Effects of Chronic Right Ventricular Volume and Pressure Loading on Left Ventricular Performance

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SUMMARY
The effects of chronic right ventricular (RV) distension on left ventricular (LV) function were assessed in dogs 3 weeks after pressure and volume loading of the RV had been produced by the emplacement of a constricting band around the pulmonary artery and the creation of tricuspid insufficiency. This resulted in ascites, RV hypertrophy and dilatation, an increase in RV end-diastolic pressure (EDP), and a reduction of RV and LV norepinephrine concentrations. Peak LV pressure, wall stress, and dp/dt were measured during isovolumic beats at LVEDP's of 1 to 20 mm Hg, and comparisons at matched EDP's were made among the normal, sham-operated, and RV stressed dogs. Under these circumstances, LV function appeared to be depressed appreciably. The effects of RV volume loading on the pressure-volume curve of the LV in the potassium-arrested heart were assessed in a separate group of dogs. The pressure-volume curve of the LV was shifted so that any given LV volume was associated with a higher LV pressure. Thus, when peak pressure and wall stress were related to LV end-diastolic volume rather than to LVEDP, the relationship did not appear to differ from normal; peak dp/dt and peak VCE, however, remained depressed. This depression may represent a decrease in LV contractility consequent to the chronic RV stress or may reflect alterations in LV geometry. Thus, when the RV is subjected to a chronic flow and pressure load, assessment of LV function may be unreliable when indices of contractility are employed that require measurements of LVEDP.

Additional Indexing Words:
Force velocity Left ventricular compliance Left ventricular contractility
Left ventricular failure Left ventricular pressure-volume relationships
Reversed Bernheim effect

The question of whether isolated right ventricular failure can lead to a depression of left ventricular function has intrigued physicians for years. Clinical studies have suggested that left ventricular hypertrophy may occur in patients with cor pulmonale, and several investigations have shown that when isolated right ventricular failure is experimentally produced in animals, biochemical and morphologic changes similar to those present in the right ventricle occur in the unstressed left ventricle. These include a decreased concentration of norepinephrine, an abnormal histochemical appearance of the adrenergic nerve fibers, depressed myofibrillar adenosine triphosphatase activity, and increased amounts of collagen. The functional significance of these changes, however, has not been assessed.

In 1962, Hecht and coworkers found that left ventricular end-diastolic pressure was elevated when right heart failure occurred in cattle with Brisket disease, an abnormality

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causing severe pulmonary hypertension. After reversal of the disease process, left ventricular end-diastolic pressure returned to normal, and in one animal a marked enhancement of the Starling ventricular function curve was demonstrated. However, increases in right ventricular filling within the physiologic range have been shown to shift the pressure-volume curve of the left ventricle so that a given left ventricular volume is associated with a higher left ventricular pressure. It is thus possible that the elevated left ventricular pressures in right heart failure may simply reflect a change in the pressure-volume characteristics of the left ventricle, rather than a depression of left ventricular contractility.

The present investigation was performed to determine if right ventricular distension produced by chronic volume and pressure loading could lead to a functional derangement of left ventricular performance, and, if so, to determine if such an alteration was caused by a change in the contractile state of the left ventricle or in its pressure-volume relationships.

