Functional Characteristics of Intramyocardial Capacitance Vessels during Diastole in the Dog

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SUMMARY. In order to evaluate the functional characteristics of the intramyocardial capacitance vessels during prolonged diastole, we analyzed the response of coronary vein flow after stepwise changes of coronary artery pressure in anesthetized open-chest dogs by using our newly developed laser Doppler velocimeter with an optical fiber. The peripheral portion of the great cardiac vein was isolated and the optical fiber tip was inserted into the vessel. The left anterior descending coronary artery was cannulated and connected to a reservoir to regulate coronary perfusion pressure. Intracoronary adenosine administration was carried out to avoid any change in coronary vasomotor tone. After 15 seconds of occlusion of the perfusion route, the heart was arrested by pacing-off. Two seconds later, coronary perfusion pressure was increased stepwise to a preset target pressure. This procedure was repeated by changing target pressure at 4 (or 5) different pressure levels (31—105 mm Hg). The great cardiac vein flow became zero due to the cardiac arrest and remained at zero for a moment (dead time) after the initiation of reperfusion. Then the flow reappeared and increased with first order time delay. The presence of dead time indicates the existence of unstressed volume, and the first order time delay represents the product of resistance and capacitance. The unstressed volume with a minimal vasomotor tone for perfusion pressure of 60—90 mm Hg was 5.2 ± 2.2 ml per 100 g left ventricle, which is comparable to coronary blood flow for several beats. The capacitance at perfusion pressure of 60—90 mm Hg was 0.08 ± 0.04 ml/mm Hg per 100 g left ventricle, while that at low perfusion pressure (30—50 mm Hg) was 0.14 ± 0.09 ml/mm Hg per 100 g left ventricle. These results indicate that the intramyocardial capacitance vessels have two functional components, and that the phasic nature of coronary vein flow is solely the result of the myocardial squeezing of the blood in the capacitance vessels. (Circ Res 58: 476-485, 1986)

THE flow dynamics of the coronary venous system are characterized by the phasic flow predominant in systole. Many investigators (Scaramucci, 1689, cited by Porter, 1898; Wiggers, 1954) have speculated that the blood which flows into the intramyocardial vascular compartment during diastole is expelled into the coronary vein during systole, and thus phasic coronary vein flow is predominant in systole. However, this plausible hypothesis still remains to be clarified, since the amount of data on the quantitative properties of intramyocardial vascular capacitance, which should store the blood entering the myocardium during diastole, is small. To investigate the properties of the intramyocardial vascular compartment, simultaneous measurements of coronary artery and vein flow are required (Chilian and Marcus, 1984), since these are the input and output of the intramyocardial vascular compartment. However, studies on the coronary venous system have been few, compared to those on the coronary arterial system, because of the technical difficulties of measurement. Thus, most reports on the coronary vein have been limited to evaluation of coronary sinus flow and pressure (Anrep et al., 1927; Scholtholt and Lochner, 1966; Stein et al., 1969).

Recently, we developed a laser Doppler velocimeter with an optical fiber (Imamura et al., 1979; Kajiya et al., 1981a, 1981b) and applied it to measurements of coronary artery and vein flow velocities (Kajiya, 1983; Tomonaga et al., 1983; Kajiya et al., 1984a). The advantages of our method are as follows: (1) excellent base line stability for zero velocity, (2) fine accessibility of the optical fiber probe to approaching the thin vessel wall on the beating cardiac surface, and (3) high spatial resolution for instantaneous measurement of blood flow velocity (both forward and backward flows).

Employing this method, the present study was undertaken to evaluate the quantitative characteristics of intramyocardial capacitance vessels. The flow velocity of the great cardiac vein (GCV) was measured after a stepwise increase in the coronary artery pressure during prolonged diastole, and the characteristics of the intramyocardial blood compartment were analyzed from these measurements.

