Isolated Ventricular Systolic Interaction During Transient Reductions in Left Ventricular Pressure

John C. Woodard, Edna Chow, and David J. Farrar

The volume and pressure of one ventricle have been demonstrated to modulate the volume and pressure in the contralateral chamber during systole and diastole. To quantitate the isolated systolic effects of left ventricular (LV) pressure on right ventricular (RV) mechanics, we rapidly withdrew blood from the LV immediately after diastole via an apex cannula during a single cardiac cycle in eight open-chest, open-pericardium anesthetized pigs (45 kg) and studied the effects on the RV. Reductions in LV pressure of up to 75 mm Hg were achieved in mid-systole without changing LV or RV diastolic volume or pressure. Resultant changes in RV flow and pressure development during these single unloaded beats may therefore be considered to result from pure systolic interaction. The instantaneous left-to-right systolic pressure gain (G(t)) was determined as the ratio of LV pressure change to LV pressure change as a function of time during systole, and the mean LV-to-RV systolic pressure gain was determined as the ratio of changes in mean systolic RV pressure to changes in mean LV systolic pressure. During LV unloading, there was an average reduction of 62.6±12.3% in the mean systolic LV pressure, which resulted in decreases of 13.6±6.4% in mean RV systolic pressure, 17.9±10.4% in RV stroke volume, and 27.0±11.3% in RV stroke work. G(t) was found to vary significantly within systole, reaching a minimum of 0.042±0.014 mm Hg/mm Hg at normalized time 0.70 of the systolic duration and a maximum of 0.079±0.029 at the end of RV ejection. The midsystolic value for G(t) was 0.055±0.028, and the mean systolic gain was 0.054±0.017 mm Hg/mm Hg. These results demonstrate that, independent of diastolic conditions, there is a substantial transmission of systolic forces from the LV that contributes to RV ejection and that the pressure interaction gain varies within the systolic portion of the cardiac cycle. (Circulation Research 1992;70:944–951)

KEY WORDS • ventricular systolic interaction • ventricular interdependence • ventricular function

It is well established that the volume and pressure in one ventricle may directly interact with the volume and pressure in the contralateral chamber. Ventricular interaction may be divided into that occurring during passive ventricular filling (diastolic interaction) and that occurring during contraction (systolic interaction). Diastolic interaction has been appropriately termed “ventricular interference” and manifests itself as decreases in ventricular compliance with increases in contralateral chamber pressure and volume, an effect that is potentiated at high end-diastolic volumes by the presence of the pericardium.

The same shared ventricular anatomy that is responsible for diastolic interaction also permits transmission of contractile forces between the ventricles. Since these forces are much greater in the left ventricle (LV) than in the right ventricle (RV), there could be a substantial left-to-right systolic ventricular interaction that contributes to RV performance. However, only a few studies or computer models have attempted to quantitate LV-to-RV systolic interaction, and there have been no studies determining the variation in systolic ventricular interaction during the cardiac cycle.

Therefore, the purpose of the present study was to measure isolated systolic LV-to-RV interaction in the intact heart with a new technique using transient reductions in LV pressure during the systolic portion of a single beat. By this means, the dependence of RV pressure on LV systolic pressure could be quantitated as a function of time during systole, independent of end-diastolic conditions. The results demonstrate a substantial contribution of LV pressure to RV systolic performance.

Materials and Methods

Eight farm pigs (weight, 42–48 kg; average, 44.6 kg) were premedicated with ketamine (20 mg/kg) and then anesthetized with thiopental sodium (initial bolus of 4.5 mg/kg and supplemental doses of 2.5 mg/kg every 15 minutes). A tracheostomy was performed, and the animal was ventilated using a mixture of oxygen and room air. Blood gases were maintained in the physiological range by adjustment of respiratory rate, fraction of inspired oxygen, and tidal volume. Administration of 3 mg pancuronium bromide every 15 minutes was used for muscle relaxation during data collection.