Methods

Right ventricular distension was produced in mongrel dogs, weighing between 15 and 23 kg, in a two-staged operative procedure. Initially, a right thoracotomy was performed under thiamylal sodium and halothane anesthesia, and a tricuspid valve leaflet was excised through a right atriotomy during temporary occlusion of venous inflow, to augment volume loading. Two weeks later a left thoracotomy was performed and a pressure load was produced by partial constriction of the pulmonary artery with an umbilical tape. A nonconstricting Teflon gauze was sewn around the ascending aorta to provide support for an intraaortic balloon that was to be introduced during the next procedure so that left ventricular performance might be studied. Ventricular function was studied when the animal had demonstrable ascites; this occurred 2 to 3 weeks following the second operation. Sham-operated dogs were subjected to similar thoracotomies: at the first operation a right atriotomy was performed, but the tricuspid valve was not damaged; at the second operation the pulmonary artery was dissected but not banded, and the ascending aorta was reinforced with Teflon gauze as in the experimental group. The animals with a pressure and volume load on the right ventricle (henceforth referred to as the "stressed" group) developed ascites (500–1500 ml) and reversed the normal relationship of right and left ventricular end-diastolic pressures, right exceeding left by at least 2 mm Hg over the entire range of end-diastolic pressures studied (right ventricular pressure, 5–30 mm Hg; left ventricular pressure, 1–20 mm Hg). In the sham-operated and control animals, left ventricular end-diastolic pressure consistently exceeded the right.

The same protocol was used to study left ventricular function in the stressed, sham-operated, and control dogs. Pentobarbital was administered intravenously until the animal was unconscious. In general, the stressed and sham-operated dogs required less anesthesia (10–20 mg/kg) than the control dogs (30 mg/kg). The animals were mechanically ventilated with a gas mixture of 97% O2 and 3% CO2, and the chest was opened by a midline sternotomy. Central venous, right and left ventricular, and systemic arterial pressures were continuously recorded. Left ventricular pressure was measured by means of a 16-bore metal cannula inserted through the apex of the heart and directly attached to a transducer with zero referred to the level of the left ventricular cavity. The first derivative of left ventricular pressure was obtained with an analog differentiating circuit.

All pressures were measured with Statham P-23Db transducers and were recorded with the electrocardiogram on a multichannel oscillograph at a paper speed of 100 mm/sec. Cardiac output was measured in duplicate by the dye-dilution technique.

A rubber balloon mounted on the tip of a metal cannula was passed through the left carotid artery and positioned in the ascending aorta just above the aortic valve. Isovolumic beats were obtained by rapid inflation of the balloon during diastole with a power injector triggered from the electrocardiogram. Beats were analyzed only if they had features characteristic of isovolumic contractions; i.e., a smooth left ventricular pressure contour, a steady, uninterrupted fall in LV dp/dt following its peak, and obliteration of the aortic systolic pressure wave (fig. 1). All isovolumic beats used for analysis were obtained when ventilation was temporarily stopped. A pressurized reservoir bottle, containing fresh heparinized donor blood from dogs lightly anesthetized with methohexital, was attached to the femoral artery so that blood volume could be increased or decreased, permitting cardiac function to be studied over a wide range of end-diastolic pressures. Arterial blood gases and body

*UD-20 Model, Electronic Gear Corporation, Valley Stream, N. Y. (phase error 1.1° at 16 Hz, 11.4° at 160 Hz).
temperature were monitored and maintained in the physiologic range. Serum electrolytes and hematocrit were also determined intermittently. Three isovolumic beats were obtained at several stable levels of left ventricular end-diastolic pressure ranging from 1 to 20 mm Hg; pressure was varied by infusion of blood in increments of 100–300 ml. The heart was subsequently arrested with potassium chloride, and the ventricles were weighed. The left ventricle was weighed as the intact chamber, including the interventricular septum and free wall, trimmed free of atrial tissue and the aorta. The weight of the right ventricle included that of the free wall only.

Since a large portion of the left ventricular wall of the experimental animal was excised for assay of norepinephrine, passive left ventricular pressure-volume relationships were studied in an additional group of seven stressed and 15 normal animals identically prepared. The left ventricular weights of these animals were similar to those of the dogs employed in the experimental procedure. After potassium arrest the hearts were flushed with saline, and the mitral and tricuspid valves were securely clamped at the atrioventricular groove. The pulmonary artery and aorta were tied at the base of the heart, and both ventricles were emptied. Left ventricular and right ventricular pressures were then recorded as saline was introduced into the left ventricle in 2-ml increments. Simultaneously, saline was introduced into the right ventricle in sufficient quantities so that right ventricular pressures were at levels corresponding to those observed in vivo at each left ventricular end-diastolic pressure. The pressure-volume curve of the left ventricle was also determined when right ventricular pressure corresponded to that present in the normal dogs at each left ventricular end-diastolic pressure. Recovery of infused volume was used as a check for leakage. Curves were performed in duplicate. Final results were normalized to 100 g left ventricular weight. Postmortem wall thickness was measured in the free wall of the right ventricle.

