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CARDIOPULMONARY BYPASS, MYOCARDIAL MANAGEMENT, AND SUPPORT TECHNIQUES

Left-to-right ventricular interaction with a noncontracting right ventricle

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Abstract

Left ventricular systole is known to contribute to generation of right ventricular pressure and stroke volume. To study the interactions in a dilated noncontractile right ventricle after cardiopulmonary bypass we created a variable volume, neo-right ventricle by excision and replacement of the right ventricular free wall with a xenograft pericardial patch. We investigated the interactions in eight dogs with neo-right ventricle, instrumented to measure cardiac pressures and cardiac output in control conditions (*n* = 69) and during partial pulmonary artery occlusion (*n* = 50). *Results:* The size of the neo-right ventricle was increased from original right ventricular volume V_0 to V_1 ($V_1 = V_0 + 54 \pm 23$ ml), V_2 (V

$_2$ = V $_0$ + 124 ± 85 ml), and V $_3$ (V $_3$ = V $_0$ + 223 ± 162 ml). Cardiac output increased with increasing left ventricular end-diastolic pressure, indicating that the Frank-Starling mechanism was operating in the left ventricle. However, cardiac output decreased with increasing neo-right ventricular size (*p* < 0.001) and during pulmonary artery occlusion (*p* < 0.001). Maximal neo-right ventricular pressure was a linear function of the maximal left ventricular pressure at each neo-right ventricular size and decreased with the increase in neo-right ventricular size (*p* < 0.001), both in control conditions and during pulmonary artery occlusion (*p* < 0.004). Stroke work of the neo-right ventricle and left ventricle decreased with increasing neo-right ventricular size (*p* < 0.002). The relationship between neo-right ventricular stroke work and left ventricular stroke work at different neo-right ventricular sizes was linear both in control conditions and during pulmonary artery occlusion: in control Y = 0.24X (*r* = 0.968, *n* = 69); in pulmonary artery occlusion Y = 0.35X (*r* = 0.986, *n* = 50). In both conditions the intercept of the linear relationship was not significantly different from zero (*p* < 0.974 in control; *p* < 0.614 in pulmonary artery occlusion). The slope was significantly increased in pulmonary artery occlusion (*p* < 0.001). *Conclusion:* Left ventricular contraction contributes 24% of left ventricular stroke work to the generation of right ventricular stroke work via the septum in the absence of a contracting right ventricle; this increases to 35% in the face of increased pulmonary afterload. This mechanism can maintain adequate global cardiac function in the case of a noncontracting right ventricle while right ventricular volume is kept small and afterload is not increased. The interventricular interaction of the ventricles must be considered when patients with postbypass right ventricular failure are treated. (J T HORAC C ARDIOVASC S URG 1994;107:1496-1502)

The role of the right ventricle (RV) as a passive conduit was enshrined after the cautery experiments of Starr, Jeffers, and Meade,[1] and its dispensibility seemed confirmed by the work that culminated in the successful clinical application of the Fontan procedure and its variants.[2,3] However, the classic medical management of RV infarction aimed at increasing the preload to "force" blood through the passive RV "conduit" and enhance left ventricular (LV) preload is clearly not always effective. Recent studies with a variety of LV assist devices have detected a rate of RV failure of up to 25% in patients after cardiotomy.[4] It has been shown in patients with RV failure after cardiopulmonary bypass that the conventional practice of volume loading in fact induces further deterioration of cardiac function.[5] Thus a failing, noncontractile RV after cardiopulmonary bypass poses an important problem for the maintenance of global cardiac function.

In normal hearts, left-to-right systolic ventricular interaction through the septum contributes to the generation of RV pressure and stroke volume.[6,7] A number of studies have confirmed that there is substantial transmission of systolic forces from the LV that contribute to RV ejection. In addition to studies of normal physiologic conditions, it was also shown that the LV contributes to RV pressure development and ejection in a nonfunctional electrically isolated RV[8] and in an RV with prosthetic myocardium.[9] However, the mechanisms of this ventricular interaction and the effects of varying size of the nonfunctional RV on overall cardiac function remain to be fully elucidated and quantified. In our present study we aimed to examine and quantify the LV contribution to RV function in a model of the postbypass noncontractile right ventricle.

We investigated left-to-right systolic interventricular interaction with surgically created nonfunctional RV by replacing RV free wall with a xenograft pericardial patch. The end-diastolic volume of such a "neo-RV" could be changed, thereby allowing us to investigate and quantify interventricular interaction as a function of the neo-RV size. We tested a number of hypotheses: (1) the LV alone is capable of generating satisfactory cardiac output and stroke work; (2) the ability of the LV to generate cardiac output and stroke work is dependent on left-to-right interaction via the interventricular septum; and (3) left-to-right interaction is affected by the size of the neo-RV and changes with alterations in RV afterload.