Methods

Laser Doppler Blood Flow Velocimeter with an Optical Fiber

Measurements of blood flow velocity were made by a laser Doppler velocimeter with an optical fiber probe.
Details of our laser Doppler velocimeter have been reported in our previous papers (Nishihara et al., 1982; Kajiya et al., 1984b). The basic optical system is shown in Figure 1. In short, the He-Ne laser beam (632.8 nm, 5 mW) is divided by a beam splitter. Half of the initial light passing through the beam splitter is introduced into the blood stream through a graded index multimode fiber (diameter: 0.125 mm, core size: 0.05 mm). The fiber tip is inserted into the vascular lumen at an angle of 60° with the aid of a light rubber cuff (weighing less than 0.4 g). Part of the light back-scattered by flowing erythrocytes is collected by the same fiber and transmitted back. The other light divided by the beam splitter is used as the reference beam. A frequency shifter (40 MHz) is interposed in the path of the reference beam to differentiate forward from reverse flow. Photocurrent from a photo-detector (avalanche photodiode) is fed into a spectrum analyzer to detect Doppler shift frequencies. A Doppler shift of 1 MHz represents a blood flow velocity of 48 cm/sec. The sample volume of our system is approximately (π X 0.053 X 0.1) mm³ and the temporal resolution, 8 msec. The minimum detectable flow velocity with our system is 0.375 cm/sec.

Animal Preparation

Eight mongrel dogs of either sex weighing from 17 to 29 kg were anesthetized with sodium pentobarbital (25 mg/kg, iv). After intubation, the animals were ventilated by a Harvard respirator pump with room air, which was supplemented with 100% oxygen at a rate sufficient to maintain arterial oxygen tension at a physiological level. A left thoracotomy was performed on the 4th or 5th intercostal space. The heart was exposed and suspended in a pericardial cradle. The great cardiac vein (GCV) and the left anterior descending coronary artery (LAD) were carefully isolated. The optical fiber probe was inserted into the peripheral portion of the GCV with the aid of a small cuff selected from several with different diameters (1.0-3.6 mm) which was placed circumferentially around the vein. The tip of the fiber probe was fixed at an optimal position to measure the central maximum velocity after it briefly traversed the vessel from the near wall to the far wall. The LAD was cannulated with a stiff polyvinyl cannula with a three-way cock that was connected to the left subclavian artery and to a reservoir. This reservoir was filled with autologous oxygenated blood. Different levels of constant perfusion pressure were applied by varying the height of the reservoir (Fig. 2). Coronary artery pressure was measured at the cannula tip through an external auxiliary tube opening at the side of the distal end of the perfusion cannula with a strain gauge pressure transducer (Nippon-Koden DHC), and the LAD flow was measured at the distal end of the cannula by an electromagnetic flow probe (Nippon-Koden FF-020T). The flow meter zero was determined by frequent coronary inflow occlusions. The flow probe was calibrated by time blood volume collections with blood from the experimental animal.

To induce long diastole, the atrioventricular (AV) node was destroyed by the injection of 40% formalin (Steiner and Kovalik, 1968). Pacing electrodes were sewn onto the right ventricular wall. When required, lidocaine (Xylocaine, Fujisawa) was administered to suppress ectopic beats of the ventricle. Stiff polyvinyl catheters were inserted into the ascending aorta through the carotid artery and into the right atrium through the right auricle. Pressure measurements were taken with a Nippon-Koden DHC. The left ventricular pressure was measured by a catheter tip micromanometer (Millar PC-470).

Experimental Procedure for Evaluating Intramyocardial Capacitance Vessels

The experimental procedure is shown in Figure 3. Adenosine (0.3-0.7 mg/min) was continuously infused into the LAD through an external auxiliary tube of the cannula to avoid any changes in the coronary vasomotor tone during experiments. Maximum vasodilation was confirmed by disappearance of the reactive hyperemic response following 15 seconds of occlusion of the LAD. After steady states of pressure and flow parameters had been observed, the
cannula was occluded to shut off the LAD flow. The blood velocity in the GCV showed a minimal steady value within 15 seconds. Fifteen seconds after the initiation of the LAD occlusion, the long diastole was induced by the cessation of pacing. Two seconds after the cessation of pacing, the cannula was reopened and the perfusion pressure was increased stepwise to a preset target pressure. This procedure was repeated by changing the target pressure at 4 (or 5) different levels (31–105 mm Hg) in a set of trials. As a rule, the set of trials was repeated five times in each animal, and a total of 35 sets was carried out. The time courses of the blood velocity in the GCV, i.e., the time of the flow reappearance and the time constant of the flow increase, were analyzed after the initiation of reperfusion.

Indian ink was injected from the cannula at the completion of each experiment to identify the region perfused by the LAD. The region of the myocardium identified was excised and weighed to express the intramyocardial capacitance relative to a unit myocardial mass.