For data analysis, left ventricular pressure and dp/dt of each isovolumic beat were read from the oscillograph records at 10-msec intervals and manually transferred to punch cards. These were processed by digital computer by a program providing peak pressure, peak wall stress, peak dp/dt, and peak contractile element velocity. Force-velocity relations for each beat were graphed directly by a Calcomp plotter. A spherical model for the left ventricle was utilized, and the calculations were similar to those described previously by others. Results for matched intervals were statistically analyzed, when applicable, by Student’s t-test. Norepinephrine concentration was determined as described previously.

**Results**

Right ventricular weight and right-to-left ventricular weight ratios of the stressed animals were greater than those of the sham-operated and normal groups; there were no changes indicative of left ventricular hypertrophy (table 1). A reduction in LV weight was noted which was not statistically significant. Right ventricular wall thickness was increased an average of 0.6 mm, or 25%, in the stressed animals. In addition, right and left ventricular myocardial norepinephrine concentrations were significantly depressed and mean central venous pressure was insignificantly elevated in the stressed group when compared with either the normal or the sham-operated group (table 1). There were no significant differences in the baseline cardiac outputs between the groups (table 1). Serum sodium and potassium as well as hematocrit were not significantly altered in the failure animals. At rest in their cages, heart rate in the stressed animals was 20–30 beats/min faster than in the sham-operated or control dogs. Under the conditions of the experimental

\[
*Tangential wall stress T = \frac{PR_1^2}{R_s^2 - R_t^2}
\]

Circumferential shortening velocity, \(V_{cf} = \frac{Q}{2R_eR_t}\)

Velocity of series elastic extension, \(V_{se} = \frac{(dT/dt)}{(dV/dt)}\)

Midwall contractile element velocity, \(V_{ce} = V_{cf} + V_{se}\)

\(R_e = epidemical ventricular radius (cm)\)

\(R_t = endocardial ventricular radius (cm)\)

\(P = intraventricular pressure (g/cm^2)\)

\(Q = aortic blood flow rate (ml/min) = 0 in isovolumic beats\)

Equations are based on a spherical model of uniform wall thickness for the left ventricle and are derived specifically for a ring of muscle at the midwall of a cross-sectional area of 1 cm². Since \(Q = 0\) in isovolumic beats, \(V_{cf}\) also = 0, and the expression for \(V_{ce}\) reduces to \(V_{ce} = V_{se}\).

\(dV/dt\) is the series elastic constant, numerically equal to 30 T. Thus, the expression for contractile element velocity becomes

\[V_{ce} = \frac{dT/dt}{30T}\]
diastolic pressure were compared, left ventricular performance appeared to be appreciably depressed (table 2, fig. 2).

**Pressure-Volume Relationships**

Figure 3 illustrates the effect of elevated right ventricular filling pressures on left ventricular pressure-volume relations in the stressed animals. When the right ventricle of the stressed dogs was filled so that pressure was relatively low (equivalent to the right ventricular end-diastolic pressure observed in the normal dogs) the pressure-volume curve of the left ventricle of these dogs was not appreciably different from that of normal dogs. The pressure-volume curve was shifted markedly to the left, however, when the right ventricle was filled to the high levels that were present in vivo. Thus, for any left ventricular end-diastolic filling pressure, left ventricular volume was considerably less when the right ventricle was filled to pressures observed in vivo in the flow and pressure loaded right heart, than when it was only filled to pressures observed in the normal dog. Statistical significance of the observed alteration in pressure-volume relations produced by changing the degree of right ventricular filling in the stressed animals was confirmed by performing paired t-tests at pressures of 4, 8, 12, 16, and 20 mm Hg. P values were less than 0.05 at each level tested.

