

## **Right ventricular function in an operating room model of mechanical left ventricular assistance and its effects in patients with depressed left ventricular function**

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**ABSTRACT** Approximately 20% of patients who receive left ventricular assist devices (LVADs) for refractory cardiac failure after open heart surgery have had complications of right ventricular failure. To evaluate this problem in the diseased heart we simulated an LVAD in the operating room by bypassing and unloading the left ventricle with the heart-lung machine before routine open heart surgery. Right ventricular function was assessed in 12 patients with preoperative left ventricular ejection fractions of less than 0.55 (poor left ventricular function) ( $\text{mean} \pm \text{SEM } 0.40 \pm 0.03$ ) and 10 patients with ejection fractions greater than 0.55 (normal left ventricular function) ( $0.63 \pm 0.02$ ). Measurements before and during left ventricular bypass in the normal left ventricular function group revealed no change in cardiac output (from  $5.7 \pm 0.6$  to  $5.8 \pm 0.4$  liters/min), with a decrease in right ventricular end-diastolic pressure (from  $8 \pm 2$  to  $6 \pm 1$  mm Hg). However, in the poor left ventricular function group, cardiac output was increased significantly during left ventricular bypass from  $4.5 \pm 0.2$  to  $5.3 \pm 0.4$  liters/min and right ventricular end-diastolic pressure was decreased significantly from  $13 \pm 2$  to  $8 \pm 2$  mm Hg. During bypass there were significant reductions in mean pulmonary arterial pressure from  $17 \pm 3$  to  $10 \pm 2$  mm Hg in the normal left ventricular function group and from  $27 \pm 3$  to  $12 \pm 2$  mm Hg in the poor left ventricular function group. These measurements reflect passive changes in pulmonary pressures due to reductions in left ventricular filling pressure during left ventricular bypass. The findings show that acute left ventricular unloading results in unchanged to slightly improved right ventricular function in the normal left ventricular function group and in significantly improved right ventricular function in the poor left ventricular function group, principally due to right ventricular afterload reduction. This demonstrates a potential beneficial effect to the right ventricle in patients with pulmonary venous hypertension secondary to poor left ventricular function. The data suggest that acute unloading of the left ventricle is not the cause of right ventricular failure in patients with LVADs, and the pathophysiology of other causes must be investigated.  
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**PREVIOUS CLINICAL EXPERIENCE** with left ventricular assist devices (LVADs) used for refractory cardiac failure after open heart surgery has demonstrated right heart failure in a significant fraction of patients.<sup>1</sup> Of 213 patients who received LVADs at 12 different centers, 49 patients either died of right ventricular failure or required biventricular mechanical

support.<sup>1</sup> Reports from many of these centers discussed right ventricular failure as a potential limitation to LVAD therapy,<sup>2-11</sup> but others reported adequate management of right ventricular function with isoproterenol<sup>12</sup> or did not mention right ventricular failure as a major problem during left ventricular assistance.<sup>13-15</sup>

In addition, our experimental studies of left ventricular bypass and unloading in the normal dog heart<sup>16, 17</sup> and findings of a preliminary clinical study<sup>18</sup> have provided no evidence of right ventricular failure. Although patient survival during mechanical support of only one ventricle is obviously dependent on the severity of biventricular dysfunction,<sup>8</sup> the problem is not clearly understood in terms of anatomic and hemody-

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namic ventricular interaction or in terms of patient selection and patient management. The purpose of the present study is to extend the experimental evaluation of the effects of left ventricular bypass on right ventricular function to the diseased human heart by studying patients during routine open heart surgery.

## Methods

Patients for study during left ventricular bypass were selected from the pool of patients referred to the Department of Cardiovascular Surgery at Pacific Presbyterian Medical Center for aortocoronary bypass grafting, aneurysmectomy, and valve replacement surgery. Studies were performed with the signed informed consent of each patient according to a protocol approved by the hospital's Institutional Review Board. Patients (table 1) were divided into the two following groups based on preoperative cineangiographic left ventricular ejection fractions (LVEFs) determined during cardiac catheterization: the normal left ventricular function group (LVEF  $\geq 0.55$ , 10 patients) and the poor left ventricular function group (LVEF  $< 0.55$ , 12 patients). None of the patients had severe pulmonary hypertension preoperatively. All studies were carried out in the operating room immediately before placing the patient on full cardiopulmonary bypass during open heart surgery. The study added 15 to 20 min to the operation and about 10 min to pump time.