After the heart was exposed via a median sternotomy and supported in a pericardial cradle, a cannula (16 mm
and pneumatic drive pressure were recorded for timing purposes from an electromagnetic flow probe (Carolina Medical Electronics) and a Statham transducer, respectively. Data were recorded on a direct-writing chart recorder (model 2800, Gould, Cleveland, Ohio) and digitized on a PDP 11/23 minicomputer (Digital Equipment Corp., Marlboro, Mass.) at a rate of 100 samples per second.

Experimental Protocol

Eight-second data sets were taken with respiration suspended at end expiration. Control data, consisting of the first five to eight cardiac cycles, were recorded with the VAD driver supplying a pressure of 200 mm Hg, which was well in excess of the LV systolic pressure and thus ensured that the VAD blood sac was empty. After the R wave of the unloaded beat, pneumatic drive pressure was rapidly reduced to −100 mm Hg, allowing the VAD to fill at a rate determined by the difference between the pneumatic drive pressure and that in the LV and the fluid impedance of the cannula. After each data set, the VAD was emptied into the heart, and sufficient time was allowed for stabilization before the unloading sequence was repeated for a total of five sets for each animal. Verification that end diastole was unaffected by unloading was provided by the absence of change in the end-diastolic pressure and cardiac dimensions from that of the preceding control beats.

Data Analysis

Data records were analyzed using software developed in our laboratory. Any recording with unstable ventricular pressures or electrocardiographic evidence of arrhythmia during the control period was excluded. Pressure and flow waveforms from the five to eight cardiac cycles before the unloaded cycle were averaged to define the control beat, which was then subtracted from the unloaded beat. The resulting differences in pressure (as a function of time) were then used to calculate the instantaneous gain. Because of heart rate differences between animals, the period of systole was normalized such that the time from end diastole to the end of RV ejection ranged from 0.0 to 1.0. The intervening time between these points was divided into 5% increments, and the corresponding pressures and flows at each of the 21 points were calculated by linear interpolation of the sampled points.

The instantaneous left-to-right pressure gain is defined as the ratio (mm Hg/mm Hg) of RV pressure change to LV pressure change (from the control to the unloaded beat). Instantaneous gains were calculated for the last 70% of systole, by which time the LV pressure was consistently reduced in all animals. The "mid-systolic" gain was defined as the average gain between normalized systolic times of 0.4 and 0.6. Mean pressure gain was defined as the ratio of changes in mean RV systolic pressure divided by changes in mean LV systolic pressure. The mean systolic pressures were in turn calculated by integration of LV and RV pressures during systole. RV stroke volume was computed by integration of pulmonary artery flow, and RV stroke work was computed from the product of mean RV developed pressure and stroke volume. The instantaneous changes in pulmonary artery flow as a result of

i.d.) was inserted into the apex of the LV and connected to a ventricular assist device (VAD) (Thoratec, Berkeley, Calif.).20 The outlet port of the VAD was blocked, and the one-way valves were removed so that blood was both withdrawn and returned to the LV through the apex cannula (Figure 1). Normal operation of a VAD involves the cyclical application of vacuum and pressure to the flexible blood sac in a rigid housing to fill and empty the pump. However, in these experiments, the VAD driver was modified to allow a single rapid filling of the VAD triggered by the R wave of the electrocardiogram.

Cardiac dimensions were obtained using a sonomicrometer (Triton, San Diego, Calif.). The anteroposterior RV dimension was measured from a pair of hemispheric crystals sutured to the epicardium; septal–free wall dimensions for both ventricles were obtained from hemispheric crystals on the epicardium of the respective ventricular free walls and a cylindrical crystal placed via a 16-gauge needle track into the ventricular septum (Figure 1).

Catheter-tipped manometers (SPC 350, Millar Instruments, Houston, Tex.) were inserted via stab wounds in the ventricular free walls to measure RV, LV, and pulmonary artery pressures. Aortic pressure was recorded from a Statham transducer/fluid-filled catheter. An electromagnetic flow probe (Carolina Medical Electronics, King, N.C.) was placed around the pulmonary artery to measure RV stroke volume. VAD inflow

FIGURE 1. Schematic diagram of experimental preparation illustrating the method for pressure unloading the left ventricle and the instrumentation used to measure the resulting ventricular interaction. AoP, aortic pressure; PA flow, pulmonary artery flow; PAP, pulmonary artery pressure; RVP, right ventricular pressure; LVP, left ventricular pressure; RVAP, right ventricular anteroposterior dimension; RVSFV, right ventricular septal–free wall dimension; LVSFW, left ventricular septal–free wall dimension; VAD, ventricular assist device. Triggering the ventricular assist device from the R wave allowed rapid withdrawal of blood from the left ventricle via the apex cannula (arrow) during ventricular systole.
LV unloading were calculated as the ratio of RV flow change to LV pressure change. RV ejection time was defined as beginning at the rapid upswing of the pulmonary artery flow signal and terminating as the flow signal crossed zero.

