SURGERY FOR CONGENITAL HEART DISEASE

HEMODYNAMIC EFFECT OF PROGRESSIVE RIGHT ATRIAL DILATATION IN ATRIOPULMONARY CONNECTIONS

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Objective: Right atrial dilation occurring late after the modified Fontan procedure is frequently associated with low output states, supraventricular arrhythmias, and atrial thrombus formation. We addressed the hypothesis that progressive right atrial dilatation contributes to inefficient right heart flow dynamics. **Methods:** Modified atriopulmonary connections were performed on explanted isolated sheep heart preparations with various degrees of surgically induced right atrial dilatation (right atrial volumes 6 to 55 cm³). Flow models were perfused in an in vitro flow loop with the use of a blood analog fluid. A fluid energy balance was performed for six flow rates (1.0 to 6.0 L/min) at each degree of right atrial dilatation, and the rate of total fluid energy loss was calculated and expressed as a function of right atrial volume and flow rate. Effective pressure drop and fluid resistance across the right atrial chamber were also determined for each flow condition. **Results:** The rate of fluid energy loss increased with increasing right atrial dilatation and flow rate for all conditions studied ($p < 0.001$). Over the range of right atrial volumes and flow rates examined, the average increase in the rate of energy loss was 3.8- and 117-fold, respectively. Calculated fluid resistance through the right atrium also increased with increasing right atrial volume and flow rate ($p < 0.001$), exhibiting an average increase of 3.2- and 3.3-fold respectively. **Conclusions:** Right atrial dilatation in atriopulmonary connections causes fluid energy losses and increases the energy required to move blood from the venae cavae to the pulmonary arteries. These observations may help explain the progressive nature of late failures of atriopulmonary connections and provide additional rationale for conversion from atriopulmonary connections to lateral tunnel total cavopulmonary connections in selected patients. (J Thorac Cardiovasc Surg 1997;114:2-8)

Shortly after the description of the first successful right ventricular bypass operation for tricuspid atresia,¹ Kruetzer and associates² described the

modified Fontan procedure and demonstrated that the entire venous return could be diverted to the pulmonary circulation through a single valveless atriopulmonary connection (APC). Whereas direct anastomosis of the right atrium to the main pulmonary artery has been widely used to separate the systemic and pulmonary circulations in patients with complex congenital heart disease, several early and late postoperative complications associated with this procedure have diminished its use in place of more hemodynamically efficient total cavopulmonary modifications.³ Although the total cavopulmonary connection (TCPC) has replaced the APC in many centers, survivors with failing APCs in the late postoperative period are a significant clinical

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Fig. 1. Schematic diagram of the in vitro flow loop. *SVCP*, Superior vena cava pressure; *IVCP*, inferior vena cava pressure; *RAP*, right atrial pressure; *MPAP*, main pulmonary artery pressure; *LPAP*, left pulmonary artery pressure; *RPAP*, right pulmonary artery pressure.

management problem. We postulated that the progressive right atrial dilatation often seen in these patients may contribute to the hemodynamic inefficiency of the APC. Although several previous studies have compared the fluid dynamics of APCs to TCPC alternatives,⁴⁻⁷ the consequence of progressive right atrial dilatation on right heart hemodynamics has not been previously addressed by means of quantitative tools. Accordingly, we addressed the hypothesis that progressive right atrial dilatation in APC results in increased fluid energy losses and increased resistance to flow in the pulmonary circulation.

Methods

Flow models. Modified APCs were performed on fresh explanted sheep heart-lung blocks (subjects: 25 to 30 kg) by means of standard surgical techniques.⁸ After an oblique incision was made, the right atrial appendage was connected to the transected proximal main pulmonary artery. Simulation of right atrial dilation was achieved surgically by creating an incision in the anterior wall of the right atrium and sewing a patch of pericardial tissue into the right atrial chamber. The heart and pericardial patch were fixed in a glutaraldehyde solution to avoid aneurysmal dilatation of the patch area. Six right atrial sizes were studied ranging from 6 to 55 cm³, which represents normal to severe right atrial dilatation in children.

