Cardiol Young 2004; 14 (Suppl. 3): 32–37

ISSN 1047-9511

Original Article

A study of mathematical modelling of the competitions of flow in the cavopulmonary anastomosis with persistent forward flow

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Keywords: Congenital heart diseases; bidirectional cavopulmonary anastomosis; haemodynamics; computer model

Among the operations which lead to a partial rerouting of the systemic venous return into the pulmonary arteries, the bidirectional cavopulmonary anastomosis[1] is frequently utilised in the staged surgical management of patients with functionally univentricular hearts. Whereas some surgeons insist on closing any other source of pulmonary blood flow while performing a bidirectional cavopulmonary anastomosis, others maintain patency of either a stenosed native pulmonary outflow tract, or of a systemic-to-pulmonary arterial shunt. It remains controversial as to whether an additional source of pulmonary arterial blood flow can safely and usefully be left at the time of the bidirectional cavopulmonary anastomosis to increase systemic saturation, or whether it should be eliminated to reduce the volume load on the ventricle.[2]

From a hydraulic point of view, the flow of blood to the lungs may produce hypertension in the superior caval vein, and produce competition between the flow in the bidirectional cavopulmonary anastomosis and that to the lungs, thus causing dissipation of energy. The techniques of computational fluid dynamics techniques,[3,4] have now been used to study local fluid dynamics created by surgical repairs. We have applied the finite element method to three-dimensional models of the bidirectional cavopulmonary anastomosis with additional pulmonary arterial blood flow.[2,5] Similar models, although allowing a very detailed description of the local haemodynamics, could not describe the effect of variations of parameters such as pulmonary and vascular resistance on the global haemodynamics, namely pressures and flows in the systemic and venous circuits.

We describe herein a simplified one-dimensional lumped parameter model we have developed to represent the circulatory system as a hydraulic network composed of resistive, inert, and compliant elements, as well as non-linear resistance components that incorporate losses of energy in the region of the caval connection of a bidirectional cavopulmonary anastomosis.

Materials and methods

We used one-dimensional lumped parameter models[6] of double outlet ventricle with pulmonary stenosis, absent right atrioventricular connection, and atrial septal defect before and after construction of a bidirectional cavopulmonary anastomosis.The models were divided into three regions: the heart, the systemic circulation, and the pulmonary circulation. The hydraulic network of the model is illustrated in Figure 1.

Each circulatory region was divided into several compliant districts: five for the systemic circulation, and seven for the pulmonary circulation. The hydraulic behaviour of each district was mathematically modelled on the basis of the laws of conservation of mass and momentum, which lead to two differential equations where the dependent variables are the volume of rate of flow, represented as "Q(t)" and the pressure of the flow, represented as "P(t)", both being functions of time.[7]

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Accepted for publication January 2004

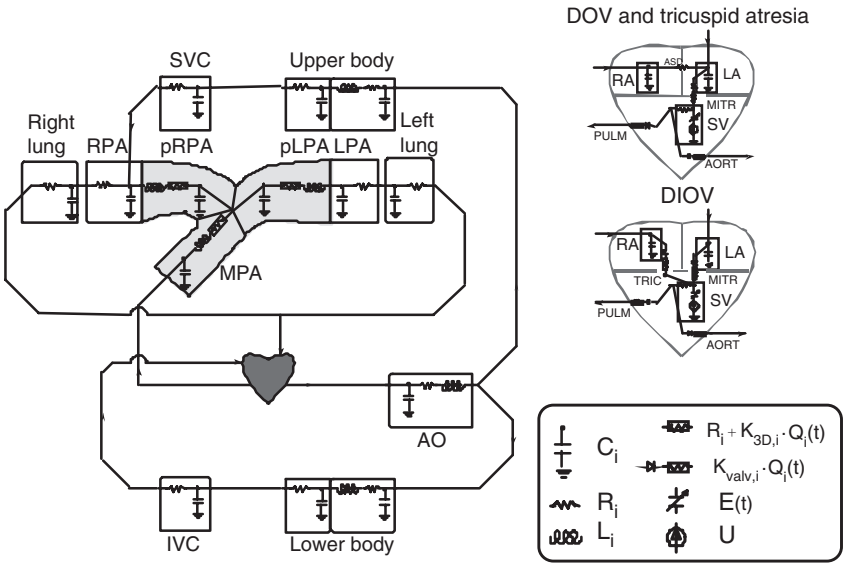

Figure 1.
Hydraulic network of the postoperative model with the bidirectional cavopulmonary anastomosis. Arrows indicate the normal direction of flow. The values of the parameters in the shaded area are evaluated from three-dimensional mathematical models of the connection (see text). AO: ascending aorta; AORT: aortic valve; ASD: atrial septal defect; DOV: double outlet ventricle; IVC: inferior vena cava; LA: left atrium; LPA: left pulmonary artery; MITR: mitral valve; MPA: pulmonary trunk; pLPA and pRPA: proximal left and right pulmonary artery; PULM: pulmonary valve; RA: right atrium; RPA: right pulmonary artery; SV: functionally single ventricle; SVC: superior caval vein; TRIC: tricuspid valve.