FIGURE 3. Schematic diagram of the experimental procedure. Fifteen seconds after the initiation of the occlusion of the left anterior descending coronary artery, a long diastole was induced by the cessation of pacing. Two seconds after the pacing-off, the cannula was reopened and the perfusion pressure was increased stepwise to a certain level. The response of the venous outflow was analyzed to evaluate the characteristics of the intramyocardial capacitance vessels. During the experiment, adenosine was continuously administered into the left anterior descending coronary artery. LAD = left anterior descending coronary artery, GCV = great cardiac vein, LDV = laser Doppler velocimeter.

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Model and Parameter Estimation

As a first approximation, we considered a simple mathematical model representing the response of the GCV flow to the stepwise increase in the LAD flow. The model assumes that the GCV flow \( F_{GCV} \) is given as

\[
F_{GCV}(t) = k \cdot F_{LAD}(t - \tau)(1 - e^{-t/\beta})
\]

where \( F_{LAD} \) is the LAD flow, \( t \) is the time after stepwise increase in \( F_{LAD} \), \( \tau \) is the dead time, and \( \beta \) is the time constant of the first order delay. The dead time is the time interval between initiation of the stepwise increase in \( F_{LAD} \) and the appearance of the GCV flow. The gain \( k \) was introduced to compensate for offsets in the flow signals in both the LAD and the GCV. Actually, we measured the GCV flow velocity \( V_{GCV} \) instead of the volume flow \( F_{GCV} \). Assuming that \( V_{GCV} \) has linear relationship with \( F_{GCV} \) and with \( F_{LAD} \), \( V_{GCV} \) is written as,

\[
V_{GCV}(t) = \alpha \cdot F_{LAD}(t - \tau)(1 - e^{-t/\beta})
\]

where \( \alpha \) is a proportional constant. The linearity between \( V_{GCV} \) and \( F_{LAD} \) will be examined later. Since 95% of the GCV flow arises from the LAD (Nakazawa et al., 1978), the unstressed volume for the LAD perfusion area was estimated by integrating the LAD flow during the dead time,

\[
\text{Unstressed volume} = \int_0^\infty F_{LAD}(t)dt.
\]
Statistical Analysis

Data are reported as the mean ± 1 SD. Differences between two means were compared using paired and unpaired t-tests. The relation between two parameters was evaluated by correlation analysis and linear regression analysis. Significant tests for the slope of the linear regression were made by t-test. The criterion for statistical significance was P < 0.05.

Results

Time Course of Hemodynamic Data

Figure 4 shows the time course of the coronary hemodynamics data during one trial. After the occlusion of the LAD, the GCV flow markedly decreased and reached a minimal, steady value within 15 seconds. Then it fell to zero with the cessation of pacing. After reopening of the LAD, it was still absent for a few seconds. Then it reappeared and increased with the first order delay, after which it finally converged to a certain level. The presence of the dead time indicates the existence of unstressed volume in the intramyocardial compartment, which is defined as the volume of the blood in a vessel at zero transmural pressure (Rothe, 1983). The first order delay relates to the viscoelastic properties of the intramyocardial reservoir, as would be predicted by a capacitive charge. The time constant of the first order delay represents the product of resistance and capacitance of the diastolic coronary circulation with minimal vasomotor tone. Thus, the mechanical lumped model illustrated in Figure 5 was adopted as the simplest, and the optimum one for explaining the results of the animal experiments. The model consists of a combination of the unstressed volume, the resistance R and the capacitance C.

Before the occlusion of the perfusion cannula, mean values of aortic pressure, coronary arterial flow in the LAD, right atrial pressure, and heart rate averaged 85 ± 16 mm Hg, 75 ± 28 ml/min, 8 ± 3 mm Hg, and 107 ± 18 beats/min, respectively.

Effect of Coronary Perfusion Pressure on the Dead Time and the Time Constant

Figure 6 shows a representative tracing of responses of the GCV flow to four different perfusion pressures. The coronary artery flow increased with

![Figure 4](image_url)

**Figure 4.** A representative tracing of variables recorded during a trial. After reopening of the left anterior descending coronary artery, vein outflow was still absent for 1.16 seconds. Then it reappeared and increased with a first order delay whose time constant was 1.21 seconds. AOP = aortic pressure, CPP = coronary perfusion pressure, CBF = coronary blood flow in the left anterior descending artery, RAP = right atrial pressure, LVP = left ventricular pressure, GCV-V = blood velocity in the great cardiac vein.