**Discussion**

The results of this investigation demonstrate that the imposition of a chronic flow and pressure stress on the right ventricle leads to a derangement of left ventricular performance. This was manifested by a diminution of left ventricular peak systolic pressure, peak dp/dt, and calculated peak systolic wall stress developed during an isovolumic contraction under a wide range of left ventricular end-diastolic pressures.

Interpretation of these changes as they relate to myocardial contractility is complicated, however, by the altered left ventricular pressure-volume relationship caused by the chronic right ventricular flow and pressure stress. During determination of the left

**Figure 1**

Pressure recordings before and during an isovolumic beat in a dog with a chronic right ventricular flow and pressure load. LVEDP = left ventricular pressure, high sensitivity; RVEDP = right ventricular pressure, high sensitivity; RVP = right ventricular pressure, low sensitivity; ECG = electrocardiogram; LVP = left ventricular pressure, low sensitivity; AoP = aortic pressure; LV dp/dt = first derivative of left ventricular pressure.

As in all stressed animals, RVEDP is higher than LVEDP. Induction of an isovolumic beat is characterized by elimination of the aortic pressure pulse, marked augmentation of LV systolic pressure, and a smooth and prolonged downslope of LV dp/dt.

procedure, however, heart rates of control and stressed animals were nearly identical; the sham-operated animals had slightly elevated heart rates, but the difference was not statistically significant. Heart rate decreased and mean arterial pressure increased in all animals as end-diastolic pressure was increased by infusion of blood.

**Isovolumic Beats**

When left ventricular peak systolic pressures, peak systolic wall stress, peak dp/dt, and peak contractile element velocity (V Council) at four matched levels of left ventricular end-
Table 1

Cardiac Effects of Chronic Right Ventricular Volume and Pressure Loading

<table>
<thead>
<tr>
<th></th>
<th>No. dogs</th>
<th>Cardiac output (liters/min)</th>
<th>Mean central venous pressure (mm Hg)</th>
<th>Norepinephrine (μg/g)</th>
<th>Heart weight (g)</th>
<th>RV/LV (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>RV</td>
<td>LV</td>
</tr>
<tr>
<td>Normal</td>
<td>6</td>
<td>2.1 ± 0.3</td>
<td>1.0 ± 0.5</td>
<td>0.68 ± 0.09</td>
<td>0.74 ± 0.14</td>
<td>39.1 ± 3.2</td>
</tr>
<tr>
<td>Sham</td>
<td>8</td>
<td>1.9 ± 0.2</td>
<td>1.6 ± 0.4</td>
<td>0.42 ± 0.04</td>
<td>0.58 ± 0.09</td>
<td>37.7 ± 1.8</td>
</tr>
<tr>
<td>P*</td>
<td>NS</td>
<td>NS</td>
<td>&lt;0.05</td>
<td>&lt;0.05</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>RV stressed</td>
<td>8</td>
<td>1.7 ± 0.2</td>
<td>11.3 ± 1.4</td>
<td>0.06 ± 0.05</td>
<td>0.16 ± 0.07</td>
<td>44.0 ± 2.6</td>
</tr>
<tr>
<td>P†</td>
<td>NS</td>
<td>NS</td>
<td>&lt;0.01</td>
<td>&lt;0.001</td>
<td>&lt;0.05</td>
<td>NS</td>
</tr>
</tbody>
</table>

P* = probability that difference between mean values for normal and sham groups is due to chance alone.
P† = probability that difference between mean values for normal and RV stressed groups is due to chance alone.
NS = not significant; RV = right ventricle; LV = left ventricle.

