BMTE 03.04

In vitro analysis of the fluid dynamics in the superior vena cava – pulmonary artery anastomosis with an additional systemic to pulmonary shunt

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Abstract

Babies with one pumping ventricle need a series of medical and surgical procedures in order to have an adequate blood flow to the lungs and consequently to survive and grow. The Fontan operation is usually the final stage of surgeries and isolates the systemic and pulmonary circulation. This study focuses on the second stage of surgeries, in which the superior vena cava (SVC) is connected directly to the pulmonary artery (PA). To improve the pulmonary perfusion, it is suggested to connect an additional systemic to pulmonary shunt (SPS) to the SVC-PA anastomosis, which should work as an ejector. The SPS as an ejector is regarded in this report.

The objective is to investigate the fluid dynamics in different shunt models. In shunt model 1, the SPS is connected at an angle of 45° and in shunt model 2 the SPS is connected perpendicularly to the left pulmonary artery. Two approaches were applied: in vitro experiments and computational fluid dynamics (CFD) simulations. The influence of the SPS flow on the flow in the SVC \(Q_{svc}\), the flow distribution in the lungs and the pressure in the SVC was determined at three different conditions: continuous SPS flow with rigid and with distensible shunt models and pulsatilie SPS flow with rigid shunt models. At continuous condition, also the influence of the pulmonary resistance and the upper body pressure is measured. With the CFD model, simulations based on the experiments with continuous SPS flow and rigid shunt models and simulations with increased pulmonary resistance were performed.

The experimental and computational results at each condition showed that with an increasing SPS flow the pressure in the SVC did not become too high (maximum increase of 13.5 %), but the total flow \(Q_{tot}\) in the pulmonary arteries did not increase much either (maximally 23.7%) with respect to the strong decrease of \(Q_{svc}\) (87.4%). Imposing a higher pressure in the SVC led to a higher flow in the SVC and a higher \(Q_{tot}\) and a high pulmonary resistance resulted in the opposite. The flow distribution appeared to be determined by the configuration of the shunt model and the pulmonary artery resistance. When the major part of the pulmonary resistance was defined after the pulmonary arteries, in SM1 a recirculation in the pulmonary arteries developed. With a higher resistance at the pulmonary arteries, the flow distribution between the right and left pulmonary artery was not influenced by the configuration anymore.