![Figure 5](image_url)

**Figure 5.** Mechanical lumped model representing the characteristics of the intramyocardial capacitance vessels during diastole. The model consists of the unstressed volume, the resistance (R) and the capacitance (C).

![Figure 6](image_url)

**Figure 6.** Responses of the great cardiac vein flow for four different perfusion pressures. The number in the figure represents each trial. Coronary inflow increased proportionally with the increment of the perfusion pressure. The dead time of the great cardiac vein flow shortened with the increment of the perfusion pressure. The time constant of the first order delay decreased with the increase in the perfusion pressure. LAD = left anterior descending coronary artery, GCV = great cardiac vein.
the coronary perfusion pressures, since autoregulation of the coronary flow was abolished by adenosine administration. The dead time decreased with the increase in the coronary perfusion pressure (the coronary artery flow). The effect of perfusion pressure on the time constant of first order delay in the GCV flow is shown in Figure 7. Each closed circle in the figure represents the mean value of the five (or four) trials in one dog. The values of the time constant decreased with the increment in coronary perfusion pressure. The negative slope of the time constant relative to the perfusion pressure was statistically significant except for two cases (Table 1).

Estimation of the Unstressed Volume in the Intramyocardial Blood Compartment

The unstressed volume in the intramyocardial blood compartment was estimated from the coronary artery inflow during the dead time. In order to express the unstressed volume relative to unit myocardial mass, it was divided by the weight of the myocardium perfused by the LAD. Figure 8 shows the relation between the coronary perfusion pressure and the unstressed volume per a unit left ventricular mass [ml per 100 g left ventricle (LV)]. The unstressed volume increased with the increase in perfusion pressure. This tendency was statistically significant except for one case (Table 1). Assuming that the diastolic coronary pressure in a physiological condition ranges from 60 to 90 mm Hg, the unstressed volume for LAD perfusion area and the volume per a unit myocardial mass were calculated for this range and resulted in values of 1.9 ± 0.8 ml and 5.2 ± 2.2 ml per 100 g LV, respectively (Table 2). The unstressed volume at low perfusion pressure (30–50 mm Hg) was 1.6 ± 0.7 ml (4.1 ± 1.8 ml per 100 g LV). The difference was statistically significant.

Estimation of the Coronary Vascular Resistance and Capacitance

The values of the coronary vascular resistance and capacitance were shown in Table 2. The resistance obtained at high perfusion pressure (60–90 mm Hg)

<table>
<thead>
<tr>
<th>No of trials (data points)</th>
<th>Time constant vs. perfusion pressure</th>
<th>Unstressed volume vs. perfusion pressure</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$(10^{-3} \times \text{sec/mm Hg})$</td>
<td>$(10^{-3} \times \text{ml/mm Hg-100 g LV})$</td>
</tr>
<tr>
<td></td>
<td>$r$</td>
<td>Significance ($P$)</td>
</tr>
<tr>
<td>Dog</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>18</td>
<td>-0.99</td>
</tr>
<tr>
<td>2</td>
<td>29</td>
<td>-2.27</td>
</tr>
<tr>
<td>3</td>
<td>23</td>
<td>-2.26</td>
</tr>
<tr>
<td>4</td>
<td>24</td>
<td>-6.07</td>
</tr>
<tr>
<td>5</td>
<td>21</td>
<td>-3.19</td>
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</tr>
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<td>7</td>
<td>19</td>
<td>-1.95</td>
</tr>
<tr>
<td>8</td>
<td>20</td>
<td>-3.43</td>
</tr>
</tbody>
</table>

$r = \text{correlation coefficient; NS = not significant.}$
was 0.23 ± 0.12 mm Hg × min/ml per 100 g LV, whereas at low pressure (30–50 mm Hg) was 0.27 ± 0.17 mm Hg × min/ml per 100 g LV. However, the increase in resistance for the lower perfusion pressure was not statistically significant. The capacitance value for the higher perfusion pressure was 0.08 ± 0.04 ml/mm Hg per 100 g LV, whereas that for the lower pressure was 0.14 ± 0.09 ml/mm Hg per 100 g LV. The difference was statistically significant.