Mean data from each animal were calculated from the five data records. These mean values were then pooled and are presented as the mean±SD. Paired t tests were performed on the data from control and unloaded beats, with a value of p<0.05 considered statistically significant. A one-way analysis of variance with repeated measures followed by the Fisher post hoc test was used to determine if the instantaneous pressure gain varied with time during the systolic period.

Results

Hemodynamic measurements from a typical data set are shown in the top four tracings of Figure 2. In the seventh beat (“unload” in Figure 2), VAD drive pressure was reduced from 200 to −100 mm Hg (first tracing), allowing ventricular emptying into the VAD and causing a rapid fall in LV systolic pressure (second tracing). Ensuing reductions in the RV pressure and pulmonary artery flow are shown on the third and fourth tracings. Beat-to-beat changes in each variable were calculated by digitally shifting each recorded variable by one cardiac cycle and subtracting, thus producing the difference from one beat to the following beat. These beat-to-beat changes in LV pressure, RV pressure, and pulmonary artery flow are shown on expanded scales on the lower three tracings of Figure 2 and demonstrate the stability of the preparation up to the unloaded beat, in which there are reductions in all three variables.

Pooled ventricular pressures and pulmonary artery flow for all experiments are shown superimposed in the left panels of Figure 3 as a function of normalized systolic time, with the control beats indicated by open symbols and unloaded beats represented by filled symbols. The right panels of Figure 3 present the changes in these variables calculated by subtracting the data at common time points. It can be seen that control and unloaded beats begin with unchanged end-diastolic pressures in both ventricles (at normalized systolic time 0.0). For the unloaded beats, pneumatic and mechanical delays were such that 60 msec elapsed between the R wave of the electrocardiogram and the flow of blood into the VAD; thus, the initial rise in LV pressure in the unloaded beat was unchanged (Figure 3, top left panel). Once VAD flow was established, LV pressure fell progressively throughout systole, ending at a value somewhat below that of the previous end diastole. Concomitant reductions in systolic RV pressure and flow are evident throughout the cycle.

The maximum reduction in LV pressure was 81±11 mm Hg or 88%, which occurred at a normalized systolic time of 0.80. This resulted in instantaneous reductions of 3.5±1.5 mm Hg (14%) in RV pressure and 1.5±0.9 l/min (21%) in pulmonary artery flow at the same systolic time. Corresponding reductions were seen throughout systole of the unloaded beat. Changes in RV pressure and pulmonary artery flow between control and unloaded beats increased rapidly in the initial phase to a normalized systolic time of ~0.3. From a time of 0.3–0.75, however, those differences were fairly constant, despite a continuing fall in LV pressure.

Pooled LV-to-RV systolic pressure gain as a function of normalized systolic time, calculated from the differences between control and unloaded data, are shown in the upper panel of Figure 4. Over the normalized systolic time period from 0.3 to 0.7, there was a significant (p<0.001) decrease in gains from 0.072±0.022 to 0.042±0.014 mm Hg/mm Hg, reflecting the fact that the change in LV pressure was increasing over this time, whereas the change in RV pressure was relatively constant. From normalized systolic time of 0.75 to the end of systole, the pressure gain increased to the highest value of 0.076±0.028 mm Hg/mm Hg at a time of 1.0, which also was significantly greater than the minimum gain at time 0.70. The corresponding
changes in pulmonary artery flow divided by changes in LV pressure also varied between a maximum of 0.043±0.021 l·min⁻¹·mm Hg⁻¹ at time 1.0 to a minimum of 0.021±0.009 l·min⁻¹·mm Hg⁻¹ at time 0.80 (Figure 4, bottom panel).

