 5.17 | 77   | 0.240 |
| 23      | N              | LCA occluded        | 0    | 6    | 2.40 | 100  | 0.250 |
| 24      | Akinesis       | LAD, LCA occluded;  | 1    | 7    | 3.30 | 76   | 0.139 |
| 25      | N              | RCA occluded        | 7    | 19   | 3.75 | 74   | 0.237 |

**Group II**

| 27      | N              | LAD occluded        | 11   | 26   | 2.30 | 100  | 0.650 |
| 28      | LAD occluded   | LAD occluded        | 6    | 12   | 3.22 | 91   | 0.168 |
| 29      | LAD occluded   | LAD occluded        | 5    | 13   | 2.10 | 73   | 0.278 |
| 30      | LAD occluded   | LAD occluded        | 8    | 32   | 4.00 | 123  | 0.740 |
| 31      | LAD occluded   | LAD occluded        | 7    | 10   | 2.60 | 90   | 0.104 |
| 32      | LAD occluded   | LAD occluded        | 0    | 9    | 5.76 | 74   | 0.397 |
| 33      | LAD occluded   | LAD occluded        | 9    | 24   | 1.92 | 110  | 0.805 |
| 34      | LAD occluded   | LAD occluded        | 13   | 25   | 1.40 | 94   | 0.870 |
| 35      | LAD occluded   | LAD occluded        | 8    | 28   | 4.40 | 88   | 0.400 |
| 36      | N              | LAD occluded        | 13   | 31   | 1.02 | 72   | 1.290 |

**Group III**

Abbreviations: ESP = end-systolic pressure; EDP = end-diastolic pressure; CO = cardiac output; HR = heart rate; N = normal; LAD = left anterior descending coronary artery; RCA = right coronary artery; LCA = left circumflex coronary artery.

indices of compliance exist, the term will not be mathematically defined.

\( \frac{dP}{dV} \) — the instantaneous rate of change in diastolic LV pressure with respect to volume, that is, the first derivative of the passive pressure-volume relationship.

\( \frac{\Delta P}{\Delta V} \) — an approximation of \( \frac{dP}{dV} \), determined as the ratio of change in pressure per unit...
change in volume during diastole. This index is derived as the difference between LV end-diastolic and end-systolic pressure divided by the stroke volume.

Wall stiffness—the property of ventricular muscle relating to its passive stress-strain characteristics, a major determinant of the diastolic pressure.

Passive elastic modulus—the slope of the linear relationship between diastolic ΔP/ΔV and intraventricular pressure, designated “a.” This index is a quantitative measure of LV wall stiffness.

Data Analysis

Previous studies from this laboratory have demonstrated a precise linear relationship between the instantaneous rate of change of diastolic ventricular pressure with respect to volume (dP/dV) and the simultaneous level of diastolic pressure (P) in the isolated anoxic-arrested canine heart, given by
dP/dV = aP + b
where a is the slope of this function, previously shown to be predominantly related to ventricular wall stiffness; and b is a constant related to intrinsic ventricular chamber size.

The data in the present study were analyzed using an approximation of this relationship by the substitution of ΔP/ΔV for dP/dV, and mean diastolic pressure, P, for instantaneous P, where ΔP is the arithmetic difference between LV end-diastolic and end-systolic pressure and ΔV is stroke volume, assuming during steady states in the absence of intracardiac shunts or valvular insufficiency that diastolic inflow equals systolic outflow.

Three consecutive LV beats immediately before and after the cardiac output determinations were averaged to obtain a single value of ΔP/ΔV and P for each subject (fig. 1). One subject with acute myocardial infarction who underwent emergency infractectomy for uncontrolled ventricular arrhythmia was studied before and after surgery. These data were analyzed separately.

Critique of Data Analysis

Certain assumptions inherent in the approximation of dP/dV by ΔP/ΔV are basic to the conclusions pertaining to ventricular compliance. These assumptions, therefore, deserve enumeration and evaluation.

Assumption 1: Compliance is Unrelated to Heart Rate or Ventricular Filling Rate and Is Relatively Constant during Diastole from One Beat to the Next

It may be expected that at rapid heart rates or very high ventricular filling rates diastolic ΔP/ΔV might become falsely elevated due to incomplete ventricular relaxation and force-dependent viscous properties of muscle tissue. Figure 2 demonstrates, however, that ΔP/ΔV bore no direct relationship to either heart rate or mean ventricular filling rate, ΔV/Δt (r = 0, P > 0.05) (stroke volume divided by diastolic filling period), in the subjects studied. All subjects were in normal sinus rhythm; therefore, beat-to-beat errors referable to a variable diastolic filling cycle were avoided. Beat-to-beat variations in ΔV were assumed to be averaged by the cardiac output determinations, and in ΔP by analysis of multiple consecutive LV waveforms.

