Effects of Coronary Venous Pressure on Left Ventricular Diastolic Distensibility

Jun Watanabe, Marc J. Levine, Fabio Bellotto, Robert G. Johnson, and William Grossman

Coronary arterial pressure and flow are known to influence left ventricular (LV) diastolic distensibility, but the influence of coronary venous pressure is unknown. To test the hypothesis that increased coronary venous pressure leads to an increase in LV wall volume and a decrease in LV diastolic distensibility, we studied excised, blood-perfused LV isovolumic dog hearts without the pericardium. In protocol I (n=8), to raise coronary venous pressure the pressure of right atrium (RA) and right ventricle (RV) was increased by the height of a blood reservoir connected with a cannula that opened in both the RA and RV. In protocol II (n=7), to isolate the effect of RV enlargement on LV diastolic distensibility (direct ventricular interaction), an isovolumic RV balloon was used with coronary venous pressure held constant at 0 mm Hg. Changes in LV diastolic distensibility were assessed by shifts of the LV end-diastolic pressure-volume relation. Changes in LV wall volume were detected by subepicardial segment length at end-diatole. The mean pressures of RA and RV (protocol I) and RV balloon only (protocol II) were increased from 0 to 15 and 30 mm Hg over a range of LV volume. In protocol I, when RA:RV pressure was increased from 0 to 30 mm Hg at three levels of LV volume (22±2, 31±3, and 40±3 ml), LV end-diastolic pressures increased significantly from 5.2±0.3 to 11.2±1.5, from 10.4±0.3 to 18.2±1.2, and from 20.2±1.0 to 28.8±1.2 mm Hg, respectively. In protocol II, when RV balloon pressure was increased from 0 to 30 mm Hg at the three LV volumes (21±3, 31±3, and 41±4 ml), LV end-diastolic pressures showed smaller increases from 5.2±0.2 to 6.6±0.2, from 9.8±0.3 to 11.6±0.6, and from 19.0±0.5 to 21.4±0.8 mm Hg, respectively. In both protocols, the LV end-diastolic pressure-volume relation shifted upward in a nearly parallel fashion, but the shift was much greater in protocol I than in protocol II. Despite constant LV volume, an increase in LV wall dimension in protocol I was significant and much greater than that in protocol II. From these results, we conclude that increased coronary venous pressure decreases LV diastolic distensibility with increasing LV wall volume, and this mechanism appears to act independently of diastolic ventricular interaction caused by RV enlargement. (Circulation Research 1990;67:923–932)

Among the determinants of left ventricular (LV) diastolic distensibility,1,2 the erectile effect of blood in the coronary vasculature has been examined by several groups.3–8 Most studies have focused on the influence of coronary arterial pressure, paying little attention to the potential contribution of coronary venous pressure. The mechanism of the coronary erectile effect has not been fully established. Because the effect correlates closely with changes in intramyocardial blood volume or LV wall volume,3,7 it is possible that coronary venous pressure may be a significant determinant of the coronary erectile effect because coronary venous pressure should be transmitted directly to coronary capacitance vessels such as capillaries, venules, and small veins through the low resistance of the venous system. The present study was designed to test the hypothesis that an increase in coronary venous pressure results in a change in intramyocardial blood volume and a decrease in LV diastolic distensibility. Because elevated coronary venous pressure most commonly results from right heart failure, we have also studied the influence of ventricular interaction on LV diastolic distensibility, specifically examining the relative roles of increased coronary venous pressure and right ventricular distention.

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Supported in part by a Program Project Grant (HL-38189) from the National Heart, Lung, and Blood Institute and by a National Research Service Award (M.J.L.) HL-07870-02.

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Received November 6, 1989; accepted May 29, 1990.
Methods

Isolated Heart Preparation

Studies were performed with isolated dog hearts perfused by a support dog anesthetized intravenously with a mixture of α-chloralose (50 mg/kg) and urethane (500 mg/kg). Details of the isolated heart model have been described elsewhere. The left panels of Figure 1 show the experimental layout in which a thin-walled latex balloon was placed in the LV through the mitral anulus and secured there, and a small drain was inserted into the left ventricle from the apex to prevent blood accumulation. The pericardium was removed in this study.