**Discussion**

Measurement of GCV flow velocity by other methods has so far been difficult, because the vein easily becomes deformed and moves with cardiac motion. To overcome this problem, the laser Doppler velocimeter with an optical fiber was used in the present study. The Doppler signals obtained in the GCV by the present method were satisfactory in all cases. Recently, Kilpatrick et al. (1982) also measured the coronary sinus flow by means of a laser Doppler anemometer with an optical fiber. Although their method could not distinguish backward flow from forward flow, they found the laser Doppler anemometer very useful for measuring coronary vein flow.

Obvious support for the concept of intramyocardial capacitance during diastole is the observation that the coronary vein flow becomes zero during an artificially prolonged diastole, although coronary inflow still continues (Tsujioka et al., 1984; Kajiya et al., 1985). However, administration of adenosine resulted in an earlier appearance of the GCV flow in the prolonged diastole. The latter observation indicates that the blood pooled in the intramyocardial capacitance vessels will overflow into the GCV when the inflow is markedly increased.

The presence of the dead time in the GCV flow after reopening of the LAD indicated that there exists an unstressed volume in the intramyocardial vascular compartment which admits blood by a small increase in the transmural pressure. The unstressed volume may be mostly distributed in the intramyocardial veins. Although the physiological characteristics of the intramyocardial vein have not been reported upon, the prominent feature of the vein is that it is easily collapsible. When extravascular pressure equals or exceeds the pressure on the inside of the vein, the cross-section of the vessel flattens. With vessels embedded in tissue as intramyocardial vessels, transmural pressure at volumes less than unstressed volume may be negative (Rothe, 1983). To return the vessel from a flat to cylindrical configuration requires only a small change in the inside pressure (Katz et al., 1969; Wong et al., 1984). This characteristic of the vein may contribute to the

**FIGURE 8.** Effect of coronary perfusion pressure on the unstressed volume. The unstressed volume increased with the increment in the perfusion pressure. The value of the unstressed volume was expressed as the volume per unit myocardial mass.
unstressed volume. This volume may also be attributable to capillaries (Spaan et al., 1981). A sucking effect with the relaxation of extravascular pressure during the diastole could play an important role in pooling blood into the unstressed volume. Under physiological conditions, the total blood volume of the left ventricular coronary vessels has been estimated at 6–15 ml per 100 g of the myocardial mass (Salisbury et al., 1961; Rakusán et al., 1969; Morgenhöfer et al., 1973; Crystal et al., 1981). Crystal et al. (1981) reported that adenosine administration caused a 35% increase in the total volume of left ventricular coronary vessels, but no change in small vessel blood volume (i.e., vessels less than 100 μm in diameter). If we assume from these reports that the total volume of intramyocardial coronary vessels is around 15 ml per 100 g of tissue mass during adenosine administration, the unstressed volume will account for about 30% of the total intramyocardial blood volume. The unstressed volume can accommodate the blood inflow of several heartbeats, since the unstressed volume for the LAD area was 1.9 ± 0.8 ml and the blood inflow averaged 0.70 ml/one beat in our experiment.

In the present study, measurements of coronary vein flow were made in the peripheral portion of the GCV. Since the GCV flow is mostly perfused by the LAD, and the high selectivity of the GCV drainage is not affected by individual reduction of the LAD or the circumflex artery flow (Nakazawa et al., 1978), we assumed that the GCV flow velocity is linear with the LAD flow for different perfusion pressures. This linearity was examined in a steady state following the stepwise increase in the perfusion pressure. The mean value of correlation coefficients between the LAD flow and the GCV flow velocity for 35 sets of experiments in eight dogs was 0.971 ± 0.023. This indicates that the estimation of dead time and time constant by Equation 2 is pertinent for different perfusion pressures. The coefficient of variation of α in each set of experiments was less than 10%. Furthermore, the linearity between the GCV volume flow and flow velocity was examined in additional experiments with three dogs. The distal portion of the GCV was cannulated with a stiff polyvinyl cannula, and the GCV volume flow was measured at the cannula tip with an electromagnetic flowmeter. The GCV flow velocity was measured in the native GCV by our laser Doppler method. The heart was arrested, and flow responses after a stepwise increase in the coronary artery pressure was measured with an electromagnetic flowmeter and our laser Doppler method. The correlation coefficients between the volume flow and the flow velocity in the three dogs were 0.984, 0.967 and 0.997, respectively, indicating validity of the use of the GCV flow velocity.