Assumption 2: ΔP/ΔV is a Valid Approximation of dP/dV

The validity of this assumption is primarily dependent upon the accuracy of the component measurements and the magnitude of ΔP. The measurement of end-diastolic pressure offered no difficulties at the heart rates observed. End-systolic pressure, which was defined as minimum diastolic pressure, was less accurately determined, primarily due to an unknown degree of catheter overshoot. This error was minimized through the use of a mechanical damping system modified from the description of Sutterman and Wood. The ratio ΔP/ΔV is thus systematically elevated due to underestimation of end-systolic pressure. This error, however, is in some measure offset by a simultaneous underestimation of dP/dV by ΔP/ΔV. An approximation of the error level was obtained by construction of chords along hypothetic P-V curves. These chords were taken as approximations of true tangents, and the overall error of the method was thus estimated to be in the range of 10%.

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

Representative tracing of left ventricular diastolic pressure (EDP = end-diastolic pressure; ESP = end-systolic pressure) from a single study subject. A sample calculation of instantaneous diastolic stiffness (ΔP/ΔV) and mean diastolic pressure is shown on the left. CO = cardiac output; HR = heart rate.

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Results

Comparison of Patient Populations

Figure 3 summarizes results in the three patient groups. There was a general linear relationship between $\Delta P/\Delta V$ and $\bar{P}$ in each group. An increase in the passive elastic modulus, $a$, the slope of this linear plot, from group I through group III ($a_{I} = 0.005$, $a_{II} = 0.011$, $a_{III} = 0.045$) was noted, suggesting that wall stiffness increased with the development of coronary artery disease and further increased with acute myocardial infarction. The regression lines were compared for significant difference in slope by analysis of covariance. The differences in the passive elastic modulus among the three groups were highly significant ($P < 0.005$). There was no significant difference in passive elastic moduli associated with

Figure 2

Relationship of $\Delta P/\Delta V$ (see Definitions and Data Analysis in Methods section) vs heart rate (A) and mean ventricular filling rate ($\Delta V/\Delta t$) (B). Note that there was no direct correlation between these parameters, although there was a rough trend for those subjects with a low filling rate to have a high $\Delta P/\Delta V$. This was primarily due to the depressed cardiac output in these subjects (normal, solid circles; coronary artery disease [CAD], open circles; myocardial infarction [MI], triangles).

Figure 3

Relationship of $\Delta P/\Delta V$ (see Definitions and Data Analysis in Methods section) to mean intraventricular diastolic pressure in normal subjects and those with coronary artery disease (CAD) and acute myocardial infarction (AMI). Open circles represent those subjects with previous infarction. Note the general linear relationship between the two variables in each group ($r = 0.72, 0.77, 0.78$, respectively). The slope of this linear regression relates directly to wall stiffness (see text).

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a history of previous infarction in groups II and III.

Relationship of Compliance to Mortality in Acute Myocardial Infarction

A comparison of survivors and nonsurvivors following acute infarction (group III) is depicted in figure 4. Although there was no significant difference in LV end-diastolic pressure between the two subgroups, there was a highly significant difference in ∆P/∆V (P < 0.0005). A fivefold increase in mortality was observed in those subjects with a calculated ∆P/∆V > 0.5 mm Hg/cc (83% vs 17% mortality).

Effect of Infarctectomy on Ventricular Compliance

Figure 5 illustrates the change in compliance that occurred in a single subject who underwent infarctectomy during the acute stage of her illness. ∆P/∆V decreased markedly after removal of the aneurysm, despite the fact that mean diastolic pressure increased and ventricular volume was surgically decreased, both of which would be expected to increase the ratio ∆P/∆V. This fall in ∆P/∆V suggests that average left ventricular wall stiffness decreased following removal of the stiff infarcted muscle segment.