**TABLE 1**  
**Patient characteristics**

Patient	Sex	Age (yr)	Operation	Weight (kg)	BSA (m <sup>2</sup> )	LVEF (%)
Normal LV function group (LVEF $\geq 55\%$ )						
R. H.	M	46	ACB (3)	89	2.14	60
D. P.	M	73	ACB (2)	77	1.98	70
W. A.	M	55	ACB (4)	84	2.06	59
D. A.	M	53	ACB (1)	81	2.03	67
J. H.	M	38	ACB (3)	102	2.18	58
I. C.	M	73	ACB (3)	80	2.00	80
T. R.	M	46	ACB (3)	68	1.76	60
P. N.	M	53	ACB (4)	66	1.67	59
A. P.	M	61	ACB (1)	89	2.16	58
O. M.	M	63	ACB (5)	59	1.66	56
Mean $\pm$ SEM		56 $\pm$ 4		80 $\pm$ 4	1.96 $\pm$ 0.06	63 $\pm$ 2
Poor LV function group (LVEF $< 55\%$ )						
W. B.	M	63	AVR	74	1.87	51
D. S.	M	58	AVR	62	1.70	41
E. C.	M	81	LVA	82	1.95	23
C. W.	M	60	ACB (3)	74	1.94	38
J. D.	M	61	ACB (2)	78	1.89	46
E. T.	M	58	ACB (2)	89	2.10	51
S. D.	M	54	ACB (3)	71	1.76	25
J. F.	M	74	ACB (5)	65	1.76	36
W. K.	M	67	ACB (2)	72	1.89	46
R. S.	M	59	ACB (4)	64	1.70	34
A. M.	M	63	ACB (2)	78	1.87	51
D. M.	F	78	ACB (3)	45	1.44	32
Mean $\pm$ SEM		65 $\pm$ 3		71 $\pm$ 3	1.82 $\pm$ 0.05	40 $\pm$ 3

LV = left ventricular; BSA = body surface area; LVEF = preoperative cineangiographic ejection fraction; ACB = aortocoronary bypass (plus number of vessels bypassed); AVR = aortic valve replacement; LVA = left ventricular aneurysmectomy.

A median sternotomy was performed and the pericardium was opened. Venous blood was returned to the heart-lung machine by No. 40F wire-reinforced Bardic cannulas placed in the vena cavae. Arterial blood was returned to the systemic circulation with a No. 28F wire-reinforced cannula introduced into the ascending aorta. The left side of the heart was unloaded via a No. 32F curved wire-reinforced Bardic cannula introduced into the right superior pulmonary vein, through the left atrium, and across the mitral valve into the left ventricle. To obtain better flow the left ventricular vent cannula that was used was larger than the usual No. 24F to 28F size routinely used at this center. Arterial pressure was measured from the radial or femoral artery. A special triple-lumen Swan-Ganz catheter with a proximal port at 13 cm (Edwards Labs) was used to measure pulmonary arterial and right ventricular pressures and thermodilution cardiac output. Left ventricular pressure was measured through a catheter inserted into the left ventricular chamber via the left ventricular vent cannula. This measurement also confirmed adequate placement of the cannula in the ventricle.

Stroke volume was calculated as cardiac output divided by heart rate. Stroke work index was calculated as stroke volume times right ventricular developed pressure divided by body surface area. Systemic vascular resistance index was calculated as mean aortic pressure minus right ventricular end-diastolic pressure (in mm Hg) times 80 (to convert to dynes  $\cdot$  sec  $\cdot$  m<sup>2</sup>/cm<sup>5</sup>) divided by cardiac index. Pulmonary arterial input resistance index was calculated as mean pulmonary arterial pressure times 80 divided by cardiac index. Pulmonary vascular resistance was not calculated because left ventricular filling pressures during the suction effect of left ventricular bypass are not representative of changes in the pulmonary vasculature. All data were recorded on a Gould 2800 eight-channel recorder and analyzed on a PDP 11/23 computer.

At the beginning of the operative procedure, all electronic recording equipment was allowed to warm up; pressure transducers were zeroed and balanced to atmosphere and calibrated with a mercury manometer. During the experimental procedures the right ventricle was not manipulated, and measurements of right ventricular function were made before (control) and during left ventricular bypass. Control measurements were made during three to five thermodilution injections of iced 5% dextrose solution, and each value for each subject was calculated from the average of these findings.