Table 2

Effects of Right Ventricular Volume and Pressure Loading on Left Ventricular Function

<table>
<thead>
<tr>
<th></th>
<th>Left ventricular end-diastolic pressure (mm Hg)</th>
<th>Peak dp/dt (mm Hg/sec)</th>
<th>Peak pressure (mm Hg)</th>
<th>Peak V_Cr (cm/sec)</th>
<th>Peak wall stress (g/cm²)</th>
<th>Heart rate (beats/min)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>3.5 ± 0.3</td>
<td>2126 ± 83</td>
<td>133 ± 4</td>
<td>36.5 ± 2.9</td>
<td>75 ± 3</td>
<td>158 ± 6</td>
</tr>
<tr>
<td>Sham</td>
<td>3.5 ± 0.3</td>
<td>2345 ± 140</td>
<td>142 ± 7</td>
<td>40.8 ± 3.2</td>
<td>81 ± 4</td>
<td>171 ± 6</td>
</tr>
<tr>
<td>RV stressed</td>
<td>3.4 ± 0.2</td>
<td>1741 ± 86</td>
<td>115 ± 3</td>
<td>25.1 ± 1.8</td>
<td>49 ± 3</td>
<td>150 ± 2</td>
</tr>
<tr>
<td>Normal</td>
<td>7.2 ± 0.2</td>
<td>2683 ± 110</td>
<td>185 ± 5</td>
<td>41.8 ± 3.3</td>
<td>177 ± 8</td>
<td>143 ± 6</td>
</tr>
<tr>
<td>Sham</td>
<td>7.2 ± 0.2</td>
<td>3210 ± 167*</td>
<td>210 ± 29</td>
<td>40.6 ± 3.6</td>
<td>191 ± 8</td>
<td>161 ± 6</td>
</tr>
<tr>
<td>RV stressed</td>
<td>6.3 ± 0.5</td>
<td>2129 ± 80</td>
<td>149 ± 4</td>
<td>27.7 ± 2.0</td>
<td>116 ± 4</td>
<td>139 ± 2</td>
</tr>
<tr>
<td>Normal</td>
<td>12.4 ± 0.3</td>
<td>2963 ± 128</td>
<td>221 ± 6</td>
<td>39.8 ± 2.7</td>
<td>283 ± 6</td>
<td>132 ± 3</td>
</tr>
<tr>
<td>Sham</td>
<td>12.0 ± 0.3</td>
<td>3215 ± 129</td>
<td>224 ± 4</td>
<td>27.2 ± 2.3</td>
<td>284 ± 6</td>
<td>154 ± 7</td>
</tr>
<tr>
<td>RV stressed</td>
<td>12.0 ± 0.3</td>
<td>2416 ± 119</td>
<td>177 ± 4</td>
<td>24.0 ± 2.3</td>
<td>198 ± 6</td>
<td>137 ± 10</td>
</tr>
<tr>
<td>Normal</td>
<td>16.9 ± 0.6</td>
<td>3313 ± 184</td>
<td>236 ± 8</td>
<td>45.5 ± 2.9</td>
<td>354 ± 10</td>
<td>129 ± 3</td>
</tr>
<tr>
<td>Sham</td>
<td>13.7 ± 0.2</td>
<td>3036 ± 127</td>
<td>225 ± 2</td>
<td>43.2 ± 2.5</td>
<td>340 ± 8</td>
<td>134 ± 3</td>
</tr>
<tr>
<td>RV stressed</td>
<td>16.1 ± 0.3</td>
<td>2425 ± 111</td>
<td>193 ± 5</td>
<td>25.4 ± 2.5</td>
<td>244 ± 7</td>
<td>129 ± 6</td>
</tr>
</tbody>
</table>