In the dog, several coronary veins drain directly into the right atrium (RA) and the right ventricle (RV) in addition to the coronary venous drainage into the coronary sinus. Accordingly, to raise coronary venous pressure uniformly it is necessary to raise the pressure in both the RA and RV. In protocol I (n=8), a large cannula (25F) with multiple holes that open in both the RA and RV was inserted from the superior vena cava (the left upper panel of Figure 1). In this preparation, we could control the pressure of the entire coronary venous outflow by raising or lowering a variable height reservoir connected with the cannula. To isolate the effect of RV enlargement and ventricular interaction, protocol II was performed (n=7), in which the RV was held isovolumic by means of a latex balloon inserted into the RV through the tricuspid anulus after opening the RA. A small drain was placed between the RV and the RV balloon. In this preparation, RV volume can be varied with a minimum change in the pressure of the coronary venous outflow.

The right panels of Figure 1 show the mean pressure in an epicardial coronary vein for protocols I and II in a representative experiment. This venous pressure was measured by a cannula (18 gauge, plastic) inserted into a small epicardial vein on the posterior surface of the left ventricle. The cannula tip was directed downstream, toward the coronary sinus. In protocol I, epicardial coronary venous pressure tracked the mean right atrial and ventricular (RA·RV) pressure closely and at steady state nearly coincided with RA·RV pressure (the right upper panel of Figure 1). On the other hand, in protocol II epicardial coronary venous pressure was not affected at all by the changes in RV balloon pressure. Thus, we could isolate the effect of coronary venous pressure on LV diastolic distensibility by evaluating the difference between data obtained in protocols I and II, when RA·RV (protocol I) or RV balloon (protocol II) pressure was changed to the same degree.

The coronary arteries were perfused by the support dog’s arterial blood via a cannula inserted into...
the aorta. Perfusion pressure was maintained constant in each experiment at approximately 80 mm Hg. Coronary blood samples were checked frequently, and pH, PO₂, and PCO₂ were kept within the physiological range. Temperature of the heart and coronary blood was kept constant at 37°C by means of a water jacket system. Blood coagulation was prevented by heparin infusion (5,000 IU, every hour). By means of LV pacing (from the LV apex), heart rate was maintained constant in each experiment. If required, the sinus node or atrioventricular node was ablated by formalin injection to prevent spontaneous tachycardia.

**Measurements**

LV pressure was measured by a micromanometer-tipped catheter (Millar Instruments, Houston) inserted into the LV balloon. Right heart pressure (RA·RV or RV balloon) was measured with a pressure transducer (Statham P23D, Gould Inc., Cleveland) through a fluid-filled system attached to the right heart cannula or RV balloon. Mean coronary perfusion pressure was measured by a pressure transducer (Statham P23D) attached to the side arm of the perfusion line. The zero of the pressure measurement system was adjusted to the midpoint of the LV. Mean coronary blood flow was measured by an in-line Doppler flow probe (5 mm i.d., Titronics Medical Instrument, Iowa City) and a Doppler unit (model 545C-A, University of Iowa, Iowa City). Calibration of the flow measurement system was performed in each experiment by means of the timed collection method.

As an indicator of LV wall volume changes, a segment length of the LV free wall was measured by a pair of ultrasonic crystals implanted horizontally in the subepicardium. In an isovolumic condition, changes in LV wall volume can be detected by changes in LV segment length as well as LV wall thickness. End-diastolic segment length was measured at the upstroke point of the LV pressure trace. Epicardial ECG was monitored to confirm adequate pacing.