Overall hemodynamic data, pooled from all experiments for both control and unloaded cardiac cycles, appear in Table 1. During the unloaded beat, LV peak systolic pressure and mean systolic pressure were significantly reduced, but LV end-diastolic pressure was unchanged. There were corresponding reductions in RV peak and mean systolic pressure without changes in RV end-diastolic pressure. No significant difference between end-diastolic dimensions or pressures are evident between control and unloaded beats. However, most systolic dimensions were significantly changed by unloading: the LV end-systolic septal–free wall dimension was significantly reduced, as was the RV end-systolic anteroposterior dimension. The fractional change from diastole to systole was significantly increased for the LV, whereas the diastolic-to-systolic dimensional change in the RV was reduced in the septal–free wall direction but increased in the anteroposterior direction. LV unloading also reduced RV ejection time and resulted in significant reductions in RV stroke volume and stroke work.

The mid-systolic LV-to-RV systolic pressure gain was 0.055±0.028, and the mean systolic pressure gain was 0.054±0.017 mm Hg/mm Hg (Table 2). For each millimeter of mercury drop in mean systolic LV pressure, there was a 0.13±0.07-ml reduction in RV stroke volume and a 0.66±0.28-mJ fall in RV stroke work (Table 3).

**Discussion**

Classification of ventricular interaction into “diastolic” and “systolic” provides a useful framework for conceptual understanding of two different mechanisms mediated by a common anatomy. However, consensus on the significance of systolic interaction has not been achieved, partially because of the variety of conditions under which it has been measured and the lack of clear delineation between systolic and diastolic events. We have chosen the label “isolated” systolic interaction to indicate the following: alterations in systolic function occurring purely as a result of changes in systolic variables of the other ventricle. Previous studies of “systolic interaction”²,³,¹,³,⁶ fall outside this definition, because end-diastolic conditions were uncontrolled and these papers more properly document a melding of actual isolated systolic interaction with the systolic sequelae of changes in end-diastolic conditions.

In our study, at constant end-diastolic conditions in both ventricles, during a single cardiac cycle, LV pressure was rapidly reduced after the isovolumic contraction phase. Utilization of this method ensures that preload in the septum and free walls remains unchanged from the previous beat and therefore that the potential work of the heart during the unloaded beat was identical to the control beat. Measurement of cardiac dimensions verified that end-diastolic conditions were constant, but during the unloaded beat, the intraventricular septum shifted to the left, RV septal–free wall contraction was reduced, and the systolic change in anteroposterior dimension was slightly enhanced. Mean LV pressure was reduced by 63±12%, resulting in reduced RV pressure, stroke volume, and
stroke work. Also, the duration of RV ejection was shortened. The gains, ranging from 0.042 to 0.076 mm Hg/mm Hg, suggest that systolic gain is time varying, with the highest gains at the earlier and later parts of systole and the lower gains occurring around mid-systole. The mean systolic LV-to-RV interaction gain was 0.054 mm Hg/mm Hg.

A quantitative relation between the developed pressure in one chamber and the volume of the other has been obtained in isolated, isovolumically beating hearts by Santamore et al., who found that reducing LV volume from its "ideal" value caused a 5.7% decrease in RV developed pressure. Weber et al., using an isolated-heart preparation, concluded that the degree of this left-to-right systolic interaction was independent of RV volume. However, in another isolated-heart preparation, Yamaguchi et al. showed that, with increases in LV volume, the slope of the RV end-systolic pressure-volume relation increases and the volume intercept decreases. These changes support the concept that RV stroke work is augmented at increasing LV pressures and volumes. Their data also imply that any change in contralateral ventricular geometry may alter regional preload in the opposite ventricular free wall and that this is, in part, responsible for the increased end-systolic pressure–volume relation that is seen at increasing volumes in the contralateral ventricle. A leftward shift in the end-systolic pressure–volume relation intercept without a change in slope had also been previously reported by Maughan et al., who maintained constant contralateral ventricular volume using a servo system.