METHODS

Operation

Eight large-chested hounds (26 ± 3 kg) were obtained from our licensed vendor and handled throughout the study in accordance with the humane standards established by the Albert Einstein College of Medicine animal care and use committee and in accordance with the "Guide for the Care and Use of Laboratory Animals" (NIH Publication No. 80-23).

Anesthesia was induced with thiopental sodium (20 mg/kg); the dogs were intubated and the lungs ventilated with a Bird respirator (Bird Corp., Palm Springs, Calif.). Anesthesia was maintained with 0.5% halothane and oxygen, and the animal's temperature was maintained at 37° C with use of a warming blanket. The right femoral artery, right carotid artery, and right internal jugular vein were exposed. Median sternotomy and right thoracotomy were done and the heart exposed in a pericardial cradle. After anticoagulation with heparin (5000 U, intravenously) a Millar (Millar Instruments, Inc., Houston, Tex.) micromanometer was inserted via the carotid artery and a Swan-Ganz catheter (Baxter Healthcare Corp., Edwards Division, Irvine, Calif.) via the jugular vein. Peratrial bicaval cannulation with 28F venous cannulas was done and tapes were placed around the superior and inferior venae

cavae. The right femoral artery was cannulated with a 15F cannula for arterial return. After cardiopulmonary bypass was begun, the caval tapes were snared to achieve total bypass.

The RV was incised and a diathermy knife was used to excise the free wall of the RV. The lines of excision extended from near the pulmonary valve anulus parallel to and 0.5 cm to the right of the left anterior descending coronary artery, around the apex, and up along the atrioventricular groove. Care was taken to preserve the attachment of the tricuspid valve. After the excision was completed, a direct-current countershock was used to defibrillate the heart and the remainder of the surgical procedure was completed with the heart beating and empty.

A diamond-shaped single piece of calf pericardium (approximately 20 cm by 10 cm) tanned in 0.625% glutaraldehyde was used to patch the RV: the material is easy to handle, moderately stiff, conforms nicely, and holds sutures well. Variations in patch size were a result of differences in the size and shape of the available pericardia. The apices of the patch were sutured to the apices of the defect with 4-0 polypropylene, and then continuous sutures were run toward the midpoint of the defect on each side. Here the sutures from above and below were knotted, and the two ends on each side were then run along the sides of the redundant patch (one mattress and one over-and-over) to form a watertight seam. Before the sutures were completed, the Swan-Ganz catheter was positioned in the right pulmonary artery. In this way, the excised free wall of the RV was replaced by a neo-RV (Fig. 1).

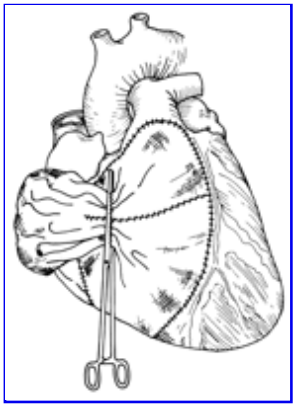

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Fig. 1. Surgical method. After RV free wall was incised, neo-RV was created using xenograft pericardial patch. Volume of neo-RV can be changed by clamping pericardial patch at various levels. At its minimum size (clamp at base of patch), neo-RV approximated volume of original RV. Clamping at different levels yielded incremental neo-RV volumes.

With the patch complete, and after hemostasis was checked, ventilation was

recommenced. The patch was clamped at its base, creating a neo-RV as near the volume of the original RV as possible, and cardiopulmonary bypass was discontinued easily in all cases. The caval cannulas were removed, but the arterial cannula was retained and used for transfusion as needed. Assisted ventilation with 40% oxygen was continued and anesthesia was provided by intermittent fentanyl (10 μg/kg intravenously) supplemented with vecuronium (0.5 μg/kg intravenously) as needed to ensure an appropriate level of anesthesia. The level of anesthesia was monitored and confirmed by corneal and toe pinch reflexes. During the course of the experiment blood pressure, heart rate, arterial blood gases, pH, and hematocrit values were monitored and maintained within normal physiologic ranges.

A pressure micromanometer catheter was placed in the carotid artery and then passed across the aortic valve into the LV. Additional pressure micromanometers were placed in the right atrium (through the appendage) and in the neo-RV (through the base of the patch). Cardiac output was measured with the thermodilution cardiac output computer. All catheters were calibrated to the mid-LV cavity level and checked for zero shift before recording.