Flow conditions. The hemodynamic effect of right atrial dilatation was studied with the use of the in vitro flow loop shown in Fig. 1. After surgical construction of the tissue models, each vena cava and pulmonary artery was cannulated with 3/8- and 1/4-inch bypass tubing connectors,

respectively, and attached to appropriate sized tubing. So that flow, pressure, and velocity artifacts could be eliminated, the heart and all vessels were positioned to match physiologic vessel/heart spatial orientations. A nonrestrictive atriopulmonary anastomosis was confirmed in each model by visual inspection and by simultaneous pressure measurements immediately proximal and distal to the anastomosis at several flow rates. The anastomosis was considered to be nonrestrictive if pressure gradients were less than 1 mm Hg at flow rates up to 5 L/min. This ensured that calculated rates of fluid energy loss were attributed to the geometry of the entire model and not to a restrictive anastomosis. The tissue models were then perfused with a blood analog fluid (33% glycerin/water, viscosity 3.5 cp) using a calibrated roller pump at a total of six flow rates (1 to 6 L/min), producing a range of Reynolds' numbers (ratio of inertial to viscous forces) of 223 to 2957. Superior and inferior vena caval flows were set to 35% and 65% of the total flow rate, respectively, to account for the greater flow contribution of the inferior vena cava.⁹ Vena caval and main pulmonary artery flows were measured with the use of a Transonics HT207 ultrasonic flowmeter and 10 to 14 mm flow probes (Transonic Systems, Utica, N.Y.) placed loosely around each vessel. Right atrial, proximal vena caval, and main pulmonary artery pressures were measured with side-hole 0.1-inch fluid-filled catheters inserted through positioned pressure ports in the respective cannulas and into the area of interest. Pressure transducers were interfaced to a clinical pressure measurement system (Electronics for Medicine, Jupiter, Fla.) and a Macintosh 840AV personal computer by means of customized software (Lab View Software, National Instruments, Austin, Tex.) for automated data acquisition. Average velocities in each vessel were calculated from instantaneous flow values and diam-

Fig. 2. Rate of fluid energy loss versus right atrial volume for two flow rates (2 and 5 L/min).

eter measurements of fully distended vessels. Flow conditions and vessel dimensions are summarized in Table I.

Data analysis: Rate of fluid energy loss. To determine the rate of fluid energy required to drive equivalent flows through hearts with different degrees of right atrial dilatation, we analyzed the rate of fluid energy loss for each flow rate and right atrial volume. This analysis requires knowledge of pressure, flow, and velocity at the inlets and outlet of the right atrial chamber and is especially useful because it includes all potential sources of energy loss: static and kinetic energy losses associated with entrance and exit effects, and viscous dissipation caused by flow collision and mixing. In this study, analysis of the rate of fluid energy dissipation consists of a hydraulic energy balance over the right atrial chamber. That is, the rate of fluid energy entering the right atrium ($E_{svc} + E_{ivc}$) must equal the rate of fluid energy leaving the right atrium (E_{mpa}) plus any incurred losses (E_{loss}):

$$E_{loss} = E_{svc} + E_{ivc} - E_{mpa} \quad (\text{Eq 1})$$

where:

$$E_i = Q_i(P_i + \frac{1}{2}\rho v_i^2) \quad (\text{Eq 2})$$

for $i = svc, ivc, mpa$. Equation 2 represents both static and kinetic energy contributions where E_{loss} is the total rate of fluid energy loss (W), Q is the flow rate (m^3/sec), P is the static pressure (N/m^2), v is the average velocity (m/sec) and ρ is the fluid density (kg/m^3). Combining equations 1 and 2, we arrive at an equation that represents the total rate of energy loss occurring across the right atrial chamber:

$$E_{loss} = Q_{svc}(P_{svc} + \frac{1}{2}\rho v_{svc}^2) + Q_{ivc}(P_{ivc} + \frac{1}{2}\rho v_{ivc}^2) - Q_{mpa}(P_{mpa} + \frac{1}{2}\rho v_{mpa}^2) \quad (\text{Eq 3})$$

Table 1. Summary of flow conditions

Fluid viscosity (cP)	3.3
Right atrial volume (cm^3)	6-55
Vessel diameters (cm)	
SVC	1.03 ± 0.11
IVC	1.23 ± 0.13
MPA	1.33 ± 0.37
Velocities (cm/sec)	
SVC	7.0-42.0
IVC	9.12-55.01
MPA	12.0-71.9
Flow rates (L/min)	
Total	1-6
SVC (35%)	0.35-2.80
IVC (65%)	0.65-5.20
Reynolds' numbers	
SVC	223-1337
IVC	349-2094
MPA	492-2957

SVC, Superior vena cava; IVC, inferior vena cava; MPA, main pulmonary artery.

For each right atrial size, the total rate of energy loss was calculated using equation 3 for equivalent flows and compared. From these values, the effective pressure drop and fluid resistance to flow through the right atrium were calculated by means of equations 4 and 5, respectively, and expressed as a function of right atrial size:

$$\Delta P_{ef} = \frac{E_{loss}}{Q} \quad (\text{Eq 4})$$

$$R_{ef} = \frac{\Delta P_{ef}}{Q} = \frac{E_{loss}}{Q^2} \quad (\text{Eq 5})$$

where ΔP_{ef} (mm Hg) and R_{ef} ($\text{mm Hg}/\text{L min}^{-1}$) are the effective pressure drop and fluid resistance across the right atrial chamber, respectively.