Using aortic occlusion to produce an abrupt increase in LV afterload, Langille and Jones demonstrated coincident increases in LV and RV peak systolic pressure, from which a pressure gain of 0.086 mm Hg/mm Hg may be derived, which is substantially higher than our mid-systolic gains of 0.058. However, Maughan et al. measured pressure gain at end systole in isolated, blood-perfused canine hearts and calculated a left-to-right systolic pressure gain of 0.08, which is very close to our value of 0.076 at end ejection. Maughan et al found that this left-to-right gain was half that of right-to-left gain over a wide range of ventricular volumes.

Elzinga et al. have also measured the ejection performance of the RV during rapid changes in LV afterload in isolated feline hearts. Differences in RV stroke volume of 30–50% were observed when the LV contracted isovolumically or against a low impedance load, which implies a greater interaction than we found. However, although RV end-diastolic pressure was controlled in their preparation, their results do not represent isolated systolic interaction, because of the inevitable variations in RV end-diastolic volume brought about by changes in left-side volume and the ensuing diastolic interaction.

In the computer model of ventricular interaction of Santamore and Burkhoff, the LV-to-RV interaction gain is predicted to be 0.095 mm Hg/mm Hg, with no provision for any time-varying phenomena. This gain was theoretically calculated from the ratio of $E_{ref}/(E_{ref} + E_e)$, where $E_{ref}$ and $E_e$ are the elastances of the RV free wall and interventricular septum from Maughan et al. This theoretical gain is greater than our mean gain of 0.054 but lies within the 95% confidence interval for our peak gain, which extends from 0.055 to 0.097. Because their gain was calculated for steady-state conditions, a direct comparison with our instantaneous value is difficult to interpret.

Our measurements of time-varying gain show higher values (0.072) in early ejection than after peak ejection (0.042). The systolic interaction gains might have been even higher if LV pressure could have been reduced earlier in the cycle. Although the absolute magnitude of the gains appears small because of the large difference in the systolic pressures of the two ventricles, this belies their true significance. Our results indicate that if the changes in flows and pressures are expressed as a percentage of the control values in each ventricle, a 100% reduction in LV pressure would result in a 25% decrease in mean RV pressure generation. Similarly, complete unloading of the LV would result in a 43% decrease in RV stroke work and 29% reduction in RV stroke volume. The 10-msec decrease in ejection time observed in our study is most probably a result of ejection occurring at a lower RV pressure, with the rate of pressure decline being unchanged.

One possible shortcoming in the ventricular unloading method is that a cannula in the LV apex may potentially disrupt normal ventricular contractile function to some degree. However, ¬5 g myocardium is
TABLE 1. Data for Control and Unloaded Cardiac Cycles