Experimental protocol

After placement of instruments, hemodynamic status was allowed to stabilize over a period of 15 minutes with the patch at minimum volume before recordings were begun. Before measurements were taken we ensured that there was no evidence of tricuspid regurgitation. The baseline hemodynamic recordings were made at V_0 (minimum volume of the neo-RV which approximated the original RV volume). Neo-RV volume was increased incrementally by changing the position of the clamp. After trial clamping across the patch we determined the sites of clamp application that would provide approximately equal increments of neo-RV volume. Marking sutures were placed at the selected sites on each of the seam lines along the patch to ensure reproducibility of clamp position. At every level of clamp application further hemodynamic recordings were made under steady-state conditions. To achieve steady-state conditions after clamp repositioning we had to wait approximately 3 to 5 minutes. After V_{max} (maximum volume of the neo-RV) was reached, and after recordings were completed, the neo-RV was slowly compressed and the clamp was again applied across the base of the patch at the V_0 position and hemodynamic records obtained at steady-state.

After the measurements were completed at control levels of pulmonary arterial afterload (control), we increased the afterload by partially occluding the main pulmonary artery (PAO). New baseline hemodynamic data were recorded at V_0 and the snare on the pulmonary artery (PA) was tightened to increase PA pressure by 10

to 15 mm Hg higher than baseline levels. The series of measurements at different neo-RV volumes (from V_0 to V_{max} and back to V_0) was repeated.

At the end of the experiment animals were killed by intracardiac injection of potassium chloride. The hearts were excised and neo-RV volumes were measured for each level of clamp application and recorded. The volume determination was made immediately after excision of the heart as follows: the PA was clamped and the neo-RV was filled with water under pressure through the right atrium until the patch was full and the tricuspid valve ballooning. A slit was made at the highest point of the patch and the water emptied into a graduated measuring cylinder. The clamp was then placed at the point corresponding to the next largest neo-RV volume, the neo-RV filled as before, and volume measured as described. Three measurements were made at each size of neo-RV and the mean value was recorded rounded to the nearest milliliter. These measurements were considered to approximate diastolic volumes of the neo-right ventricle at steady-state.

Date acquisition and analysis

Pressures, flows, and electrocardiogram were recorded on a standard strip chart recorder (Gould, Inc., Cleveland, Ohio) and a high-speed photographic recorder (Electronics for Medicine, White Plains, N.Y.). Pressure parameters were measured manually from the paper recordings. Cardiac output by the thermodilution method was determined at each level of RV volume (three times) and the average value was accepted. Stroke volume was determined from the ratio of the averaged cardiac output and heart rate. Stroke work of the LV and neo-RV was determined as a product of stroke volume (SV) and peak ejection pressure (P) ($SW = SV \times P_{max}$).

To test for significant relations between hemodynamic variables and to assess the changes after acute increase in RV afterload, we used a multiple linear regression implementation of a repeated-measures analysis of variance with dummy variables.[10] We used the multiple linear regression model that accounted for the effects of neo-RV size, increased RV afterload caused by PAO, and differences between dogs. The specific regression model was as follows:

$$Y = a_0 + a1 \cdot X + a2 \cdot I + a3 \cdot I \cdot X + a4 \cdot S + \sum(b_i D_i) \quad (1)$$

where Y and X are dependent and independent variables of interest. Coefficients a_0 and a_1 are intercept and slope of the linear relationship between Y and X. The dummy variable I (intervention) equals 0 before PAO and 1 afterward. Coefficients a_2 and a_3 describe the change in intercept and slope caused by PAO and the coefficient a_4 describes the effects of neo-RV size on the intercept. The n - 1 dummy variables b_i

account for between-dog variability in the intercept. These dummy variables are defined according to the following:

$D_i = 1$ (if dog $i < n - 1$)

$D_i = -1$ (if dog n)

$D_i = 0$ otherwise

All regression coefficients are reported with their associated standard errors. Computations were done with SIGMASTAT (Jandel Scientific, Mountain View, Calif.). We considered differences to be significant when $p < 0.05$.

RESULTS

We analyzed a total of 119 different hemodynamic conditions under normal RV afterload ($n = 69$) and with increased RV afterload caused by PAO ($n = 50$).