Statistical analysis. The rate of fluid energy loss and resistance data are expressed as the mean value of three consecutive measurements at the same flow conditions ($n = 108$ experiments). Differences in the rate of fluid energy loss and resistance with increasing right atrial dilation and flow rate were considered significant at a level of $p < 0.01$ (two-way analysis of variance).

Results

Fig. 2 demonstrates the effect of right atrial volume on the rate of fluid energy loss for two flow rates representing resting and moderate output states. For these two flow rates (1 and 5 L/min), there was a 4.1- and 3.2-fold increase in the rate of fluid energy loss, respectively, across the range of right atrial sizes. The effect of flow rate on the rate of fluid energy loss for two right atrial volumes (11 and 38 cm^3) representing mild and moderate right atrial dilatation is shown in Fig. 3. Fluid energy losses at these two volumes increased 96 and 126

Fig. 3. Rate of fluid energy loss versus flow rate for two right atrial volumes (11, 38 cm³).

times for the 11 and 38 cm³ chambers, respectively, from the lowest to highest flow rates. A summary of the rate of fluid energy losses for all conditions is shown in Fig. 4. At lower flow rates, energy losses are small and do not exhibit significant changes with increasing right atrial volume. At higher values, however, the rate of fluid energy loss is a clear function of flow and right atrial volume ($p = 2.55E^{-11}$, $p = 3.71E^{-4}$).

Effective pressure drop versus right atrial volume and flow rate is shown in Fig. 5. As expected from equation 4, the effective pressure drop is a function of both flow and right atrial volume with values ranging from 0.1 to 7 mm Hg. Fig. 6 compares the individual contributions of flow rate and right atrial volume to the calculated fluid resistance for all conditions studied. Resistance increased with increasing right atrial volume for each flow rate and averaged a 3.2-fold increase over the range of volumes ($p = 5.46E^{-9}$). The dependence on flow rate was similar, with resistance increasing with flow rate and averaging a 3.3-fold increase over the range of flows ($p = 4.02E^{-11}$).

Discussion

The initial report of the Fontan procedure² has revolutionized the management of patients with complex congenital heart disease unsuitable for biventricular repair and has created an exciting forum for innovative Fontan modifications. The description of the valveless APC by Kreutzer and colleagues² was the first in a series of modifications to the original procedure. Although this

Fig. 4. Rate of fluid energy loss versus right atrial volume (RAV) and flow rate for all conditions.

procedure was quickly endorsed and has had widespread use at many centers, long-term outcome remains relatively disappointing.¹⁰⁻¹⁴ Early demonstration that inlet and outlet valves were not needed for the successful function of the circulation,¹⁵ as well as a high incidence of right atrium-related complications (e.g., supraventricular arrhythmias and atrial thrombus formation), led many to question the role of the pulsating right atrium and its actual contribution to the Fontan circulation. de Leval and associates³ performed studies to determine the hemodynamic efficiency of APCs and to determine the role of the right atrium in APC. They concluded that the hydrodynamic design of the APC was relatively poor and that the interposition of a passive chamber with impaired systolic function between the systemic veins and the pulmonary arteries was a major cause of flow inefficiency in the pulmonary circulation. On the basis of these experimental results, de Leval's group described the lateral tunnel TCPC and demonstrated more streamlined flow patterns with less turbulence and fluid energy losses compared with the APC connection.³ These experimental fluid dynamic observations have been confirmed by in vitro⁴⁻⁷ and computational studies¹⁶ comparing the hydrodynamic efficiency of APC and TCPC. More recent studies have placed emphasis on optimization of the TCPC.¹⁷⁻¹⁹

Although the TCPC has been widely adopted in place of the APC in many centers, long-term survi-

Fig. 5. Summary of effective pressure drop versus right atrial volume (RAV) and flow rate for all experiments (calculated by means of equation 4).

vors of APC pose difficult management problems. In the “failing” atriopulmonary Fontan circulation, it is common to see marked right atrial distention, which is often associated with low output states, supraventricular arrhythmias, and right atrial thrombus formation. The relationship between low cardiac output states and exercise intolerance with increasing right atrial dilatation, however, is not clear. We postulated that right atrial dilatation may not only be a consequence of adverse hemodynamic conditions (chronic right atrial hypertension and increased intravascular volume), but also may contribute to hydrodynamic inefficiency in the pulmonary circulation.

This study used an in vitro flow loop and data analysis techniques designed specifically to isolate the effect of right atrial dilatation on the fluid dynamics of the pulmonary circulation. These results confirm our hypothesis that right atrial dilatation decreases the efficiency of the pulmonary circulation and therefore may have important clinical implications.