Discussion

A number of investigators have recently reported that ventricular compliance is diminished in the presence of coronary artery disease1-3 and acute myocardial infarction,4-6 and have suggested that this results from increased LV wall stiffness. Those methods which rely upon interpretation of P-V curves, however, encounter serious conceptual difficulties. First, since the P-V relationship is curvilinear, the rate of change of volume in respect to pressure becomes steeper in the absence of changes in intrinsic wall stiffness. Second, comparison of P-V relations from different hearts is often complicated by crossing of curves such that relative compliances at low pressures may be reversed at higher pressures. The greatest interpretative difficulty, however, lies in the fact that the P-V curve is the resultant of multiple factors, only one of which is wall stiffness. Major shifts in P-V curves may be produced by changes in initial ventricular volume or geometry in the absence of any change in wall stiffness.7

Figure 4
Mortality of subjects with acute myocardial infarction. Note that there was no significant difference in mortality as predicted by left ventricular end-diastolic pressure (LVEDP) illustrated to the right. On the left is illustrated the predictive value of ∆P/∆V (see Definitions and Data Analysis in Methods section). A fivefold increase in mortality was present in those subjects in whom ∆P/∆V was greater than 0.5 mm Hg/cc.

Figure 5
Effect of infarctectomy on ∆P/∆V (see Definitions and Data Analysis in Methods section) in a single subject with acute myocardial infarction. Although postoperative mean ventricular diastolic pressure is greater and ventricular size less, ∆P/∆V is reduced to near normal (solid circle) in comparison with a preoperative determination (open circle), suggesting that ventricular stiffness decreased following excision of the infarcted segment.
LEFT VENTRICULAR COMPLIANCE

Recent studies from this laboratory have attempted to overcome these difficulties in analysis of LV compliance. Thus, a precise linear relationship between instantaneous dP/dV and pressure has been demonstrated. Analysis of this relationship eliminates those problems related to comparison of curvilinear functions. The slope of this linear function, termed the *passive elastic modulus*, is by definition independent of pressure and was found to be unrelated to large alterations in initial volume, only slightly altered by changes in ventricular geometry, and directly and predominantly determined by wall stiffness. The extension of this mathematical P-V analysis to man encounters conceptual difficulties relating to the high rate of ventricular filling, the contribution of the mitral valve and left atrium to LV stiffness, and the approximation of instantaneous dP/dV by total diastolic ΔP/ΔV. For these reasons, the calculated in vivo passive elastic modulus would not be expected to correlate directly with that obtained from analysis of isolated P-V curves. In addition to conceptual limitations in extension of these animal studies to man, several practical considerations may obtain. In the animal studies it was not possible to alter ventricular geometry without changing ventricular volume, nor was it possible to increase the intrinsic ventricular chamber size. In clinical coronary heart disease, however, significant changes in chamber geometry may occur in the absence of altered ventricular volume, and chronic increases in intrinsic ventricular chamber size are common. Within limitations, however, assuming mitral valve function to be normal and variations in ventricular geometry to be small, changes in wall stiffness may be evaluated by changes in the passive elastic modulus.

In the present study it was found that the passive elastic modulus was greater in subjects with coronary artery disease than in normal individuals; subjects with acute myocardial infarction demonstrated a further increase in this index. These increases in wall stiffness are not related to the level of end-diastolic pressure, since the passive elastic modulus is independent of pressure. It is also unlikely that depression of contractility secondary to myocardial disease could account for the observed changes in the passive elastic modulus, since contractile state apparently does not affect ventricular compliance. It was concluded, therefore, that a measurable decrease in LV compliance occurred with the development of coronary artery disease and that a further decrease occurred with the development of acute infarction.

Of particular importance is the preliminary observation that the magnitude of increase in stiffness correlated directly with immediate prognosis. Thus, 83% of those subjects with a ΔP/ΔV greater than 0.5 mm Hg/cc died of power failure during the acute stage of their illness, in comparison with 17% mortality in those subjects with a ΔP/ΔV less than 0.5. In this context it was of interest that within both the coronary artery disease group and the acute myocardial infarction groups, a history of previous infarction was not associated with greater wall stiffness. Because no serial data are available on changes in compliance following acute infarction, reasons for this observation can only be suggested. First, since the increase in wall stiffness that occurs with infarction may relate predominantly to the development of intramyocardial edema and cellular infiltration, it may be a transient and largely reversible phenomenon. In addition, those individuals with greater increases in wall stiffness may be selected out by death during the acute illness. Although a decrease in LV wall stiffness was demonstrated following infarctectomy in a single patient, the effects of this operation on the overall ventricular P-V relationship may be variable. Simultaneous reductions of LV wall stiffness and intrinsic ventricular chamber size (both of which occurred with removal of the fibrotic aneurysm) have opposite effects on the P-V curve. The effect of infarctectomy on overall LV compliance, therefore, is dependent upon both the size and the stiffness of the resected segment.
Diminished infarction is left. Diminished compliance (lower left). Diminished compliance (lower right), however, will result in identical alterations. Thus, an elevated left ventricular filling pressure in acute myocardial infarction may not constitute evidence of left ventricular failure, even in the presence of radiologic evidence of pulmonary congestion.