The size of the neo-RV was varied in each dog. Mean values of neo-RV were changed from V_0 (minimum volume, which is close to the original RV volume) to V_1 ($V_1 = V_0 + 54 \pm 23$ ml), V_2 ($V_2 = V_0 + 124 \pm 85$ ml), and V_3 ($V_3 = V_0 + 223 \pm 162$ ml). Illustration of the changes in hemodynamic variables with the change of neo-RV size is shown in Fig. 2. Numeric results of analysis by equation 1 are summarized in Table I. Dependent variables are shown as column headings and independent variables as raw labels. Cardiac output increased with increasing LV end-diastolic pressure (LVEDP). However, cardiac output decreased with increasing neo-RV size ($p < 0.001$) and during PAO ($p < 0.001$).

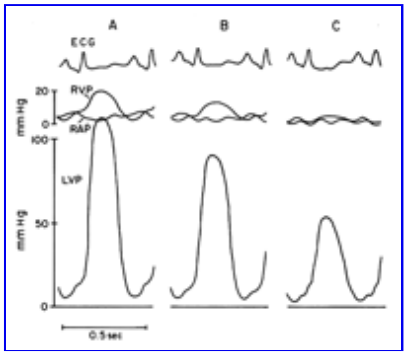

Fig. 2. Hemodynamic data obtained at different sizes of neo-RV: minimum size *(panel A)*, intermediate size *(panel B)*, and maximal size *(panel C)*. *LVP*, Left ventricular pressure; *RVP*, right ventricular pressure; *RAP*, right atrial pressure; *ECG*, electrocardiogram.

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Table I. Hemodynamic variables and change with neo-RV size and PAO

Maximal neo-RV pressure was a linear function of the maximal LV pressure at each neo-RV size (Table I, Fig. 3). Maximum LV pressure and neo-RV pressure decreased with increasing neo-RV size ($p < 0.001$) both in control conditions and during PAO. The linear relationship was preserved after the increase of RV afterload but with an increased slope. The influence of the PAO was statistically significant ($p < 0.004$, Table I, Fig. 3).

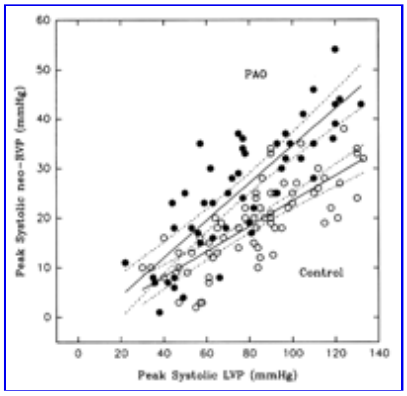

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Fig. 3. Maximal neo-RV pressure *(RVP)* as function of maximal LVpressure *(LVP)* in control (hollow symbols) and afterpartial PAO *(filled symbols)*. After effects of between-subject variability were taken into account, relationships between peak systolic neo-RV pressure (dependent variable, Y) and peak systolic LV pressure (independent variable, X) were as follows: in control Y = 0.27 X – 3.79 ($r = 0.892$, $n = 69$); in PAO Y = 0.42 X – 6.72 ($r = 0.871$, $n = 50$). Slopes of two relationships were significantly different ($p < 0.001$), whereas intercepts were not different ($p < 0.079$). Dashed lines indicate 95% confidence limit.

Stroke work of both the neo-RV and LV decreased with increasing neo-RV size ($p < 0.002$). The relationship between stroke work generated by the neo-RV and LV at different neo-RV sizes was linear in both control and PAO runs: in control Y = 0.24X ($r = 0.968$, $n = 69$); in PAO Y = 0.35X ($r = 0.986$, $n = 50$). In both conditions the intercept of the linear relationship was not significantly different from zero ($p < 0.974$ in control; $p < 0.614$ in PAO). Thus

neo-RV stroke work = slope · LV stroke work (3)

where the slope represents the ratio of the stroke work generated by the neo-RV and LV. This ratio (slope) was significantly increased in PAO ($p < 0.001$, Table I, Fig. 4).

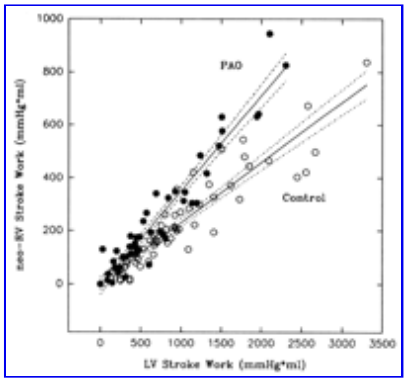

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Fig. 4. Neo-RV stroke work as function of LV stroke work in control *(hollow symbols)* and after partial PAO (filled symbols). After effects of between-subject variability were taken into account, relationships between neo-RV stroke work (dependent variable,Y) and LV stroke work (independent variable, X) were as follows: in control Y = 0.25X (r = 0.968, n = 69); in PAO Y = 0.35X (r = 0.975, n = 50). Slopes of two relationships were significantly different ($p < 0.001$), whereas intercepts were not different from zero (in control $p < 0.191$; in PAO $p <$ 0.12). *Dashed lines* indicate 95% confidence limit.