From these results, it can be concluded that the SPS did not work as an ejector. The increase of \(Q_{tot}\) was small, because \(Q_{svc}\) was blocked instead of entrained. The difference in angle and position of the SPS did not improve the effect of the ejector. Neither did the distensibility and the pulsatile flow through the SPS.
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<th>Explanation</th>
<th>Unit</th>
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<tbody>
<tr>
<td>A</td>
<td>Cross sectional area</td>
<td>m$^2$</td>
</tr>
<tr>
<td>a</td>
<td>Factor in flow-pressure relationship</td>
<td>mmHg min$^2$/l$^2$</td>
</tr>
<tr>
<td>b</td>
<td>Factor in flow-pressure relationship</td>
<td>mmHg min/l</td>
</tr>
<tr>
<td>C</td>
<td>Constant</td>
<td>l/m</td>
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<tr>
<td>CSA</td>
<td>Cross sectional area</td>
<td>m$^3$</td>
</tr>
<tr>
<td>D$_{sh}$</td>
<td>Shunt diameter</td>
<td>mm</td>
</tr>
<tr>
<td>D$_{vessel}$</td>
<td>Vessel diameter</td>
<td>mm</td>
</tr>
<tr>
<td>P</td>
<td>Pressure</td>
<td>mmHg</td>
</tr>
<tr>
<td>P$_{at}$</td>
<td>Pressure in the atrial reservoir</td>
<td>mmHg</td>
</tr>
<tr>
<td>PI</td>
<td>Average pulsatility index</td>
<td>-</td>
</tr>
<tr>
<td>P$_{in}$</td>
<td>Inlet pressure</td>
<td>mmHg</td>
</tr>
<tr>
<td>P$_{lpa}$</td>
<td>Pressure in the left pulmonary artery</td>
<td>mmHg</td>
</tr>
<tr>
<td>P$_{out}$</td>
<td>Outlet pressure</td>
<td>mmHg</td>
</tr>
<tr>
<td>P$_{rpa}$</td>
<td>Pressure in the right pulmonary artery</td>
<td>mmHg</td>
</tr>
<tr>
<td>P$_{svc}$</td>
<td>Pressure in the superior vena cava</td>
<td>mmHg</td>
</tr>
<tr>
<td>P$_{svc,0}$</td>
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</tr>
<tr>
<td>P$_{wk}$</td>
<td>Pressure in the windkessel (WK1)</td>
<td>mmHg</td>
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<tr>
<td>Q</td>
<td>Flow</td>
<td>l/min</td>
</tr>
<tr>
<td>Q$_{in}$</td>
<td>Inlet flow in the vessel</td>
<td>l/min</td>
</tr>
<tr>
<td>Q$_{lpa}$</td>
<td>Flow in the left pulmonary artery</td>
<td>l/min</td>
</tr>
<tr>
<td>Q$_{max}$</td>
<td>Maximum flow</td>
<td>l/min</td>
</tr>
<tr>
<td>Q$_{min}$</td>
<td>Minimum flow</td>
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<tr>
<td>Q$_{out}$</td>
<td>Outlet flow in the vessel</td>
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</tr>
<tr>
<td>Q$_{rpa}$</td>
<td>Flow in the right pulmonary artery</td>
<td>l/min</td>
</tr>
<tr>
<td>Q$_{sh}$</td>
<td>Flow in the systemic-to-pulmonary shunt</td>
<td>l/min</td>
</tr>
<tr>
<td>Q$_{svc}$</td>
<td>Flow in the superior vena cava</td>
<td>l/min</td>
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<tr>
<td>r</td>
<td>Radius</td>
<td>m</td>
</tr>
<tr>
<td>R$^2$</td>
<td>Regression coefficient</td>
<td>-</td>
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<tr>
<td>RI</td>
<td>Average resistance index</td>
<td>-</td>
</tr>
<tr>
<td>R$_{in}$</td>
<td>Resistance in first part vessel</td>
<td>mmHg min/l</td>
</tr>
<tr>
<td>R$_{lpa}$</td>
<td>Clamp resistance left pulmonary artery</td>
<td>mmHg min/l</td>
</tr>
<tr>
<td>R$_{out}$</td>
<td>Resistance in last part vessel</td>
<td>mmHg min/l</td>
</tr>
<tr>
<td>R$_{pa}$</td>
<td>Resistance of the pulmonary arteries</td>
<td>mmHg min/l</td>
</tr>
<tr>
<td>R$_{rpa}$</td>
<td>Clamp resistance right pulmonary artery</td>
<td>mmHg min/l</td>
</tr>
<tr>
<td>R$_{TPR}$</td>
<td>Total pulmonary resistance</td>
<td>mmHg min/l</td>
</tr>
<tr>
<td>v</td>
<td>Velocity</td>
<td>m/s</td>
</tr>
<tr>
<td>v$_{sh}$</td>
<td>Velocity in the systemic-to-pulmonary shunt</td>
<td>m/s</td>
</tr>
<tr>
<td>w</td>
<td>Mass flow</td>
<td>kg/s</td>
</tr>
<tr>
<td>α</td>
<td>Factor for the cross sectional area</td>
<td>-</td>
</tr>
<tr>
<td>β</td>
<td>Factor for the velocity</td>
<td>-</td>
</tr>
<tr>
<td>γ</td>
<td>Permeability porous jump</td>
<td>m$^2$</td>
</tr>
<tr>
<td>Δm</td>
<td>Thickness porous jump</td>
<td>m</td>
</tr>
<tr>
<td>θ</td>
<td>Angle</td>
<td>radians</td>
</tr>
<tr>
<td>μ</td>
<td>Dynamic viscosity</td>
<td>cP</td>
</tr>
<tr>
<td>ρ</td>
<td>Density</td>
<td>kg/m$^3$</td>
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<th>Explanation</th>
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<tbody>
<tr>
<td>AR</td>
<td>Atrial reservoir</td>
</tr>
<tr>
<td>BDG</td>
<td>Bi-directional Glenn shunt</td>
</tr>
<tr>
<td>CFD</td>
<td>Computational fluid dynamics</td>
</tr>
<tr>
<td>CP</td>
<td>Centrifugal pump</td>
</tr>
<tr>
<td>DSM</td>
<td>Distensible shunt model</td>
</tr>
<tr>
<td>FR</td>
<td>Fluid reservoir</td>
</tr>
<tr>
<td>FVM</td>
<td>Finite Volume Method</td>
</tr>
<tr>
<td>HPR</td>
<td>High pulmonary resistance</td>
</tr>
<tr>
<td>IVC</td>
<td>Inferior vena cava</td>
</tr>
<tr>
<td>LPA</td>
<td>Left pulmonary artery</td>
</tr>
<tr>
<td>NPR</td>
<td>Normal pulmonary resistance</td>
</tr>
<tr>
<td>PA</td>
<td>Pulmonary artery</td>
</tr>
<tr>
<td>PR</td>
<td>Porous resistance</td>
</tr>
<tr>
<td>PC</td>
<td>Polycarbonate</td>
</tr>
<tr>
<td>PVC</td>
<td>Polyvinyl chloride</td>
</tr>
<tr>
<td>RP</td>
<td>Rollerpump</td>
</tr>
<tr>
<td>RPA</td>
<td>Right pulmonary artery</td>
</tr>
<tr>
<td>SM</td>
<td>Shunt model</td>
</tr>
<tr>
<td>SPS</td>
<td>Systemic-to-pulmonary shunt</td>
</tr>
<tr>
<td>SR</td>
<td>Shunt resistance</td>
</tr>
<tr>
<td>SVC</td>
<td>Superior vena cava</td>
</tr>
<tr>
<td>TCPC</td>
<td>Total cavo-pulmonary connection</td>
</tr>
<tr>
<td>VAD</td>
<td>Ventricular assist device system</td>
</tr>
<tr>
<td>VSD</td>
<td>Ventricular septum defect</td>
</tr>
<tr>
<td>WK</td>
<td>Windkessel</td>
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1 Introduction