**Protocols**

**Protocol I.** To uniformly increase the pressure of the entire coronary venous outflow, the mean RA·RV pressure was increased from 0 to the target pressures of 15 and 30 mm Hg at three different LV volumes. These three levels of LV volume were determined so that LV end-diastolic pressure (LVEDP) was approximately 5, 10, and 20 mm Hg when RA·RV pressure was 0 mm Hg. In this protocol, LV diastolic distensibility might be influenced not only by the coronary outflow pressure but also by enlargement of the RV; that is, by ventricular diastolic interaction through displacement of the interventricular septum. To isolate the effect of diastolic ventricular interaction, protocol II was performed.

**Protocol II.** To increase RV volume without changing coronary venous outflow pressure, an isovolumic RV balloon was used. To match the degree of RV enlargement between protocols I and II, the RV balloon was inflated with saline so that mean RV balloon pressure was 0, 15, and 30 mm Hg at three levels of LV volume (determined by the same approach as in protocol I). The unstressed volume of this balloon was 90–100 ml; that is, pressure in the balloon remained 0 mm Hg when the balloon was inflated with less than 90–100 ml of saline outside the RV. The maximum RV balloon volume that we used in protocol II was 65 ml. Thus, the RV balloon was large enough to be flaccid at all levels of distension used in this study. Figure 2 shows the phasic patterns of the RV pressure in both protocols I (panel A) and II (panel B). In both panels, the upper tracings show slow speed and the lower tracings are expanded to show the steady state. In protocol I (panel A), the mean RA·RV cannula pressure (RA·RV) was increased from 0 to 30 mm Hg by increasing the height of the reservoir. In protocol II (panel B), the mean RV balloon pressure was decreased from 30 to 0 mm Hg. Note that the phasic patterns of RV pressure in both protocols are nearly identical when the mean pressure of the RA·RV cannula or the RV balloon was 30 mm Hg. Therefore, controlling these mean pressures should be an acceptable way to match direct ventricular interaction in these two protocols. The order of the individual experimental trials was randomized in each group.

In protocol I, the effect of coronary arterial perfusion pressure was examined to assess the relative contributions of coronary inflow pressure, outflow or coronary venous pressure, and direct ventricular interaction to changes in LV diastolic distensibility caused by the increased right heart pressure.

We defined the word “distensibility” as a change in diastolic properties of the ventricular chamber such that diastolic pressure is changed at the same diastolic ventricular volume. In other words, a decrease in distensibility means that the static end-diastolic pressure-volume relation shifts upward, whether the slope changes or not. Data analysis

**Data Analysis**

Pressure, flow, and LV wall dimension data were recorded in a steady state for each trial with an eight-channel recorder (model ES 1000, Gould). Data are presented as mean±SEM. Statistical difference was evaluated by an analysis of variance with differences considered significant at the p<0.05 level.

**Results**

**Effects of an Increase in the Right Side Heart Pressure**

Figure 3 shows tracings in a typical experiment when RA·RV pressure was changed in protocol I. Note that isovolumic LVEDP decreased substantially when RA·RV pressure was lowered. Interestingly, end-diastolic subepicardial segment length decreased simultaneously despite constant LV balloon volume, suggesting that the ventricular wall volume was
decreased. Data on heart rate, peak LV pressure, and total coronary flow for all experiments are summarized in Table 1. Heart rate was not changed significantly during the interventions. Peak LV pressure was not altered significantly by changes in RA·RV pressure (protocol I) or RV balloon pressure (protocol II). Total coronary blood flow decreased in protocol I, but not in protocol II. The decrease in total coronary flow in protocol I presumably resulted from the drop in net coronary perfusion pressure (coronary arterial pressure minus coronary venous pressure).

Figure 4 shows superimposed tracings of LV diastolic pressure, RA·RV pressure (protocol I), mean RV balloon pressure (protocol II), and LV circumferential segment length of the subepicardium at two levels of RA·RV pressure (left panel) and two levels of RV balloon pressure (right panel). In protocol I, when RA·RV pressure was increased from 0 to approximately 30 mm Hg, LVEDP rose from 18 to 35 mm Hg. In protocol II, when mean RV balloon pressure was increased from 0 to 30 mm Hg, LVEDP rose from 17 to 21.5 mm Hg. LV volume was maintained constant during the interventions. Accordingly, the decrease in LV diastolic distensibility appeared to be greater in protocol I than in protocol II. Also, an increase in LV end-diastolic subepicardial segment length was greater in protocol I than in protocol II (Figure 4, lower panels), indicating an increase in LV wall volume in protocol I.