Table 1. Data for the typical preoperative situation.

BSA* (m^2)	0.6
HR* (bpm)	100
PAR* (Wood units)	2.2
SVR* (Wood units)	21.7
ΔP_{syst} (mmHg)	35
Q_p (l/min)	2.22
Q_s (l/min)	1.55
Q_p/Q_s (−)	1.68
P_{SVC} (mmHg)	8.6
P_{RA} (mmHg)	7.3
P_{LA} (mmHg)	7.0
P_{AO} (mmHg)	41

Abbreviations: BSA: body surface area; HR: heart rate; P_{AO}: mean aortic pressure; P_{LA}: mean left atrium pressure; P_{RA}: mean right atrium pressure; P_{SVC}: mean superior caval venous pressure; Q_p and Q_s: mean pulmonary and systemic blood flow rate; ΔP_{syst}: systolic pressure gradient across the pulmonary outflow obstruction; PAR: pulmonary arteriolar resistance; SVR: systemic vascular resistance.
*These parameters are input data for the model, while the remaining are results of the model

The values of the input parameters

The model allows us to evaluate the preoperative and postoperative haemodynamics, with us setting the value of the systemic vascular resistances, the pulmonary arteriolar resistances, the heart rate, and the degree of obstruction in the pulmonary outflow tract on the basis of the drop in systolic pressure between the functionally single ventricle and the pulmonary arteries.

Table 1 shows some haemodynamic data calculated by means of the preoperative model with reference to systemic vascular resistance and pulmonary arteriolar resistance equal to 21.7 and 2.2 Wood units, respectively, with the heart rate fixed at 100 beats per minute, and a pulmonary outflow obstruction of 50%, specifically with a pressure gradient of 35 millimetres of mercury.

The numerical simulations

Pulmonary arteriolar resistances ranging from 0.8 to 7.9 Wood units, and the severity of pulmonary outflow obstruction, were taken as independent variables. We then investigated models with 50%, 60%, 75% and 100% of reduction of cross-sectional area of the pulmonary outflow tract. The model with 100% reduction corresponds to pulmonary atresia, whereas those with lesser degrees of obstruction represent various degrees of supravalvar pulmonary stenoses comparable to a pulmonary arterial banding, leading to different pressure gradients, namely from 20 to 40, 40 to 50, and 50 to 60 millimetres of mercury, respectively. Using the model, we recorded, both in pre- and postoperative situations, the pressure in the superior caval vein, the ratio of pulmonary-to-systemic flows, and the cardiac output.

From the postoperative haemodynamic data, in particular the systemic flow and the ratio of flows, it proved possible to calculate the systemic arterial oxygen saturation and the availability of systemic arterial oxygen.[6] The equation system which describes the model consists of 24 differential and 19 algebraic equations. We used an interactive programme for simulating dynamic systems (SIMNON, SSPA System, Lund, Sweden), and adopted the Runge-Kutta algorithm of order 4/5 to solve the equation system. Computations were carried out on a HP486/33XM personal computer with 16 megabytes of random access memory. The time required to compute a typical simulation was less than 1 minute.

https://doi.org/10.1017/S1047951104006535 Published online by Cambridge University Press

Table 2. Haemodynamic results in postoperative models as functions of pulmonary arteriolar resistance and severity of the pulmonary outflow obstruction.