<table>
<thead>
<tr>
<th>Pressures</th>
<th>n</th>
<th>Control beat</th>
<th>Unloaded beat</th>
<th>Change (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>LV</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PSP (mm Hg)</td>
<td>8</td>
<td>96.3±7.2</td>
<td>67.3±11.9*</td>
<td>−30.1</td>
</tr>
<tr>
<td>MSP (mm Hg)</td>
<td>8</td>
<td>75.3±8.7</td>
<td>28.2±9.1*</td>
<td>−62.6</td>
</tr>
<tr>
<td>EDP (mm Hg)</td>
<td>8</td>
<td>9.9±4.2</td>
<td>10.1±4.2†</td>
<td>2.0</td>
</tr>
<tr>
<td>RV</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PSP (mm Hg)</td>
<td>8</td>
<td>29.8±6.0</td>
<td>26.7±5.3*</td>
<td>−10.4</td>
</tr>
<tr>
<td>MSP (mm Hg)</td>
<td>8</td>
<td>22.1±5.2</td>
<td>19.1±4.1*</td>
<td>−13.6</td>
</tr>
<tr>
<td>EDP (mm Hg)</td>
<td>8</td>
<td>3.6±1.0</td>
<td>3.5±1.1†</td>
<td>−2.8</td>
</tr>
<tr>
<td>Aortic (mm Hg)</td>
<td>8</td>
<td>83.5±9.8</td>
<td>64.9±10.0*</td>
<td>−22.3</td>
</tr>
<tr>
<td>PAP (mm Hg)</td>
<td>7</td>
<td>23.2±5.8</td>
<td>21.5±5.3*</td>
<td>−7.3</td>
</tr>
<tr>
<td>Dimensions</td>
<td></td>
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<td></td>
</tr>
<tr>
<td>LV</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ED SFW (mm)</td>
<td>7</td>
<td>55.5±6.0</td>
<td>55.5±6.0†</td>
<td>0.0</td>
</tr>
<tr>
<td>ES SFW (mm)</td>
<td>7</td>
<td>51.9±5.7</td>
<td>44.5±4.6*</td>
<td>−14.3</td>
</tr>
<tr>
<td>ΔSFW (%)</td>
<td>7</td>
<td>6.5±2.2</td>
<td>19.4±7.1*</td>
<td>199.0</td>
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<tr>
<td>RV</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ED SFW (mm)</td>
<td>7</td>
<td>45.1±9.6</td>
<td>45.1±9.6†</td>
<td>0.0</td>
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<tr>
<td>ES SFW (mm)</td>
<td>7</td>
<td>40.3±10.2</td>
<td>43.8±8.9†</td>
<td>8.7</td>
</tr>
<tr>
<td>ΔSFW (%)</td>
<td>7</td>
<td>11.3±4.6</td>
<td>2.5±6.5*</td>
<td>−75.2</td>
</tr>
<tr>
<td>ED AP (mm)</td>
<td>7</td>
<td>63.1±6.9</td>
<td>63.1±6.9†</td>
<td>0.0</td>
</tr>
<tr>
<td>ES AP (mm)</td>
<td>7</td>
<td>61.4±6.8</td>
<td>60.4±6.4*</td>
<td>−1.6</td>
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<tr>
<td>ΔAP (%)</td>
<td>7</td>
<td>2.7±1.5</td>
<td>4.2±2.2*</td>
<td>55.6</td>
</tr>
<tr>
<td>SW, SV, and $T_{eq}$</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>RV</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SW (mJ)</td>
<td>8</td>
<td>114.1±36.0</td>
<td>83.6±30.9*</td>
<td>−26.7</td>
</tr>
<tr>
<td>SV (ml)</td>
<td>8</td>
<td>32.5±5.1</td>
<td>26.7±6.5*</td>
<td>−17.9</td>
</tr>
<tr>
<td>$T_{eq}$ (msec)</td>
<td>7</td>
<td>255.0±29.0</td>
<td>245.0±23.0*</td>
<td>−3.9</td>
</tr>
</tbody>
</table>

Values are mean±SD. $n$, Number of pigs; LV, left ventricular; PSP, peak systolic pressure; MSP, mean systolic pressure; EDP, end-diastolic pressure; RV, right ventricular; PAP, pulmonary artery pressure; ED, end diastole; SFW, septal-free wall dimension; ES, end systole; Δ, change between diastole and systole; AP, anteroposterior dimension; SW, stroke work; SV, stroke volume; $T_{eq}$, ejection time. *
*p<0.05 and †p=NS compared with control beat by paired $t$ test.

removed from the ventricular apex for cannula insertion, which comprises only 2–3% of the average porcine heart and therefore may be considered negligible. An additional small amount of tissue may be rendered noncontractile by cannula fixation sutures. Shortening of the heart in the base-apex direction may also be slightly altered by some “tethering” action of the cannula and VAD, which are attached to the heart but which still allow movement. But these effects are unlikely to significantly alter our results since any effect would alter the control and unloaded beats in a similar manner. Our preparation probably introduces less artifact than isolated-heart preparations, which use rigid sewing rings in the mitral and tricuspid positions with interruption of the mitral chordae. Interruption of the chordae has been demonstrated to significantly change

TABLE 2. Systolic Left Ventricular-to-Right Ventricular Pressure Gains at Normalized Systolic Times

<table>
<thead>
<tr>
<th>LV-to-RV systolic pressure gain (mm Hg/mm Hg)</th>
<th>Normalized time</th>
</tr>
</thead>
<tbody>
<tr>
<td>Maximum</td>
<td>0.076±0.028</td>
</tr>
<tr>
<td>Minimum</td>
<td>0.042±0.014</td>
</tr>
<tr>
<td>Midsystolic</td>
<td>0.055±0.028</td>
</tr>
<tr>
<td>Mean</td>
<td>0.054±0.017</td>
</tr>
</tbody>
</table>

Values are mean±SD. LV, left ventricular; RV, right ventricular. Normalized time was 0 at end diastole and 1.0 at end ejection. Mean systolic gain was calculated from changes in mean LV and RV systolic pressures.