Hemodynamic findings in left ventricular isovolumic beats of six normal dogs, eight sham-operated dogs, and eight RV stressed dogs with standard errors. Peak V_Cr = peak contractile element velocity.
P values: When normal are compared to sham values, peak pressure, peak V_Cr, peak wall stress, and peak dp/dt are not significantly different except at * where P < 0.05. When normal are compared to RV stressed values, peak pressure, peak V_Cr, peak wall stress, and peak dp/dt are all significantly different (P < 0.01).
ventricular passive pressure-volume curve in dogs subjected to such a chronic right ventricular stress, the right ventricle was filled so that right ventricular pressure corresponded to values measured at each left ventricular end-diastolic pressure used during the in vivo assessment of left ventricular performance. It was found that under these conditions the normal left ventricular passive pressure-volume curve was displaced to the left toward smaller volumes (fig. 3). A similar alteration in the pressure-volume characteristics of the left ventricle was shown to occur in normal canine hearts consequent to increases in right ventricular filling. These results indicate that the differences in left ventricular performance between the stressed and control animals must, at least in part, be due to a change in the pressure-volume relationship of the left ventricle. Although the pressure-volume curve of the left ventricle shifted to the left when the right ventricle was filled to match the in vivo conditions, it is interesting to note that the left ventricular pressure-volume curve was normal when the right ventricle was empty. This finding demonstrates that the intrinsic compliance of the left ventricle was not altered and that the changes observed in the pressure-volume curve of the left ventricle were due solely to the effects of changes in right ventricular volume.

Whether or not an alteration in myocardial contractility contributes to the derangement in left ventricular performance could best be tested by comparing parameters of contractile function in control and stressed animals at matched end-diastolic volumes rather than end-diastolic pressures. Unfortunately, a large portion of the left ventricular wall was excised for norepinephrine determinations in those dogs in which left ventricular performance was assessed. Pressure-volume curves therefore were not determined in these animals, since their reliability would have been suspect. Nevertheless, a qualitative appreciation of how this change in the pressure-volume relationship could influence the assessment of left ventricular function is seen in fig. 4. Calculations of volumes were made from the averaged pressure-volume curves of the group of normal and stressed animals depicted in figure 3; these animals were prepared in an identical manner as the original animals in which left ventricular performance was assessed.

Mean results and standard error of peak pressure, wall stress, and peak dp/dt plotted against left ventricular end-diastolic pressure as obtained during isovolumic beats in normal and RV stressed dogs.

**Figure 2**
Mean left ventricular pressure volume curves ± standard errors normalized to 100 g left ventricular weight. The dashed line shows the mean values in 15 normal dogs when pressures in right and left ventricles were kept at the end-diastolic pressures normally observed in vivo. Open circles represent the mean pressure-volume curve of seven RV flow and pressure stressed dogs when RV pressure was maintained at the low level found in vivo in the normal animals. Closed circles represent the mean pressure-volume curve of the seven RV flow and pressure stressed dogs when RV pressure was at the high level found at end-diastole in vivo in the stressed animals.

Systolic wall stress are related to left ventricular end-diastolic volume rather than pressure, the differences between the stressed and normal dogs are greatly diminished. In fact, the curves depicting peak systolic pressure and wall stress are virtually superimposable, although a small difference in peak dp/dt and depression of myocardial force velocity curves remain (fig. 5). The precise quantitative significance of these findings is uncertain in view of the errors introduced by estimation of ventricular volume from averaged pressure-volume curves, and by determination of the pressure-volume relation under the necessarily altered circumstances present in the postmortem state.\textsuperscript{11,12} In addition, unusual mechanical forces exist when the right ventricle is sufficiently distended so that right ventricular pressure exceeds that of the left ventricle at end-diastole, but is lower during systole. The geometric changes that may accompany such stresses could limit the reliability of the usual indices of contractility even when comparisons are made at matched end-diastolic volumes. These considerations serve to emphasize the complex interrelationship that exists between the right and left ventricles, and they demonstrate the difficulties inherent in attempts to determine whether or not a derangement in left ventricular performance occurring during right ventricular dilatation or failure is due to a true impairment in left ventricular contractility or to a mechanical effect caused by the close anatomic relation of the two ventricles.
The possible influence that alterations in sympathetic stimulation to the heart would exert on interpretation of the present findings must also be considered. Since arterial pressure was always somewhat lower in the dogs subjected to chronic right ventricular stress than in the normal animals, any resulting increase in sympathetic activity due to baroreceptor-mediated reflexes would tend to minimize the differences in myocardial contractility between the two groups. Similarly, the increased levels of circulating catecholamines present in heart failure would also tend to minimize any impairment in intrinsic myocardial contractility present in the right ventricular-stressed animals. Although disparities in the relative amounts of sympathetic stimulation between the normal and right ventricular-stressed animals could have been eliminated by the administration of a beta-blocking agent, this was deemed inadvisable because of the deterioration and instability of animals subjected to a chronic flow and pressure stress.