PAR (Wood units)	Degree of stenosis in pulmonary outflow tract (%)	0.8	2.2	4.9	7.9
P_{SVC} (mmHg)	50	9.6	12.0	16.0	19.2
	60	9.3	11.5	15.1	18.2
	75	8.9	10.9	14.2	17.1
	100	8.2	9.2	11.1	12.9
CO (l/min)	50	3.44	3.24	2.89	2.60
	60	3.17	2.99	2.71	2.46
	75	2.89	2.74	2.51	2.31
	100	2.03	1.97	1.88	1.80
Q_p/Q_s	50	1.73	1.69	1.59	1.50
	60	1.46	1.43	1.36	1.29
	75	1.21	1.19	1.15	1.10
	100	0.50	0.49	0.47	0.46
SAOA (ml oxygen/ min)	50	223	208	186	166
	60	228	213	191	171
	75	232	217	194	173
	100	221	209	188	172
Sat_{art} (%)	50	82	81	79	77
	60	81	79	77	75
	75	78	77	75	72
	100	62	60	57	54

Abbreviations: CO: total ventricular outflow; P_{SVC}: mean superior caval venous pressure; PAR: pulmonary arteriolar resistance; Q_p and Q_s: mean pulmonary and systemic blood flow rate; SAOA: systemic arterial oxygen availability; Sat_{art}: percent arterial blood saturation

Results

The effects of different values of pulmonary arteriolar resistance equal to 0.8, 2.2, 4.9 and 7.9 Wood units in the presence of various degrees of obstruction in the pulmonary outflow tract are shown in Table 2, presenting the mean values of pressure in the superior caval vein, cardiac output, and ratios of flows for the various postoperative models. Systemic arterial oxygen availability and saturation, calculated using a pulmonary venous saturation of 95%, and assuming systemic consumption of oxygen of 120 millilitres per minute, are reported in the same table. As pulmonary arteriolar resistance increases, the cardiac output and the ratio of flows decrease, while the mean pressure increases monotonically in the superior caval vein.

The effects of changes in the severity of obstruction in the pulmonary outflow tract, and pulmonary vascular resistance, on the predicted postoperative pressure in the superior caval vein, the systemic flow, and the ratio of flows are represented diagrammatically in Figure 2. The pulmonary arteriolar resistance is plotted along the horizontal axis, while three intervals are defined along the vertical axis, which correspond to increasing preoperative drops in pressure across the stenosis. Each interval was assumed to represent a class of pulmonary obstruction. Mild stenosis was deemed present with a gradient from 20 to 40 millimetres of mercury, moderate stenosis from 40 to 50, and severe stenosis from 50 to 60 millimetres of mercury. Strictly speaking, it should be noted that the pressure gradient across the stenosis depends upon both the severity of the obstruction and the volume of flow through it, which in turn depends upon the pulmonary afterload combined with the efficiency of the ventricular pump.

The upper margin of the diagram represents the model with absence of forward flow. The map can be used to predict surgical outcomes, in particular the pressure in the superior caval vein, from preoperative catheterisation data. This shows that a low superior caval venous pressure should be obtained to avoid central venous hypertension. Furthermore, in the map it is possible to localise areas of high or low ratios of pulmonary-to-systemic flow, as well as areas of different systemic flow, as functions of the values of the pulmonary arteriolar resistance and the systemic pressure. The combination of the ratio of flows with systemic flow determines the value of systemic arterial oxygenation and the arterial saturation, and hence the suitability for a cavopulmonary anastomosis. In principle, ratios of flow close to unity, together with a high systemic flow, could give good oxygenation. The white area in Figure 2 represents the region where ratios of flow range between 0.75 and 1.25, and the mean superior caval venous pressure is lower than 15 millimetres of mercury. In this area, systemic flow has values between 1.5 and 2.0 litres per minute. If, for example, the value of pulmonary arteriolar resistance is equal to 6 Wood units, the forward flow entering the area should be reduced by severe banding, giving an optimal surgical outcome.

A clinical example

The patient analysed had double inlet left ventricle, discordant ventriculo-arterial connections, atrial septal defect, subaortic stenosis, right atrioventricular valvar regurgitation, a small left atrioventricular valve, and a banded pulmonary trunk, the banding producing a gradient of 30 millimetres of mercury. The preoperative cardiac catheterisation data are shown in Table 3. The pulmonary arteriolar resistance was 14.89 Wood units, while the systemic vascular resistance was 23.5 Wood units. The patient underwent a bidirectional cavopulmonary anastomosis and, at the end of the operation, the pressure in