1.1 The clinical problem

There is a group of patients who have only one effective ventricle instead of two. The commoner abnormalities in this group are one of the following:
- A single ventricle pumps blood into both great arteries (the aorta and the pulmonary artery).
- Two ventricles are present but one is too small to perform its normal function.
- The connections between the atria and ventricles are unsuitable for an operation which separates the ventricles into two effective chambers which would then pump blood into the appropriate great arteries.

These abnormalities can occur with tricuspid atresia, hypoplastic right or left heart and other types of single ventricle diseases. Survival of the patients depends on the relative degree of pulmonary blood flow. Patients with a diminished pulmonary flow present with varying degrees of cyanosis. Patients with an increased pulmonary blood flow present with congestive heart failure.

Babies with one pumping chamber need a series of medical and surgical procedures in order to have adequate blood flow to the lungs and consequently to survive and grow. The Fontan procedure is usually the final stage of surgeries and isolates the systemic and pulmonary circulation. It provides a normal systemic arterial oxygen saturation without volume overloading of the working ventricle.

Figure 1.1, The modified Blalock-Taussig shunt, a tube made of Gore-Tex is stitched between the innominate artery and pulmonary arteries. ia: innominate artery; pa: pulmonary arteries; st: shunt tunnel [Pennati, 2001].