To simulate the unloading effects of an LVAD, left ventricular bypass was initiated by opening the left ventricular vent to the heart-lung machine without opening the venous drainage system from the right atrium. The goal was to use this bypass circuit to pump blood from the left ventricle to the aorta to reduce left ventricular pressure as much as possible. No complications occurred as a result of this procedure. If any patient had become unstable, full bypass would have been initiated immediately. Once pressures had stabilized, measurements of ventricular function were again made during three to five thermodilution injections. Data were averaged by patient group, and statistical tests comparing control with left ventricular bypass for each value were performed with a paired *t* test. Comparisons between patient groups under control or left ventricular bypass conditions were performed by the Student *t* test.

## Results

Under control conditions, stroke volume and cardiac output were significantly lower in the poor left ventricular function group than in the normal left ventricular function group, and right ventricular end-diastolic pressure, mean pulmonary arterial pressure, and pul-

TABLE 2

Summary of average values ( $\pm$  SEM) for each parameter before (control) and after left ventricular (LV) bypass

	Normal LV function group			Poor LV function group		
	Control	LV bypass	% Change	Control	LV bypass	% Change
SV (ml)	77 $\pm$ 6 (10)	71 $\pm$ 5 (10)	-3 $\pm$ 10	58 $\pm$ 3 <sup>D</sup> (12)	64 $\pm$ 6 (12)	12 $\pm$ 10
CO (l/min)	5.7 $\pm$ 0.6 (10)	5.8 $\pm$ 0.4 (10)	9 $\pm$ 11	4.5 $\pm$ 0.2 <sup>C</sup> (12)	5.3 $\pm$ 0.4 <sup>A</sup> (12)	19 $\pm$ 8
CI (l·m <sup>2</sup> /min)	2.9 $\pm$ 0.2 (10)	3.0 $\pm$ 0.2 (10)	9 $\pm$ 10	2.5 $\pm$ 0.1 (12)	2.9 $\pm$ 0.2 (12)	18 $\pm$ 8
SWI (J/m <sup>2</sup> )	0.128 $\pm$ 0.013 (10)	0.110 $\pm$ 0.016 (10)	-13 $\pm$ 9	0.129 $\pm$ 0.021 (12)	0.097 $\pm$ 0.020 <sup>A</sup> (12)	-22 $\pm$ 10
HR (beats/min)	74 $\pm$ 4 (10)	84 $\pm$ 3 <sup>A</sup> (10)	16 $\pm$ 5	78 $\pm$ 3 (12)	86 $\pm$ 5 (12)	10 $\pm$ 6
RVEDP (mm Hg)	8 $\pm$ 2 (10)	6 $\pm$ 1 <sup>A</sup> (10)	-18 $\pm$ 7	13 $\pm$ 2 <sup>C</sup> (12)	8 $\pm$ 2 <sup>A</sup> (12)	-37 $\pm$ 10
RVPSP (mm Hg)	32 $\pm$ 3 (10)	27 $\pm$ 2 (10)	-15 $\pm$ 10	42 $\pm$ 4 (12)	27 $\pm$ 3 <sup>B</sup> (12)	-34 $\pm$ 5
PAP (mm Hg)	17 $\pm$ 3 (9)	10 $\pm$ 2 <sup>A</sup> (9)	-36 $\pm$ 8	27 $\pm$ 3 <sup>C</sup> (12)	12 $\pm$ 2 <sup>B</sup> (12)	-53 $\pm$ 5
LVEDP (mm Hg)	15 $\pm$ 3 (10)	4 $\pm$ 3 <sup>A</sup> (9)	-59 $\pm$ 22	20 $\pm$ 3 (11)	0 $\pm$ 3 <sup>B</sup> (12)	-107 $\pm$ 29
LVPSP (mm Hg)	108 $\pm$ 6 (10)	56 $\pm$ 11 <sup>B</sup> (10)	-45 $\pm$ 11	101 $\pm$ 6 (12)	27 $\pm$ 9 <sup>B,C</sup> (12)	-79 $\pm$ 8
AOP (mm Hg)	81 $\pm$ 4 (9)	68 $\pm$ 3 <sup>B</sup> (9)	-15 $\pm$ 2	80 $\pm$ 2 (12)	76 $\pm$ 4 (12)	-4 $\pm$ 4
RinI (dynes·sec·m <sup>2</sup> /cm <sup>5</sup> )	532 $\pm$ 97 (9)	281 $\pm$ 39 <sup>A</sup> (9)	-33 $\pm$ 15	885 $\pm$ 105 <sup>C</sup> (12)	354 $\pm$ 50 <sup>B</sup> (12)	-57 $\pm$ 6
SVRI (dynes·sec·m <sup>2</sup> /cm <sup>5</sup> )	2160 $\pm$ 260 (9)	1804 $\pm$ 206 (9)	-11 $\pm$ 11	2287 $\pm$ 124 (12)	2033 $\pm$ 160 (12)	-10 $\pm$ 8