Table 2 summarizes data on changes in isovolumic LVEDP in response to an increase in RA·RV or RV balloon pressure. As can be seen, the increases in LVEDP in protocol I were significantly greater than those in protocol II at all levels of initial LV volume.
Watanabe et al. Coronary Venous Pressure and Left Ventricle

Figure 3. The tracings of protocol I, including total mean coronary flow (CF), coronary perfusion pressure (CPP), left ventricular pressure (LVP), right atrial and ventricular pressure (RA RV), subepicardial segment length of the left ventricular free wall (SL), and ECG. Left ventricular end-diastolic pressure and segment length decreased when RA RV pressure was lowered. Note that the changes in left ventricular diastolic pressure and the diastolic SL occurred immediately after the RA RV pressure reduction. CPP remained nearly constant.

Figure 5 shows LV end-diastolic pressure-volume relations when RA-RV (protocol I) or RV balloon pressure (protocol II) was increased from 0 to the target pressures of 15 and 30 mm Hg. In protocol I, the LV end-diastolic pressure-volume relations showed nearly parallel upward shifts that were significantly greater than the shifts in protocol II.

Table 1. Heart Rate, Peak Left Ventricular Pressure, and Total Coronary Flow During Changes in Right Heart Pressure

<table>
<thead>
<tr>
<th></th>
<th>Protocol I with RA-RV pressure of</th>
<th>Protocol II with RV balloon pressure of</th>
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<tr>
<td></td>
<td>0 mm Hg</td>
<td>15 mm Hg</td>
</tr>
<tr>
<td>Low LV volume</td>
<td></td>
<td></td>
</tr>
<tr>
<td>HR (beats/min)</td>
<td>113±9</td>
<td>114±12</td>
</tr>
<tr>
<td>LVP (mm Hg)</td>
<td>84±18</td>
<td>82±14</td>
</tr>
<tr>
<td>CF (ml/min)</td>
<td>242±14</td>
<td>215±16</td>
</tr>
<tr>
<td>Medium LV volume</td>
<td></td>
<td></td>
</tr>
<tr>
<td>HR (beats/min)</td>
<td>109±6</td>
<td>116±10</td>
</tr>
<tr>
<td>LVP (mm Hg)</td>
<td>101±13</td>
<td>100±10</td>
</tr>
<tr>
<td>CF (ml/min)</td>
<td>235±22</td>
<td>220±23</td>
</tr>
<tr>
<td>High LV volume</td>
<td></td>
<td></td>
</tr>
<tr>
<td>HR (beats/min)</td>
<td>109±7</td>
<td>118±13</td>
</tr>
<tr>
<td>LVP (mm Hg)</td>
<td>123±12</td>
<td>124±12</td>
</tr>
<tr>
<td>CF (ml/min)</td>
<td>230±25</td>
<td>218±26</td>
</tr>
</tbody>
</table>

In protocol I, the right atrial and ventricular (RA-RV) pressure was changed at three levels of left ventricular (LV) balloon volumes. In protocol II, only mean RV balloon pressure was changed. HR, heart rate; LVP, peak left ventricular pressure; CF, total coronary flow. *p<0.05 vs. value at RA-RV pressure=0 mm Hg.
Table 3 summarizes data on changes in the end-diastolic subepicardial segment length. The end-diastolic segment length at the initial condition of each LV volume was normalized to 100%. In protocol II, the changes in segment length were small or nonexistent. On the other hand, in protocol I the end-diastolic segment length increased significantly when RA-RV pressure was increased, and these changes were significantly greater than those in protocol II.