DISCUSSION

Diagnosis and treatment of acute RV failure presents a major problem in cardiac transplantation and in the use of LV assist devices. A major issue not yet settled is how much RV function is necessary for adequate systemic cardiac output. Because the function of the RV is physiologically influenced by the LV, an important subsidiary question is how much of the RV mechanical output is a result of RV-LV interaction in the case of a nonfunctional RV.

Starr, Jeffers, and Meade [1] cauterized the free wall of the RV and found no change in acute hemodynamics, and ligation of coronary vessels in long-term studies produced a similar result. Others reproduced this result but noted that if the "pulmonary vascular resistance was raised, the heart does not do well" and that if the ventricles are not efficiently coupled, maintained cardiac output requires a high end-diastolic pressure. [2] Bypassing the RVwas done by Rodbard and Wagner [11] and later by Glenn [2] and Fontan and Baudet [3] and others, culminating in the successful repair of tricuspid atresia. It was the accepted wisdom that the RV is redundant, and in 1984 Furey, Zieske, and Levy [12] hypothesized that the essential function of the RV is not perfusion of the pulmonary circulation at all, but rather is to maintain an acceptably low pressure in the very distensible central veins.

The septal contribution of LV work to RV output has been studied in a variety of elegant ultrasonic crystal experiments [13,14] and by echocardiography [15] and in a clinical study [5] that confirmed that the LV contributes to the generation of systolic pressure in the RV, a normal effect that is usually masked by the near synchrony of contraction of the two ventricles.

Sawatani and associates [16] replaced the free wall of the RV (using deep

hypothermia and inflow occlusion) with a Dacron patch and used angiography to demonstrate that the interventricular septum bulges into the RV during systole, while the fixed prosthetic wall of the RV moves toward the interventricular septum. In the normal RV with its high surface-to-volume ratio, they concluded that slight movement of the two walls of the RV toward one another can displace a large volume of blood at low pressure into the low-resistance pulmonary circulation.

We have developed an experimental model that produces a noncontractile, variable-volume RV. Our results show that the LV is capable of generating satisfactory cardiac output when the noncontracting RV is relatively small. The cardiac output was a function of LVEDP, which indicated that in this preparation the Starling mechanism for the whole heart is operating because of the preload of the LV only. With the use of this model we have been able to confirm the important role of interventricular interaction in maintaining output from an impaired RV. Our results show the existence of important systolic LV-RV interaction, which enables the preservation of heart function in the absence of a functional RV. We have been able to quantify the contribution of the LV to the stroke work of the neo-RV under a variety of loading conditions: with normal afterload, 24% (slope in equation 2 in percent) of the LV stroke work is "transmitted" through the septum to the RV. Recent study in pigs showed that when the LV is rapidly unloaded with an LV assist device RV stroke work in normal hearts decreased 26.5%,[17] which is consonant to the finding in our study. Our results also indicate that 35% (slope in equation 2 in percent) of LV stroke work is "transmitted" to the generation of the RV stroke work during PAO. The higher systolic neo-RV pressures during PAO contributed to this increase in the "transmitted" work.

If the noncontractile RV is distended excessively, or the pulmonary vascular resistance is increased, the heart fails rapidly. Systolic "support" of the RV by the LV cannot maintain cardiac output in the face of high pulmonary vascular resistance when the RV is and acts as a capacitance chamber.

Although our model was developed primarily to study the physiology of ventricular interaction, it has clinical relevance too inasmuch as our experimental preparation simulates the dilated and nonfunctional RV that might result after cardiopulmonary bypass from severe RV ischemia (but without tricuspid regurgitation). Extrapolating our findings to the clinical scenario of a poorly functioning, dilated RV after cardiopulmonary bypass, the striking clinical implication is that if LV function is well preserved, and therefore capable of generating satisfactory cardiac output, the damaged RV should not be aggressively volume-loaded. Excessive volume loading and/or an acute increase in RV afterload will impair the interventricular interaction and interfere with the LV contribution to RV ejection, which in turn will limit LV

filling and ultimately lead to a decrease in cardiac output.

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Footnotes

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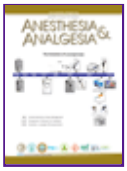

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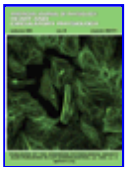

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