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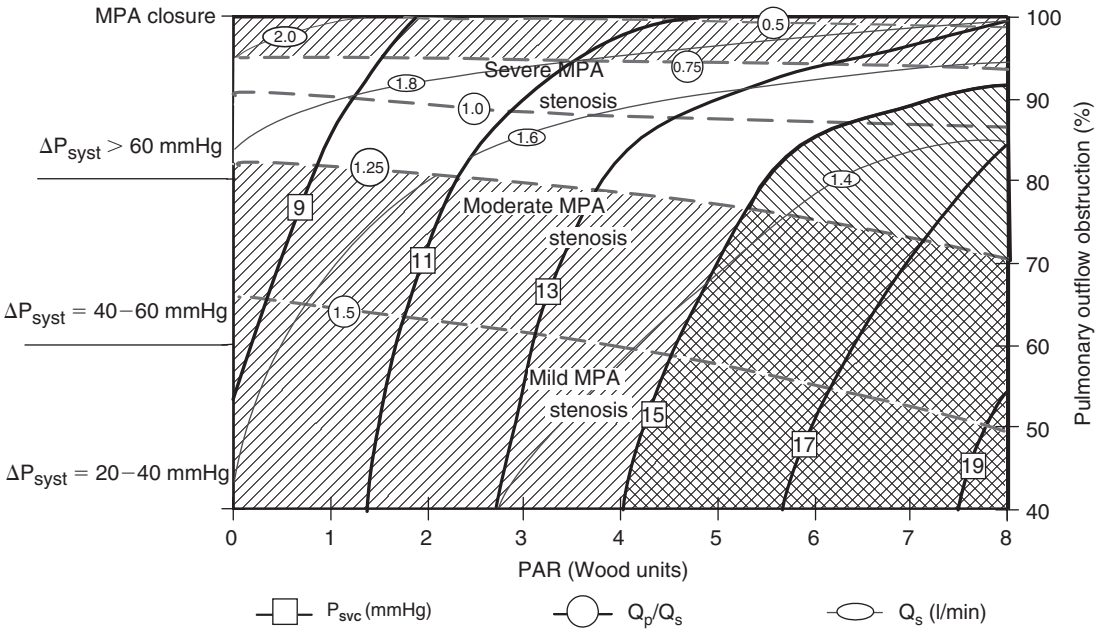

Figure 2.
Map with areas corresponding to different possible surgical outcomes after the bidirectional cavopulmonary anastomosis as functions of the pulmonary arteriolar resistances (PAR) and the preoperative systolic pressure gradient across the pulmonary outflow obstruction (ΔP_{syst}). The latter is divided into three intervals, roughly corresponding to a mild stenosis (ΔP_{syst} = 20–40 millimetres of mercury), a moderate stenosis (ΔP_{syst} = 40–50 millimetres of mercury) and a severe stenosis (ΔP_{syst} = 50–60 millimetres of mercury). See text for explanation. The white area indicates the optimal surgical outcome, with a postoperative ratio of flows in a range between 0.75 and 1.25, and a postoperative superior caval venous pressure lower than 15 millimetres of mercury. The darkest shaded area represents the postoperative conditions with caval venous pressure greater than 15 millimetres of mercury, and a ratio of flows outside the above range, simultaneously. MPA: mean pulmonary artery; P_{SVC}: mean superior caval venous pressure; Q_p: mean pulmonary blood flow rate; Q_s: mean systemic blood flow rate.

Table 3. Comparison between simulated and clinical data for the patient simulated (BSA = 0.5 m^2, weight = 13.1 kg, pulmonary arteriolar resistances = 14.9 Wood units, systemic vascular resistances = 23.5 Wood units, HR = 176 bpm, ΔP_{syst} = 30 mmHg, oxygen capacity = 20.4 ml oxygen/100 ml blood, body oxygen consumption = 95 ml oxygen/min). SAOA and Sat_{art} were calculated assuming Sat_{PV} = 95%. Reported pressure values are the highest and the lowest in the cardiac cycle, except for superior caval vein.

	Catheterisation report	Preoperative simulation	Glenn simulation	Unidirectional simulation
Q_p/Q_s (–)	1	0.99	1.31	1.14
Q_s (l/min)	2.2	1.61	1.09	1.34
P_{SV} (mmHg)	9/51	4/62	3/56	3/63
P_{MPA} (mmHg)	16/21	25/31	23/28	34/39
P_{SVC} (mmHg)	N/A	7.9	25.4	17.3
SAOA (ml oxygen/min)	–	–	175	218
Sat_{art} (%)	–	–	79	80