The number of patients used to calculate each mean is shown in parentheses.

SV = stroke volume; CO = cardiac output; CI = cardiac index; SWI = stroke work index; HR = heart rate; RVEDP and RVPSP = right ventricular end-diastolic and peak systolic pressures; PAP = mean pulmonary arterial pressure; LVEDP and LVPSP = left ventricular end-diastolic and peak systolic pressures; RinI = pulmonary vascular input resistance index; SVRI = systemic vascular resistance index.

<sup>A</sup>p < .05; <sup>B</sup>p < .01 compared with control, <sup>C</sup>p < .05; <sup>D</sup>p < .01 compared with normal group.

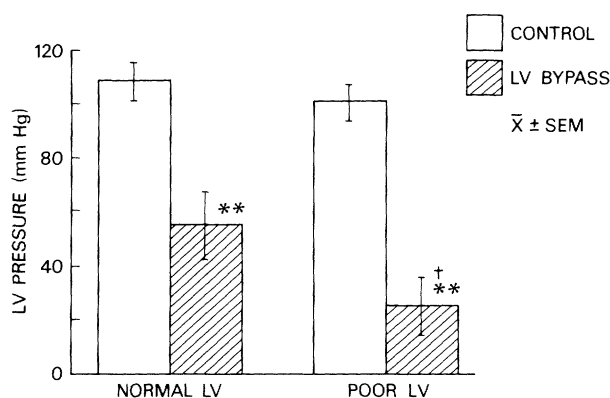
monary arterial input resistance were higher (table 2). Left ventricular bypass produced significant reductions in left ventricular peak systolic and end-diastolic pressures for both groups, and left ventricular peak systolic pressure was reduced to a lower level in the poor left ventricular function group than in the normal group (figure 1, table 2). There was no change in cardiac output during left ventricular bypass in those patients with normal left ventricular function, but output was increased, with a drop in right ventricular end-diastolic pressure, in those with poor left ventricular function (figure 2, table 2). The relationship between cardiac output and right ventricular end-diastolic pressure (figure 3) shows a slight shift to the left during left ventricular bypass in the normal left ventricular function group but a significant shift upward and to the left in poor function group.

Right ventricular end-diastolic pressure, mean pul-

monary arterial pressure (figure 4), and pulmonary arterial input resistance index were all significantly reduced in both groups during left ventricular bypass (table 2). There was a significant increase in heart rate and a decrease in mean aortic pressure during bypass only in the normal left ventricular function group, and there was no change in systemic vascular resistance index.

## Discussion

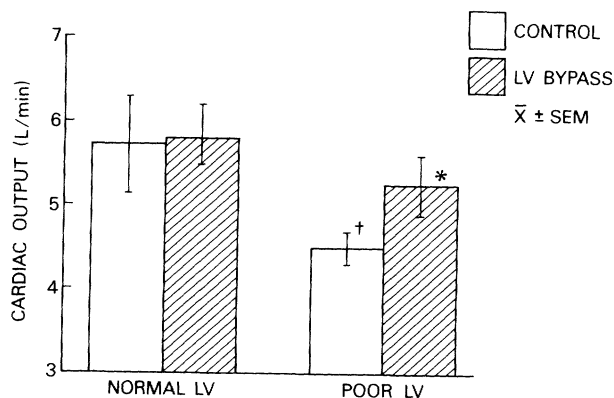
This study indicates that left ventricular assistance in patients with normal left ventricular function produces no change in right ventricular function in terms of cardiac output, but does result in a beneficial reduction in right ventricular filling pressure. In patients with poor left ventricular function, left ventricular assistance results in improved right ventricular function, as demonstrated by an increase in cardiac output and a