Effects of Coronary Arterial Occlusion on LVEDP

Table 4 summarizes the effects of occlusion of the coronary perfusion line on LVEDP when RA-RV pressure was changed at the medium LV volume of protocol I (n = 4). Although coronary occlusion decreased initial LVEDP significantly, an increase in RA-RV pressure still affected LVEDP. In Figure 6, data on LVEDP were reconstructed from Tables 2 and 4 to assess the relative contributions of the coronary bed and direct ventricular interaction to LVEDP increases. LVEDP was normalized by the value when RA-RV or RV balloon pressure was 0 mm Hg (bar d, 100%). When RV balloon pressure was increased by 30 mm Hg (protocol II), LVEDP increased by 18% (bar c). On the other hand, when RA-RV pressure was increased by 30 mm Hg, LVEDP increased by 75% (bar a). With coronary occlusion, LVEDP increased by 35% when RA-RV pressure was 30 mm Hg (bar b) and decreased by 24% when RA-RV pressure was 0 mm Hg (bar e).

Effects of an Increase in Coronary Arterial Perfusion Pressure

Figure 7 shows simultaneous tracings of LV pressure, coronary arterial perfusion pressure, and total coronary flow when coronary arterial perfusion pressure was increased by 30 mm Hg. LVEDP was almost unchanged (difference is 0.5±0.1 mm Hg [mean±SEM], n = 4, NS), in contrast to the significant increase in LVEDP when coronary venous outflow pressure was increased by 30 mm Hg (Table 2).

Discussion

The present results strongly support the hypothesis that an increase in coronary venous outflow pressure

**TABLE 2. Changes in Left Ventricular End-Diastolic Pressure (mm Hg) Caused by an Increase in Right Atrial and Ventricular Pressure (Protocol I) or Mean Right Ventricular Balloon Pressure (Protocol II).**

<table>
<thead>
<tr>
<th></th>
<th>Protocol I with RA-RV pressure of</th>
<th>Protocol II with RV balloon pressure of</th>
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<tbody>
<tr>
<td></td>
<td>0 mm Hg</td>
<td>15 mm Hg</td>
</tr>
<tr>
<td>Low LV volume</td>
<td>5.2±0.3</td>
<td>8.3±0.9*</td>
</tr>
<tr>
<td>Medium LV volume</td>
<td>10.4±0.3</td>
<td>14.2±0.7*</td>
</tr>
<tr>
<td>High LV volume</td>
<td>20.2±1.0</td>
<td>24.2±1.0*</td>
</tr>
</tbody>
</table>

RA-RV, right atrial and ventricular; LV, left ventricular.

*p < 0.05 vs. values when RA-RV pressure was 0 mm Hg in each group.
†p < 0.01 vs. values when RA-RV pressure was 0 mm Hg in each group. p < 0.05 vs. protocol II at the same pressure level.
Considerations

It is difficult to isolate the effect of a change in coronary venous pressure on LV diastolic distensibility, because there are many coronary venous pathways in the dog that open directly to the RA or RV in addition to the pathway through coronary sinus, and there are many interconnections between these coronary sinus and nonsinus pathways. Thus, increasing coronary sinus pressure alone is not an effective way to increase coronary venous pressure uniformly. In fact, some investigators have reported that obstruction of the coronary sinus has little effect on coronary blood flow, indicating that the coronary venous pressure was not increased proportionately to coronary sinus pressure. Therefore, we increased RA RV pressure by means of a variable height reservoir (protocol I) and confirmed that coronary venous pressure changed with RA RV pressure (Figure 1). However, with this approach it is clear that LV diastolic distensibility might be influenced not only by coronary venous pressure but also by the enlargement of the RV chamber. Therefore, we attempted to isolate this direct effect of RV enlarge-

| TABLE 3. Changes in End-Diastolic Subepicardial Segment Length (Percent of Control) After an Increase in the Right Heart Pressure (Protocol I) and in Mean Right Ventricular Pressure (Protocol II) |
|----------------------------------|----------------------------------|----------------------------------|----------------------------------|
| Protocol I with RA RV pressure of | Protocol II with RV balloon pressure of |
| 0 mm Hg  | 15 mm Hg  | 30 mm Hg  | 0 mm Hg  | 15 mm Hg  | 30 mm Hg  |
| Low LV Volume | 100  | 103±0.5*†  | 104±0.6††  | 100  | 101±0.1  | 102±0.4*  |
| Medium LV Volume | 100  | 102±0.5††  | 104±0.8††  | 100  | 100±0.1  | 101±0.4  |
| High LV Volume | 100  | 102±0.3††  | 103±1.3††  | 100  | 99±0.1   | 100±0.1  |

The end-diaстolic epicardial segment length at the initial condition of each left ventricular (LV) volume was normalized to 100%. RA RV, right atrial and ventricular.