The acute alterations in LV compliance following infarction require reevaluation of traditional concepts regarding the basis for the clinical and hemodynamic diagnosis of heart failure as it occurs in this setting. A significant number of patients with acute myocardial infarction demonstrate an elevated left ventricular filling pressure above traditional “normal” limits. Although hemodynamic parameters such as cardiac output, stroke work, and left ventricular dp/dt may remain normal, this has been interpreted by some authors as prima facie evidence of left ventricular failure, since increased end-diastolic pressure implies increased ventricular volume. Hosono has shown, however, that in patients with myocardial infarction the level of left atrial pressure measured by transseptal catheterization correlated poorly with LV end-diastolic volume. Such results are to be expected since an increase in LV diastolic pressure may result solely through an increase in ventricular wall stiffness with no increase in left ventricular volume. This pressure is transmitted across the open mitral valve directly to the pulmonary veins and capillaries and is, therefore, the proximate hemodynamic “cause” of pulmonary congestion and pulmonary edema. Thus, when increased LV filling pressure occurs in the presence of normal left ventricular chamber size and otherwise normal ventricular function, the term “heart failure” is probably not appropriate.

The presence of rales and a ventricular filling sound are frequently taken as the clinical criteria of left-heart failure. Both these findings, however, may result from diminished compliance alone. Moreover, such alterations in compliance may allow misinterpretation of Starling LV function curves. Such curves are most often constructed by plotting ventricular filling pressure against stroke work. Analysis of these curves obtained before and following myocardial infarction has led to the conclusion that left ventricular contractility is acutely diminished. In fact, however, the ventricular function curve fails to distinguish diminished contractile state from diminished ventricular compliance (fig. 6). Failure

![Figure 6](image-url)

Alternate mechanisms for apparent reduction in left ventricular function following acute myocardial infarction (heavy arrow), in which ventricular filling pressure is plotted against stroke volume (top graph). A true decrease in the Starling curve will produce this result (lower left). Diminished compliance (lower right), however, will result in identical alterations. Thus, an elevated left ventricular filling pressure in acute myocardial infarction may not constitute evidence of left ventricular failure, even in the presence of radiologic evidence of pulmonary congestion.

![Figure 7](image-url)

Hypothetic triaxial representation of the relationship between left ventricular diastolic pressure, volume, and stroke work. In this representation, the true Frank-Starling relationship (volume vs stroke work) is increased (a → b), and ventricular compliance (volume vs pressure) is simultaneously diminished (c → d). Observed contractility, as represented by the plot of filling pressure vs stroke work, is apparently diminished (e → f). Thus altered left ventricular compliance may mask changes in left ventricular function when pressure is used as an index of volume.
to recognize this fact may lead to erroneous conclusions regarding contractile state. Such compliance alterations may even mask actual increases in contractility when pressure is used as an index of volume (fig. 7). In the absence of a direct measure of ventricular volume or fiber length, therefore, no conclusions can be drawn as to the relative contribution of compliance and contractility to the resultant ventricular function curves. Definitive characterization of left ventricular performance, therefore, requires simultaneous measurement of LV diastolic pressure, volume, and stroke volume. This evaluation is currently most feasible at the bedside using echocardiographic determination of ventricular volumes\(^6\) and a pulmonary artery balloon catheter for determination of LV filling pressure and stroke volume.\(^7\)

It is concluded that LV wall stiffness may be evaluated quantitatively in the intact heart by extension of mathematical concepts developed in the isolated canine heart. Such analysis has revealed that altered ventricular wall stiffness is a significant dynamic variable which must be considered in the evaluation of both acute myocardial infarction and coronary artery disease. Failure to recognize these alterations of LV compliance may lead to invalid interpretation of ventricular function and misdiagnosis of heart failure.

References


Circulation, Volume XLV, January 1972
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_Circulation_. 1972;45:11-19
doi: 10.1161/01.CIR.45.1.11

_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

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