Collateral flow could cause variation in the estimation of the unstressed volume. The driving force of the collateral flow is the pressure differences between stems of the collaterals in the LAD and those in other coronary arteries. Recently, Eng and Kirk (1984) reported that the slope of the outflow pressure vs. the retrograde flow, which is considered to be a measure of the collateral conductance was −0.13 ± 0.07 ml/min per mm Hg, while the retrograde flow was independent of the outflow pressure below approximately 20 mm Hg. Messina et al. (1985) reported that a minimal interarterial pressure gradient of 40–70 mm Hg is required before the collateral flow is detected. Thus, the collateral flow is not considered to have much influence on the estimation of the unstressed volume. Nevertheless, the presence of the GCV flow at a steady state after the LAD occlusion indicates that it originates from collateral circulation and/or venovenous anastomoses. This flow could result in underestimations of the unstressed volume. However, the peak velocity of the GCV flow just before cardiac arrest was much smaller, and the duration of the forward flow was also shorter than that before the LAD occlusion. Thus, a great part of this flow may be squeezed out by cardiac contractions. After cardiac arrest, the pressure difference between aorta and the LAD (aorta minus LAD) at 1 second after the arrest was 46 ± 12 mm Hg. The pressure differences at the mid portion of the dead time were −22 ± 13 mm Hg for the target pressure of 60–90 mm Hg and 4 ± 12 mm Hg for 30–50 mm Hg. These pressure differences could not cause significant collateral flow. Furthermore, the collateral flows before and after reperfusion have a reverse effect on the estimation of unstressed volume (underestimation and overestimation), as long as target pressures are higher than the decaying aortic pressures.

The positive correlation between the unstressed volume and the perfusion pressure indicates that the unstressed volume is not constant with respect to the perfusion pressure. Although the real reason for the pressure-dependent change in the estimated unstressed volume is not clear, it may be attributable to the increase in recruitment for higher perfusion pressure. The transmural variation in the unstressed volume, if any, may contribute to the pressure-dependent change in the unstressed volume, since changes in perfusion pressure could alter transmural perfusion during the dead time. Changes in the collateral flow and/or GCV drainage during the dead time by varying perfusion pressures may cause the pressure-dependent variation in the estimation of the unstressed volume. However, high linearity was shown between the LAD flow and the GCV velocity for a variety of perfusion pressures, and the coefficient of variations of α in Equation 2 was less than 10%. Thus, changes in collateral flow and/or variations in the GCV drainage cannot account for the positive relationship between perfusion pressure and the unstressed volume, and, at most, these confounding factors would introduce only a relatively small error.
Although 15 seconds of occlusion of the LAD, which is a necessary procedure of our experiment, is as short as in the study of the reactive hyperemic response, ischemic changes in the regional cardiac function might have developed and the regional muscle length at end-systole might have increased during this period (Theroux et al., 1974). However, during the succeeding long diastole, the regional muscle length may be restored to the condition of the long diastole without the LAD occlusion.

The time constant of the increasing phase in the GCV flow indicates the product of resistance and capacitance of the diastolic coronary circulation with a minimal vasomotor tone. The value for the perfusion pressure of 60–90 mm Hg was 1.0 ± 0.4 second. This indicates that intramyocardial capacitance with a minimal vasomotor tone can discharge into coronary veins with a time constant of 1 second during diastole. Spaan et al. (1981) estimated the effective time constant, i.e., the product of the intramyocardial capacitance and the resistance of the coronary system, at about 3 seconds from the decay curve of the coronary artery pressure after clamping the perfusion line (mean initial coronary artery pressure = 95 mm Hg). The difference in the time constant between the two experiments may be mainly due to the difference in the vasomotor tone.