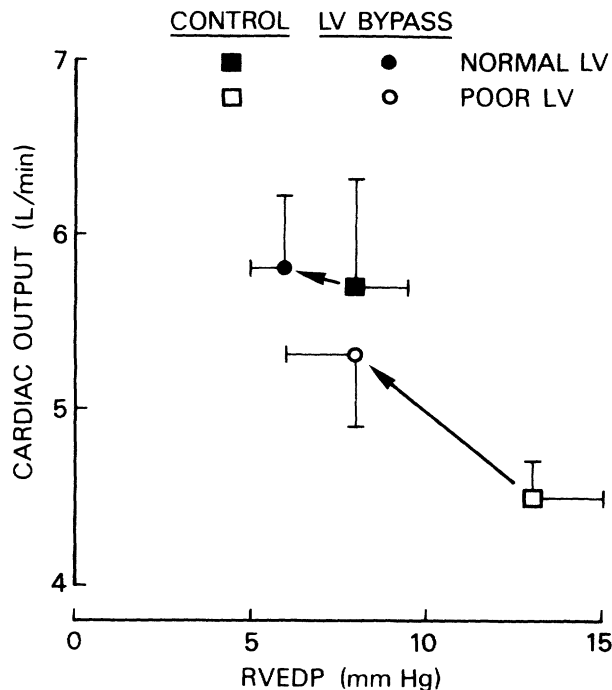
**FIGURE 1.** Left ventricular (LV) peak systolic pressure was significantly (\*\* $p < .01$ ) reduced from control during LV bypass in patients with normal (normal LV) and in those with poor (poor LV) preoperative LVEFs. During LV bypass LV peak systolic pressure was significantly ( $\dagger p < .05$ ) lower in the poor LV group than in the normal LV group.

lower filling pressure. Left ventricular assistance in this acute operating room model produces no evidence of right ventricular failure in typical surgical patients with heart disease, and the data suggest that in patients with elevated pulmonary pressures secondary to poor left ventricular function it may offer a beneficial effect to the right heart.

The beneficial effect to the right ventricle during left ventricular bypass is primarily in the form of reduced pulmonary arterial pressure resulting from a reduction in left ventricular filling pressure. In those patients with poor left ventricular function, right ventricular peak systolic pressure and mean pulmonary arterial pressure were reduced 15 mm Hg during bypass, which paralleled the 20 mm Hg drop in left ventricular end-diastolic pressure. In the normal left ventricular function group, mean pulmonary arterial pressures fell 7 mm Hg during an 11 mm Hg drop in left ventricular

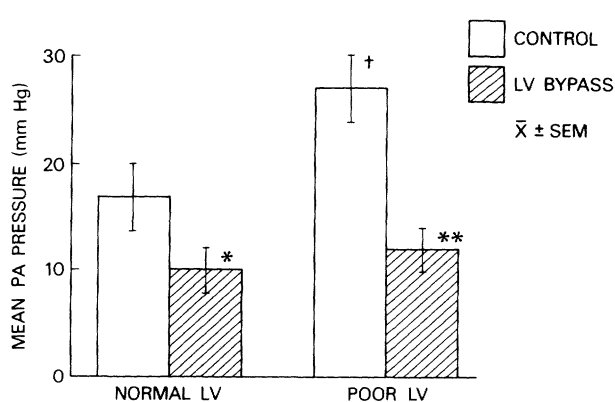


**FIGURE 2.** There was no change in cardiac output from control to during left ventricular (LV) bypass in the normal LV group, but it was significantly (\* $p < .05$ ) increased in the poor LV group. Under control conditions cardiac output was significantly ( $\dagger p < .05$ ) lower in the poor LV group than in the normal group.



**FIGURE 3.** The relationship between cardiac output and right ventricular end-diastolic pressure (RVEDP) revealed a slight shift to the left from control to during left ventricular (LV) bypass in the normal LV group, and demonstrated a significant shift upward and to the left in the poor LV group.

end-diastolic pressure. These findings in human subjects compare with a 3 to 7 mm Hg decrease in mean pulmonary arterial pressure in normal dog hearts during a 5 to 16 mm Hg reduction in left ventricular end-diastolic pressure.<sup>16</sup> The decrease in right ventricular afterload produced by left ventricular bypass is more accurately described by a change in pulmonary arterial input resistance index, which is a measure of the resistive portion of the hydraulic afterload of the right ventricle. In the poor left ventricular function group input



**FIGURE 4.** Mean pulmonary arterial (PA) pressure was significantly reduced from control to during left ventricular (LV) bypass in the normal LV group (\* $p < .05$ ) and the poor LV group (\*\* $p < .01$ ). Under control conditions mean PA pressure was significantly ( $\dagger p < .05$ ) greater in the poor LV group than in the normal LV group.

resistance index fell 57%, while in the normal left ventricular function group it decreased 33%; in studies on normal dog hearts input resistance decreased approximately 18%.