Abbreviations: N/A: not available; BSA: body surface area; HR: heart rate; Q_p and Q_s: mean pulmonary and systemic blood flow rate; P_{SVC}: mean superior caval venous pressure; P_{MPA}: mean pulmonary artery pressure; P_{SV}: univentricular pressure; SAOA: systemic arterial oxygen availability; Sat_{art}: percent arterial blood saturation

the superior caval vein was 25 millimetres of mercury. This value was thought to be too high, and therefore the proximal right pulmonary artery was occluded, producing a unidirectional classical Glenn anastomosis. The results of the simulations are also shown on Table 3. First, the preoperative situation was reproduced, setting in the model the pulmonary arteriolar resistance, systemic vascular resistance, heart rate, and the severity of obstruction within the pulmonary outflow tract according to the catheterisation data. We then simulated the bidirectional cavopulmonary anastomosis. This produced a clear correlation between the superior caval venous pressure predicted from the simulation and the value

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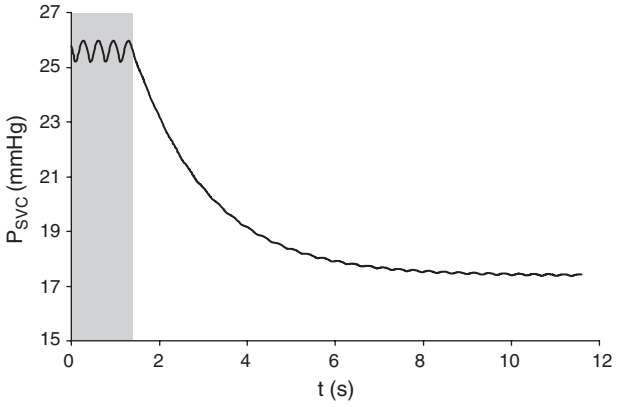

Figure 3.
The simulated pressure tracing in the superior caval vein (P_{SVC}) for a specific patient (see text). It shows the effect of occluding the proximal right pulmonary artery when constructing unidirectional classical Glenn anastomosis.

measured at the end of the operation. Comparing this situation with the map of Figure 2, it can be seen that this patient does not enter the white area, whatever the level of banding, because the pulmonary arteriolar resistance is too high. In the last simulation, which takes into account the changes produced by the surgeon during the operation, we reproduced the occlusion of the right pulmonary artery. In this way, blood from the superior caval vein perfuses the right lung, and the blood from the pulmonary trunk feeds the left lung. The results (Fig. 3) show that the superior caval venous pressure suddenly decreases, as was observed in the operating theatre.

Discussion

Our model describes the entire circulatory system as a closed loop. This allows us to evaluate the mutual interactions among all the involved haemodynamic variables. Barnea et al.[7] theoretically investigated the effect of the distribution of the flow of blood between the systemic and pulmonary circulations on systemic delivery of oxygen in neonates with hypoplastic left heart syndrome. They found a relation between the ratio of pulmonary-to-systemic flow and the systemic arterial oxygen availability for different values of cardiac output, fixed saturations of pulmonary venous oxygen, and metabolic consumption of oxygen. Their model, however, does not describe how the values for the ratio of flows, cardiac output, and pulmonary venous saturation can be achieved.

The major drawback of our approach is the need to use many parameters to characterise the resistance, the inertia, and the compliance of the districts of the hydraulic network. Evaluating these aspects proved to be quite a troublesome task, due to the impossibility of measuring those data during clinical investigations and procedures, except for the pulmonary arteriolar resistance and the systemic vascular resistance. In spite of these limitations, the results from our simulations are in a fairly good agreement with clinical observations.[8–10]

Although until now we have been able to provide only a limited clinical validation of the model, we believe that its use is already valuable in the comprehension of global haemodynamics in patients submitted to the bidirectional cavopulmonary anastomosis. In particular, our simulations led to the definition of an optimal set of haemodynamic variables which depend upon the circulatory parameters of the patient. It should be noted, nonetheless, that the developed model was used in this setting to investigate the behaviour of the bidirectional cavopulmonary anastomosis exclusively in treating patients with functionally univentricular hearts. The flexibility of the model, and the fact that it can be used on personal computers, could allow quantitative investigations of further cardiac congenital malformations and/or extensive surgical reconstructions, such as the atriopulmonary and the total cavopulmonary connections.

As regards the clinical use of the model as a tool to predict the haemodynamic outcome of surgery, it should be borne in mind that the current version only takes into account hydraulic phenomenons. Indeed, its major limitation may be the lack of consideration of any autoregulative mechanisms, which generally operate when the equilibrium of a biological system is disturbed. The model deals with the situation characterised by severe pathological disturbances, and it appears reasonable to assume that such mechanisms have already failed, at least in part. The Frank-Starling mechanism that relates end-diastolic volume of the ventricle to stroke volume, nonetheless, remains intact. The model also does not take into account the effects of respiration on the flows in the caval veins.

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