TABLE 3. Changes in Right Ventricular Function During Reductions in Left Ventricular Pressure

<table>
<thead>
<tr>
<th>Changes in RV function</th>
<th>Normalized time</th>
</tr>
</thead>
<tbody>
<tr>
<td>$ΔRV$ SV/ΔLV MSP (ml·mm Hg$^{-1}$)</td>
<td>0.13±0.07</td>
</tr>
<tr>
<td>$ΔRV$ SW/ΔLV MSP (mJ·mm Hg$^{-1}$)</td>
<td>0.66±0.28</td>
</tr>
<tr>
<td>$ΔQ_{pa}$/ΔLV MSP (1·min$^{-1}$·mm Hg$^{-1}$)</td>
<td>0.037±0.016</td>
</tr>
</tbody>
</table>

Values are mean±SD. RV, right ventricular; Δ, change; SV, stroke volume; LV, left ventricular; MSP, mean systolic pressure; SW, stroke work; $Q_{pa}$, pulmonary artery flow. Normalized time was 0 at end diastole and 1.0 at end ejection.
global systolic performance in the ejecting heart and modify ventricular geometry.\textsuperscript{21}

Ventricular systolic interactions are primarily mediated via the intraventricular septum. Although the septum may be considered to be morphologically part of the left ventricle,\textsuperscript{2} its motion (thickening) during systole in the normal heart contributes equally to LV and RV ejection.\textsuperscript{22} The LV free wall is also implicated in RV function, as shown by reduction in RV developed pressure during LV free wall ischemia\textsuperscript{11} and after LV free wall transsection.\textsuperscript{17} Further evidence of both septal and free wall-mediated interaction mechanisms is illustrated by unchanged systemic venous pressure,\textsuperscript{6} RV pressure, and maximum RV dP/dt\textsuperscript{23} after prothetic RV free wall replacement. One study explained these findings with cineangiography, showing that contraction of the LV influenced RV ejection by two means: bulging of the interventricular septum into the RV cavity and pulling the patch toward the septum.\textsuperscript{24}

Farrar et al\textsuperscript{25} and Chow and Farrar\textsuperscript{26} have studied the effects of steady-state reductions in LV pressure on RV function. These studies found that reducing LV peak systolic pressure by up to 90% produced parallel shifts but no changes in slope in the RV septal-free wall pressure--dimension relation and preload-recruitable stroke work. Thus, reduced LV pressure produced a septal shift without major changes in RV output, which we also found under conditions of RV ischemia.\textsuperscript{27} These studies of ventricular interaction have direct clinical significance in patients with LV assist devices for end-stage heart failure, because good RV function is required while the device is operating with reduced LV pressure.\textsuperscript{28} However, the findings of these studies cannot be attributed to isolated systolic interaction because of concomitant changes in diastolic function. To separate these two effects, we\textsuperscript{29} previously used a computer simulation of LV pressure unloading with a LV assist device, which was based on the Santamore-Burkhoff model.\textsuperscript{18} These results showed that diastolic and systolic interaction tend to counteract each other during steady-state LV pressure unloading in the normal heart. The simulation predicted that although systolic function of the RV may be diminished when LV pressure is reduced (via systolic interactions), the concomitantly increased RV diastolic compliance (via diastolic interaction) allows the RV to compensate by operation at higher end-diastolic volumes. That is, the heart may provide diastolic compensation for the reduction in systolic function caused by depressurization of the contralateral ventricle.

This study confirms that there are substantial contributions from LV pressure to RV systolic function. In contrast to other methods, as a result of systolic ventricular interactions we were able to observe instantaneous changes in RV pressure throughout systole in response to reductions in LV pressure and in the absence of preload changes in either ventricle. The results suggest that systolic ventricular interaction is time varying within the cycle.

References


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