The comparison between the two patient groups plus the results from previous animal studies illustrate that the greater the depression of left ventricular function and the associated increase in pulmonary venous and arterial pressures, the greater the effect left ventricular bypass can have on reducing right ventricular afterload. Because of the linear relationship between pulmonary arterial and left atrial pressures in patients without pulmonary vascular obstructive disease,<sup>19</sup> an LVAD should be capable of reducing elevated pulmonary venous and arterial pressures back toward normal levels. However, in the presence of pulmonary vascular obstruction, right heart pressures are in excess of this relationship,<sup>19, 20</sup> and left ventricular assistance may have an opposite and detrimental effect on right ventricular outflow pressure due to augmented flow through the elevated pulmonary vascular resistance.<sup>1</sup> This is apparently what happened in a number of patients with LVADs reported by Pennington et al.,<sup>8</sup> in whom pulmonary vascular resistance increased in the first 12 postoperative hours, probably because of microemboli from multiple blood transfusions.

The most common cause of right ventricular failure is the increase in right ventricular afterload resulting from left ventricular failure.<sup>20</sup> Although at least one study has shown normal right ventricular performance in patients with mitral stenosis and moderate pulmonary hypertension,<sup>21</sup> most studies show the right ventricle to be markedly afterload dependent, with right ventricular ejection fraction being lower in patients with elevated pulmonary arterial pressure than in normal subjects.<sup>22–24</sup> In a preliminary transesophageal echocardiographic study in humans, we have shown right ventricular cross-sectional area to decrease by 29% in diastole and by 54% in systole, and fractional area change to increase from 42% to 63%, in response to a 19 mm Hg decrease in pulmonary arterial pressure during left heart bypass.<sup>18</sup> Similar results were also found in a patient supported with an LVAD for 10 days.<sup>18</sup> These decreases in right ventricular dimensions during left ventricular bypass are consistent with right ventricular afterload dependence, but appear to conflict with results of studies using ultrasonic crystals in dogs indicating an increase in right ventricular free wall-to-septum dimension with no change in fractional shortening during left ventricular unloading.<sup>17</sup> However, the differences can be explained by the fact that the large reduction in pulmonary arterial pressure was the

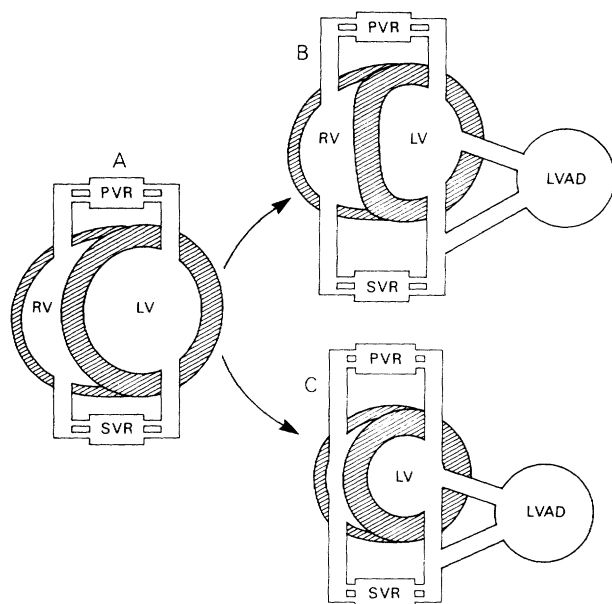
dominant factor in the clinical studies and that the leftward septal shift was the dominant factor in the animal study, in which a much smaller change in pulmonary arterial pressure was noted.