The first surgical procedure is usually undertaken either in the new-born period or in the first few months of life. Depending on the underlying problem, it may be a shunt operation (for example the Blalock-Taussig shunt, figure 1.1) in order to increase the pulmonary blood flow [de Leval, 1981], or it may involve banding of the pulmonary artery (PA) in order to decrease the flow.
The second procedure, the bi-directional Glenn shunt (BDG), is performed between the ages of four months and one year (figure 1.2). The superior vena cava (SVC) is connected directly to the pulmonary artery. As a result, venous blood from the head and upper limbs will pass directly to the lungs, bypassing the underdeveloped ventricle. However, the venous blood from the lower half of the body will continue to return to the heart. Post-operatively, oxygen saturation is improved. While avoiding the risk of failure of a complete Fontan operation, such an operation helps to relieve symptoms. The procedure decreases the volume of blood delivered to the single ventricle, thus reducing the amount of work the ventricle must perform. In addition, because the BDG is a low-pressure shunt, it does not carry the risk of causing thickening and hardening of the blood vessels of the lungs. This operation creates a more favourable setting in which to complete a Fontan reconstruction.

The final procedure, the Fontan procedure, is usually not undertaken in a child below the age of around three years. The inferior vena cava (IVC) is connected to the pulmonary artery by creating a channel or baffle through the heart or bypassing the heart to direct its flow to the pulmonary artery. The Fontan operation can be done in several ways. This depends both on the patient's specific situation and the preferences of the surgeon. As an example the total cavo-pulmonary connection with a hypoplastic left heart is shown in figure 1.3.
Analysis of the fluid dynamics in the SVC – PA anastomosis with an additional SPS

Figure 1.3, The total cavo-pulmonary connection with a hypoplastic left heart. The systemic and pulmonary circulation are separated [Royal Children’s Hospital, 2002].

The venous return is redirected to the pulmonary artery and into the lungs by joining the superior vena cava and the inferior vena cava by a large patch that creates a 'tunnel' through the right atrium. The venae cavae are then connected directly to the pulmonary arteries above the heart. The atrial septum is removed. Blood returns via the pulmonary veins in the left atrium. It flows in the ventricle, the ventricle pumps the blood into the aorta and it flows to the rest of the body.

Two other examples, the double outlet right ventricle and tricuspid atresia, are shown in appendix A [Michler, 1986; Castañeda, 1994; Yamada, 2000].

This study focuses on the second stage of surgeries. Some patients suffer from cyanosis after the placement of the BDG. It has already been mentioned that the first surgical procedure towards a Fontan circulation in case of a diminished pulmonary flow is a systemic-to-pulmonary shunt (SPS) operation, usually the Blalock-Taussig shunt. During the BDG construction the SPS is usually closed. The flow in the pulmonary arteries of the cyanosed patients after the BDG construction has to be increased. However, it is important that this is done without increasing the upstream pressure too much, because pressures in the vena cava higher than about 15 mmHg result in the formation of oedema [Robbins, 1994]. A possible solution could be an additional SPS, like the Blalock-Taussig shunt or a SPS in another configuration. The idea is that this SPS will work as an ejector. This is treated in §1.2.

Figure 1.4, The ejector: a pressure drop through an aperture increases the velocity on the discharge side without increasing much the pressure on the inlet side.
1.2 Rationale and preliminary analysis

Ejectors operate on the principle of a pressure drop through an aperture, which increases velocity on the discharge side (figure 1.4). The operating medium (liquid, steam or gas) enters the inlet under pressure and travels through the nozzle into the suction chamber. The nozzle converts the pressure of the operating medium into a high velocity stream, which passes from the discharge side of the inlet nozzle. Pumping action starts when the liquid, vapour or gases in the suction chamber are entrained by the high velocity stream emerging from the inlet nozzle, lowering the pressure in the suction chamber. This results in the liquid, gas or vapour in the suction chamber to flow towards the discharge. The advantage of an ejector is that the velocity on the discharge side is increased without increasing much the pressure on the inlet side. Figure 1.4 shows an example of an ejector in an industrial application. Figure 1.5 shows a schematic diagram of a liquid-liquid ejector in a (simplified) physiological set-up.