It is also possible that barbiturate anesthesia might have produced dissimilar effects on myocardial contractility in the two groups. Although we could have obviated this possibility by studying unanesthetized dogs, it then would have been impossible to rapidly expand blood volume in order to measure and compare parameters of left ventricular function under a wide variety of end-diastolic pressures.

It should be emphasized that the results of this investigation were obtained under very specific experimental conditions. The right ventricle was stressed by both a flow load (imposed by the creation of tricuspid insufficiency) and a pressure load (imposed by the creation of pulmonary stenosis). This combination of stresses resulted in ascites, right ventricular hypertrophy, elevated right ventricular end-diastolic pressures, and a reduction of myocardial norepinephrine concentrations. Although these changes could be interpreted as indicating right ventricular failure, it seems possible that such changes might occur in the volume-overloaded ventricle without there necessarily being any impairment in its intrinsic contractile state. Thus, under the conditions of the experimental design used in this investigation, it is not certain whether the alteration in left ventricular function is the result of right ventricular failure, an excessive right ventricular volume load, or a combination of the two.
The hypothesis that hypertrophy and dilatation of one ventricle could affect the performance characteristic of the other was first suggested by Bernheim in 1910. He postulated that left ventricular dilatation could result in elevated right heart pressures and venous congestion by causing the septum to shift to the right, thereby compressing the outflow tract of the right ventricle. More recently, Dexter suggested that a similar mechanism might be responsible for the signs and symptoms of left ventricular failure occasionally seen in a patient with an atrial septal defect; he postulated that the increased filling pressure of the distended right ventricle might shift the ventricular septum so as to compress the left ventricle. Results of the present investigation are compatible with this concept and suggest that a "reversed" Bernheim effect may occur when the right ventricle is subjected to a volume and pressure stress. It is therefore suggested that such a mechanism be considered in patients who demonstrate evidence of left ventricular failure under circumstances that would be expected to produce isolated impairment of right ventricular function.

In summary, we have found that at matched levels of end-diastolic pressure, peak dp/dt, peak pressure, peak wall stress, and peak $V_{CE}$ of the left ventricle are depressed in dogs in which the right ventricle has been...
subjected to a chronic flow and pressure load. When the right ventricle of such animals is filled to end-diastolic pressures observed in vivo, the pressure-volume relationship of the left ventricle changes markedly so that there is a decreased volume present at any level of end-diastolic pressure. When isovolumic beat data matched for estimated end-diastolic volume rather than for end-diastolic pressure are compared, the peak pressures and wall stresses achieved by the animals subjected to chronic pressure and flow loads are similar to those found in the control groups, although peak dp/dt and peak VCE appear to remain somewhat depressed. These results indicate that when the right ventricle is subjected to a chronic pressure and flow load, the pressure-volume relationship of the left ventricle is altered such that the assessment of left ventricular function may be unreliable when indices of contractility are employed which require measurements of end-diastolic pressure.

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