The inverse relation between the reperfusion pressure and the time constant can be attributed to the changes in both resistance and capacitance with the pressure. Recently, Hanley et al. (1984) examined the relationship between coronary vascular resistance and inflow pressure by keeping the driving pressure (inflow-outflow pressure) constant, and reported that resistance varied inversely with changes in both inflow pressure (20–80 mm Hg) and outflow pressure (10–70 mm Hg). Uhlig et al. (1984), in the same laboratory, found that quadratic regression always reduced the residual sum of squares for the curve fitting of the diastolic coronary pressure-flow relations as compared with a linear regression, indicating the existence of a pressure-dependent change in resistance, although the interpretation of the coronary pressure-flow relations is still controversial. In our experiments, the values of resistance obtained for higher perfusion pressure (60–90 mm Hg) was lower than those for lower perfusion pressure (30–50 mm Hg), although this was not significant statistically. Analyzing the phasic flow in response to high frequency oscillation in the perfusion pressure, Canty et al. (1985) reported that coronary epicardial artery capacitance increased with decreasing pressure. According to their data, the capacitance at a mean arterial pressure of 50 mm Hg during vasodilation is 0.012 ml/mm Hg per 100 g LV, while that at a pressure of 110 mm Hg is 0.0034 ml/mm Hg per 100 g LV. Since our values for capacitance are much larger than those reported by Canty et al., the site of the capacitance has been mostly in the distal intramyocardial vasculature. Nevertheless, we also found that the capacitance values at high pressures (60–90 mm Hg) were lower than those at low pressures (30–50 mm Hg).

Regarding the value of intramyocardial capacitance, Salisbury et al. (1961) and Scharf et al. (1973) have reported values of 0.071 and 0.08 ml/mm Hg per 100 g LV, respectively, as measured by weight changes in isolated dog heart. According to Morgenstern et al. (1973), coronary blood volume varies by 0.085 ml/mm Hg per 100 g LV in beating dog heart with vasomotor tone. Spaan et al. (1981) reported a capacitance value of 0.07 ml/mm Hg per 100 g LV from observation of the decaying course of coronary perfusion pressure after coronary artery occlusion in a beating dog heart with vasomotor tone. Spaan (1982) also obtained capacitance values of 0.1–0.25 ml/mm Hg per 100 g LV during long diastole, as measured by related decreases in coronary artery pressure to total vein outflow. Recently, Chilian and Marcus (1984) reported values of 0.10 ml/mm Hg per 100 g LV with vasomotor tone intact and 0.21 during maximal coronary dilation in the dog heart, as measured by the coronary vein outflow after cessation of coronary artery inflow during a single long diastole. The capacitance values calculated for high perfusion pressures in the present study are in fair agreement with the values of Salisbury et al. (1961), Scharf et al. (1973), Morgenstern et al. (1973), and Spaan et al. (1981), while our estimates at low perfusion pressures are compatible with those of Spaan (1982), and Chilian and Marcus (1984). Since the studies by Spaan (1982), and Chilian and Marcus were made at low perfusion pressures, this agreement between their capacitance values and ours at low perfusion pressures may be considered reasonable.

Although measurements of coronary vein flow velocity were made in the peripheral portion of the GCV, attention to the epicardial coronary vein is required. Right atrial pressure increased slightly (from 8.4 ± 2.8 mm Hg to 10.3 ± 3.1 mm Hg) during the LAD occlusion and/or the long diastole. Granted that similar changes occur in the GCV pressure, such an increase in venous pressure would actually cause us to underestimate the magnitude of capacitance, since the epicardial venous system would be functioning as a charging capacitor. The initial overshoot of the LAD flow (Fig. 6) was due to the capacitance of the epicardial coronary arteries. Although the overshoot transient seemed to be a significant fraction of the unstressed volume, the role of the epicardial coronary artery in coronary capacitance may be small, because the distensibility of the artery is low and its partition to coronary blood volume is small. Assuming that the value of the epicardial artery capacitance is 0.0022 ml/mm Hg per 100 g LV according to Spaan (1985), we made an approximation of its contribution to the unstressed volume. The maximum value of the stepwise pressure increase was about 50 mm Hg in our
experiment. Thus, the volume change in the epicardial coronary arteries was calculated as 50 mm Hg × 0.0022 ml/mm Hg per 100 g LV = 0.11 ml/mm Hg per 100 g LV at most. This volume corresponds to about 2% of unstressed volume.

In summary, the intramyocardial capacitance vessels have two functional components, unstressed volume and ordinary capacitance. The unstressed volume with a minimal vasomotor tone is approximately 5% of the myocardium, which is comparable to coronary blood flow for several heartbeats. The time constant relating to the ordinary capacitance is about 1 second, which determines the time course of blood discharge from the intramyocardial compartment into the coronary veins during diastole. The value of the ordinary capacitance is 0.08 ml/mm Hg per 100 g LV. These data on the intramyocardial vascular compartment support the hypothesis that the process of storing blood in the capacitance vessels during diastole and expelling it during systole is the origin of the phasic coronary vein flow.

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