![Figure 1.5. Liquid-liquid ejector.](image)

Regarding this ejector, calculations are made to determine the $Q_{sh} - Q_{in}$ relationship. In surface 1 two currents enter. One current comes from the shunt ($Q_{sh}$), with velocity $v_{sh}$ and cross sectional area $\alpha A_1$ and the other origins at the inlet from the large vessel ($Q_{in}$) and has a velocity $\beta v_{sh}$ and cross sectional area $(1-\alpha)A_1$. In surface 2 at a certain distance from surface 1 the currents are fully mixed and the velocity is practically uniform, $v_2$. The motion is turbulent and the velocity profile is assumed to be completely flat. After surface $A_2$, $Q_{sh} + Q_{in} (= Q_{out})$ flows through a part of the vessel with resistance $R_{out}$ and discharges through the outlet. The vessel from the inlet until surface $A_1$ has a resistance $R_{in}$ and the resistance of the vessel part between $A_1$ and $A_2$ is neglected. The only surface force is due to the pressure. Regarding mass balance and momentum balance three equations are stated (see Appendix B):

\begin{align}
    P_2 - P_1 &= \rho v_{sh}^2 \left( \alpha^2 + \beta^2 - 2\alpha \beta \right) \\
    P_2 &= P_{out} + R_{out} (Q_{in} + Q_{sh}) \\
    Q_{in} &= \frac{P_{in} - P_{1}}{R_{in}}
\end{align}

with $P_1$ and $P_2$: the pressures at surface $A1$ and surface $A2$ respectively, $P_{in}$: the pressure at the inlet side of the vessel, $P_{out}$: the pressure at the outlet side. For $P_{in}$, $P_{out}$, $R_{in}$, $R_{out}$, $D_{sh}$ and $D_{ves}$ (the shunt and vessel diameter respectively), the values according to table 1.1 were taken to approximate physiological values. $Q_{sh}$ is the input for the ejector model. Equations 1.1-3 are solved for the three unknowns: $P_1$, $P_2$ and $Q_{in}$, so that $Q_{in}$ and $Q_{out}$ can be determined as a function of $Q_{sh}$.

<table>
<thead>
<tr>
<th>$P_{in}$ (mmHg)</th>
<th>$P_{out}$ (mmHg)</th>
<th>$R_{in}$ (mmHg*min)/l</th>
<th>$R_{out}$ (mmHg*min)/l</th>
<th>$D_{sh}$ (mm)</th>
<th>$D_{ves}$ (mm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>10</td>
<td>3.67</td>
<td>0.1</td>
<td>10</td>
<td>5</td>
<td>10</td>
</tr>
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</table>

Table 1.1, Input values for the ejector model.
The relations of $Q_{in}$ versus $Q_{sh}$ and of $Q_{tot}$ versus $Q_{sh}$ are depicted in figure 1.6.

Figure 1.6, Results for the simplified physiological ejector model (figure 1.5). The flow at the inlet from the vessel ($Q_{in}$) and the outlet flow ($Q_{out}$) are plotted versus the shunt flow ($Q_{sh}$).

According to this model the results with the ejector are poor, but the in vivo situation is more complicated. In a more physiological configuration, the effect of the ejector is more difficult to predict. The position and the angle of the shunt regarding to the vessel will have their influences on the efficiency of the ejector. There will be influences of other factors, like the distensibility of the materials, the pulsatile flow through the SPS and the influence of the size of the anastomosis made by the surgeon. For example, if the size and the shape of the distensible anastomosis are dependent on the local pressure, the fluid dynamics are influenced. Therefore, it is chosen to do in vitro experiments.

The objective of this study is to investigate the fluid dynamics in the shunt models. Two shunt models are tested. The shunt models consist of the SVC connected to the pulmonary arteries, at which a SPS is connected. In shunt model 1 (SM1), the SPS is connected directly at an angle of 45° to the left pulmonary artery and in shunt model 2 (SM2), the SPS is connected perpendicularly to the LPA at a distance of 1.8 cm from the anastomosis between the superior vena cava and the pulmonary artery (SVC-PA anastomosis).