Etiology of fluid energy losses with right atrial dilatation. Evaluation of the geometry of the dilated right atrium and the location of inlet and outlet vessels suggests three potential sources of fluid energy loss and adverse hemodynamics: (1) fluid losses associated with flow expansion from the venae cavae into the right atrial chamber, (2) collision and mixing of vena caval flows, and (3) flow contraction from the distended right atrium into the main pulmonary artery. Under ideal flow conditions

Fig. 6. Effective fluid resistance versus right atrial volume (RAV) and flow rate for all experiments (calculated by means of equation 5).

(i.e., nonviscous flow), as a fluid expands from the venae cavae into the dilated right atrium, a portion of kinetic energy is converted to potential energy. On flow constriction into the main pulmonary artery from the right atrial chamber, the same portion of energy is completely converted back into kinetic energy (i.e., no fluid energy losses). For actual right atrial flow conditions, however, flow separation and collision in the right atrial chamber results in irrecoverable fluid energy losses associated with mixing and viscous dissipation, such that a portion of the potential energy in the right atrium is not converted back into kinetic energy. The most important parameter affecting the degree of energy loss when considering the flow of a viscous fluid through an abrupt flow expansion or contraction is the ratio of the initial to expanded flow area. For unidirectional flow, this ratio indicates the degree of flow separation that controls the mechanical energy loss accompanying the abrupt change in flow area. In the case of the right atrium, in addition to losses associated with flow expansion and contraction, there is also a collision of the two inlet streams, which increases the degree of atrial mixing and viscous dissipation, resulting in additional fluid energy losses. Last, fluid contraction from the large right atrial chamber into the main pulmonary artery results in mechanical energy loss owing to flow separation at the wall of the main pulmonary artery.

Implications for exercise tolerance. Patients with the modified Fontan operation have been reported to have reduced cardiac output and a limited car-

diopulmonary response to exercise.^{20, 21} As demonstrated in this report, the rate of fluid energy loss through the right atrium increased with flow rate for all right atrial volumes, and this effect was especially pronounced at flow rates greater than 3 L/min. As the total venous return increases, the total energy per volume of fluid in each of the corresponding inlet and outlet vessels also increases. High-energy inferior and superior vena caval stream expansions and collision produce significant energy losses in the right atrial chamber. It is possible that these changes in energy may have significant implications regarding the performance of the circulation during moderate to high output states.

To fully understand the consequence of poor right atrial hemodynamics on the performance of the Fontan circulation, it is helpful to consider the individual components of the total resistance of the pulmonary circuit. These components include the resistance to flow through the pulmonary circulation proximal to the lungs and the pulmonary arteriolar resistance. The proximal circulation includes the right atrium, the atriopulmonary anastomosis, and flow through each of the branch pulmonary arteries. The principal site of resistance in the pulmonary circulation is the pulmonary vascular bed (ideally <2 Wood units). Under resting conditions, the resistance proximal to the pulmonary vascular bed is very small. However, this study has shown that an important increase in resistance proximal to the pulmonary vascular bed is present at high right atrial volumes and at higher flow rates, as would occur during exercise. Although the reasons for limited exercise tolerance in patients who have had the Fontan operation are complex,^{20, 21} it remains plausible that increases in resistance to flow in the giant right atrium may contribute to the limited cardiac output that is seen in these patients.

Implications for the timing of APC to TCPC conversion. Besides cardiac transplantation, there are few therapeutic options for patients with failing APCs. A recently proposed option for the management of these patients, however, is conversion to a lateral tunnel TCPC.²² The goal of this technique is to eliminate the pressure load on the right atrium, improve hemodynamics, and reduce the risk of supraventricular arrhythmias and right atrial thrombus formation. Although the efficacy of this approach has been demonstrated in a small number of patients in recent publications,^{22, 23} the clinical criteria for, and timing of, conversion from APC have

not been established. Results from this study demonstrate that right atrial volume is an important determinant of the efficiency of the APC circulation and suggest that at physiologic flow rates, right atrial volume reduction by conversion to lateral tunnel TCPC may significantly reduce fluid energy losses and lower vena caval pressures. Ultimately, it will be necessary to document this improved efficiency and exercise tolerance in patients where total blood volume is increased and may also contribute to late failure of the Fontan procedure.

Conclusions

Fluid flow through a dilated right atrial chamber causes fluid energy dissipation, which contributes to poor flow dynamics in the APC. The rate of energy losses increased with increasing right atrial size and flow rate, resulting in increased energy requirements to move blood from the venae cavae to the pulmonary arteries. These results may help explain the progressive nature of APC failure and provide additional rationale for conversions from APCs to lateral tunnel TCPCs in selected patients.

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