*<p<0.05 vs. RA RV or RV pressure of 0 mm Hg.
†<p<0.05 vs. corresponding protocol II data.
‡<p<0.01 vs. RA RV or RV pressure of 0 mm Hg.
TABLE 4. Effects of Coronary Arterial Occlusion When Right Atrial and Ventricular Pressure Was 0, 15, and 30 mm Hg in Protocol I

<table>
<thead>
<tr>
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<th>Before coronary occlusion and RA·RV pressure of</th>
<th>After coronary occlusion and RA·RV pressure of</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>0 mm Hg</td>
<td>15 mm Hg</td>
</tr>
<tr>
<td>LVEDP (mm Hg)</td>
<td>9.9±0.8</td>
<td>12.9±0.6</td>
</tr>
</tbody>
</table>

Coronary occlusion (perfusion line cross clamp) was performed when left ventricular volume was medium (the initial target left ventricular end-diastolic pressure [LVEDP] was 10 mm Hg) in protocol I.

*p < 0.05 vs. value at right atrial and ventricular (RA·RV) pressure of 0 mm Hg in each group.
†p < 0.05 vs. value before occlusion.

ment11-13,17-21 in protocol II. It is not likely that enlargement of the right atrium affects LV diastolic distensibility in the present model. In this regard, Maruyama et al22 reported that enlargement of the RA with a balloon had no significant effect on LV diastolic distensibility after pericardiectomy. However, enlargement of the RV can affect LV diastolic distensibility after pericardiectomy22-24 through the interventricular septum.11-13 Therefore, we isolated the effect of RV enlargement on LV diastolic distensibility by using an isovolumic RV balloon (protocol II). In protocol II, coronary venous pressure was not affected by an increase in RV balloon pressure (Figure 1). Thus, the differences between protocols I and II should be caused by a variation in coronary venous pressure. These protocols allowed us to assess the relative contributions of coronary venous pressure and direct ventricular interaction to decreased LV diastolic distensibility caused by the right heart overload.

The quantitative and instantaneous assessment of the LV wall volume is recognized to be difficult. We used the circumferential subepicardial segmental length of the ventricular free wall as a qualitative indicator of the wall volume. Basically, in the isovolumic LV, LV wall dimensions such as wall thickness and segment length should reflect changes in LV wall volume. Vogel et al7 reported that both wall thickness and segment length increased with elevated coronary arterial perfusion pressure in isovolumic preparations and that there was a linear relation between these two parameters. A change in LV diastolic geometry after RV enlargement12,13 might explain LV wall dimension changes in protocol II (Table 3). Therefore, it is likely that the differences of the wall dimension data between protocols I and II were due to LV wall volume changes related to the variation in coronary venous pressure, but not to the geometric change, because geometric change occurred with both protocols I and II.