Hypotheses based on studies of ventricular interdependence can explain at least three changes during left ventricular bypass that can affect the preload, afterload, and contractility of the right ventricle.<sup>1</sup> First, a translocation of blood volume from the pulmonary venous circulation to the systemic circulation by the LVAD can result in increased venous return to the right ventricle, thereby modifying right ventricular preload. Second, pulmonary arterial and right ventricular systolic pressures and wall stress (afterload) in patients with normal pulmonary vascular resistance may be reduced indirectly due to a reduction in left ventricular filling pressure produced by the translocation in blood volume away from the pulmonary circulation. Third, the septal contribution to right ventricular contraction may be reduced in some patients due to left ventricular pressure and volume unloading, effectively reducing right ventricular contractility.

The results of the present study support the second hypothesis, especially in the patients with poor preoperative left ventricular ejection fractions. The data from the poor left ventricular function group also show venous return and cardiac output to increase during left heart bypass, as predicted by the first hypothesis. In fact, increased venous return appeared to be handled easily by the right ventricle since the pulmonary arterial and right ventricular end-diastolic pressures decreased during left ventricular bypass. The third hypothesis cannot be tested by the data presented here, but it is supported by studies in normal dog hearts,<sup>16, 25</sup> as well as by studies from isolated hearts that show reduced right ventricular performance and reduced coupling between the ventricles when the left ventricle is pressure unloaded.<sup>26–28</sup>

Because it is easier to pressure unload a left ventricle with poor function (79% decrease in left ventricular peak systolic pressure) than with normal function (45% decrease in left ventricular peak systolic pressure), the effects on interventricular septal wall stress and its contribution to right ventricular function may differ in each group. Further unloading, as in some of the animal preparations, may produce even greater reductions in this contribution. Right ventricular function during left heart assistance is determined, therefore, by the resultant balance among these factors, a balance that most likely varies under different conditions of cardiac pathology.

An illustration of the interaction between these hy-



**FIGURE 5.** Hemodynamic interactions between the right ventricle (RV) and the left ventricle (LV) are due to the two hearts being in series, connected by the systemic (SVR) and pulmonary (PVR) vascular resistances; mechanical interactions are due to the anatomic coupling provided by the shared interventricular septum (A). An LVAD in parallel with the left ventricle (B and C) can alter right ventricular preload and afterload by shifting blood volume from the pulmonary to the systemic vascular systems. An LVAD that unloads the left ventricle can result in a leftward septal shift by reducing the transseptal pressure gradient (B). If the pulmonary arterial and right ventricular pressures are also reduced during left ventricular bypass then both ventricles can become dimensionally unloaded (C).

potheses is shown in figure 5. Left ventricular unloading with an LVAD may result in a leftward shift of the interventricular septum (figure 5, B) as blood is pumped by the LVAD in parallel with the left heart and in series with the right heart.<sup>1, 17</sup> However, if the pulmonary arterial pressure also falls during left ventricular bypass, then the right ventricle as well as the left ventricle may be dimensionally unloaded, as demonstrated by echocardiographic studies<sup>18</sup> and illustrated in figure 5, C. The relative septal displacement in each condition is determined by the transseptal pressure gradient,<sup>29</sup> or more accurately by the transseptal force gradient, which includes the effects of chamber configuration.<sup>17</sup>

Caution should be exercised in extending the results of this study of short-term left ventricular assistance to patients with LVADs over the long term or to those who receive LVADs after open heart surgery. Our results demonstrate that acute left ventricular unloading during left ventricular bypass does not result in right ventricular failure in patients undergoing routine open heart surgery. In fact, the resultant reduction in

right ventricular afterload in patients with depressed preoperative left ventricular function produced an improvement in right ventricular function. The high incidence of right ventricular failure in the clinical experience with temporary LVADs is probably due to the fact that patients who require LVAD support have much more severely depressed biventricular function, possibly with perioperative infarction,<sup>8</sup> than any of the groups of patients or dogs from the model studies. Mechanical support of only one ventricle in a patient with biventricular failure may unmask the preexisting dysfunction of the other. In addition, patients with LVADs and elevated pulmonary vascular resistance due to obstructive disease or to other factors, such as those reported by Pennington *et al.*,<sup>8</sup> may not benefit from the right ventricular afterload reduction seen in the present study. In these cases the pumping ability of the right ventricle may not be able to match that of the LVAD. Future research should concentrate on the effect of left ventricular bypass on the three determinants of right ventricular function in patients with biventricular dysfunction or elevated pulmonary vascular resistance. The pathophysiology of other causes of right ventricular failure should also be investigated.

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