Erectile Effect of Coronary Venous System

In protocol I, LV end-diastolic pressure-volume relationships showed nearly parallel upward shifts that were significantly greater than the shifts in protocol II (Figure 5). Thus, the present results demonstrate that an increase in coronary venous outflow pressure decreases LV diastolic distensibility, and the associated change in epicardial segment length indicates that this finding is associated with increasing LV wall volume. An increase in LV wall volume is a consistent finding in reports concerning the erectile effect of the coronary arterial system.5,7,8,25,26 Therefore, it is likely that changes in coronary venous pressure act on LV diastolic distensibility through essentially the same mechanism as changes in coronary arterial pressure. However, some other potential mechanisms have to be considered. An increase in coronary venous pressure may result in tissue edema,27,28 which may change LV wall volume and LV diastolic distensibility. However, it is not likely that tissue edema played a role in the present study, because the duration of the venous pressure increase was less than 10 minutes,29 and the changes in LVEDP and the ventricular wall dimension that were seen when RA·RV pressure was altered occurred within seconds (Figure 3). The present results may be influenced not only by coronary venous pressure but also by the vasodilation caused by coronary autoregulation, because the effective coronary driving pressure was decreased with an increase in the coronary venous pressure. Therefore, the relation between a
decrease in coronary resistance and an increase in LVEDP was assessed. As seen in Figure 8, total coronary resistance decreased by 22% (range, −5% to +47%) when RA-RV pressure was increased by 30 mm Hg in protocol I, and there was no significant correlation between these two factors. Thus, this vasodilation is not likely to be a major determinant of the present results in protocol I.

Mechanism of the Coronary “Erectile” Effect

The probable mechanism of the coronary erectile effect was first postulated by Salisbury et al. They proposed that increased perfusion pressure resulted in increased intravascular blood volume, myocardial turgor, and stiffening of the LV wall. Our findings are basically in agreement with this mechanism; however, our data suggest some modifications to previous concepts. The relative importance of coronary pressure versus flow as determinants of myocardial stiffness has been controversial. The present results indicate that a change in coronary flow per se is much more important in the coronary erectile effect, because a decrease in coronary venous pressure increased LV diastolic distensibility despite an actual increase in coronary flow (Figure 3). Vogel et al report that the erectile effect was increased by adenosine-induced vasodilation at constant arterial perfusion pressure and suggested that an absolute increase in coronary vascular volume was likely to be a direct determinant. In addition to vasodilation, a redistribution of coronary vascular pressure after vasodilation may explain this coronary flow dependency; that is, microvascular pressure was increased significantly after vasodilation, even when coronary arterial perfusion pressure was maintained constant.

Interestingly, we did not observe a significant erectile effect when coronary arterial pressure was changed by the same degree as coronary venous pressure in protocol I; that is, from 80 to 110 mm Hg (Figure 7). Some other studies have also failed to observe an erectile effect with coronary arterial perfusion pressure changes of approximately 30 mm Hg. Thus, similar increments in coronary arterial and venous pressures have different effects on LV diastolic distensibility; that is, coronary venous pressure was more effective than arterial pressure in the range tested. These findings suggest that the coronary erectile effect is related to intramyocardial blood volume in an area of the coronary microcirculation to which coronary venous pressure might transmit effectively through the low resistance of the venous system. This explanation is supported by the study of Spaan et al in which intramyocardial coronary capacitance appeared to distribute in the relatively distal portion of the coronary circulation.

Finally, the present results indicate the importance of the coronary venous erectile effect on LV diastolic distensibility when right heart pressure is increased. In the past, coronary venous pressure received little attention as a determinant of LV diastolic distensibility. Coronary venous effects on LV diastolic distensibility are clearly greater than direct ventricular interaction caused by RV enlargement in the present model. However, the potential role of the pericardium was not examined in this study. The presence of the pericardium would be expected to enhance direct ventricular interaction. On the other hand, the presence of pericardium might alter the intramyocardial blood volume by changing perivascular tissue pressure indicated by changes in the zero-flow pressure of the coronary arterial pressure-flow relation. The intrapericardial pressure might affect coronary venous pressure. Thus, potential effects of the pericardium are complex, and further experiments are needed to assess the role of the pericardium in determining the effect of right heart overload on LV diastolic distensibility.

Acknowledgment

We wish to thank Alvin Franklin for his excellent technical assistance.

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KEY WORDS • coronary veins • right heart overload • intramyocardial blood volume • ventricular diastolic dysfunction • diastolic ventricular interaction
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Circ Res. 1990;67:923-932
doi: 10.1161/01.RES.67.4.923

Circulation Research is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7330. Online ISSN: 1524-4571

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