Two approaches were applied: in vitro experiments and computational fluid dynamics (CFD) simulations. The influence of the SPS flow on the flow in the SVC ($Q_{svc}$), the flow distribution in the lungs and the pressure in the SVC was determined at three different conditions: continuous SPS flow with rigid and with distensible shunt models and pulsatile SPS flow with rigid shunt models. At continuous condition, also the influence of the pulmonary resistance and the upper body pressure is measured. With the CFD model, simulations based on the experiments with continuous SPS flow and rigid shunt models and simulations with increased pulmonary resistance were performed.
1.3 Report structure

This report is organised as follows. First, the experimental set-up of the mock simulator, the shunt models and the experimental protocol are treated in chapter 2. Before that the fluid dynamic behaviour of the shunt models could be investigated, preliminary tests had to be performed to obtain proper pulmonary resistances. After these preliminary experiments, two rigid shunt models were investigated. At continuous conditions the pressure and flow in the SVC and PA were measured at different flow rates through the SPS at different pressures in the SVC ($P_{\text{svc, 0}}$) and with two different pulmonary resistances. These measurements were partly repeated with distensible shunt models and with rigid shunt models at pulsatile conditions.

A CFD model is used to have a closer look at the local fluid dynamics in the shunt models. The results of the experiments and the CFD model are presented in chapter 3. Finally the results and the assumptions are discussed and the conclusions are summarised.
2 Materials and methods

2.1 Set-up of the hydraulic mock loop

2.1.1 Continuous condition
The model set-up (figure 2.1) includes two different circuits. The main circuit consists of a fluid reservoir (FR), which represents the upper part of the body. It is fed by a rollerpump (RP) (Stöckert® Instruments, München, Germany). The purpose of the fluid reservoir is to dampen the “ripple flow” of the peristaltic rollerpump and to deliver a steady flow to the shunt model. The height of the fluid reservoir is variable to supply the shunt model with different upstream heads. The fluid reservoir flows out in a tube, which represents the superior vena cava (SVC). The tubes are called after what they represent in a physiological situation. The vena cava is directly connected to the pulmonary artery (PA). This connection, the superior vena cava-pulmonary artery anastomosis (SVC-PA-anastomosis) and the systemic-to-pulmonary shunt (SPS) form the shunt model (SM). The fluid flows out of the shunt model through two tubes of equal length representing the left and right pulmonary artery (LPA and RPA), joining in one tube at a T-connection leading through a 1-liter windkessel (WK1). The windkessel functions as a pulmonary compliance in case of a pulsatile flow. After the windkessel the fluid flows through the pulmonary resistance (normal resistance NPR or high resistance HPR) and discharges into the atrial reservoir (AR), which is connected to the rollerpump. The fluid reservoir contains an inlet, outlet and overflow section (figure 2.2). A wall to minimize vortices in the outlet separates the inlet and outlet sections. These vortices are caused by the high inlet flow velocities. The function of the overflow section is to maintain a constant pressure level. The overflow section is connected to the atrial reservoir.

At continuous conditions, the second circuit consists of a centrifugal pump (CP) (Medtronic Bio-Medicus®, 550 Bio-control®, Minneapolis, MN, USA) which supplies the SPS with a constant flow.

The resistance of the pulmonary arteries (fixed) and the porous resistances (PR) (variable) define the pulmonary resistance. The desired pulmonary resistance values are obtained by properly setting two different porous resistances. The porous resistances consist of two tubes which contain a porous medium, a sponge.

The lines are realised with ½-inch-ID PVC pipes and PC connectors.

The pressures are measured with a manometer (accuracy of ±1 mmHg). The positions of pressure measurements are in the superior vena cava (P_{svc}), the right pulmonary artery (P_{rpa}), the atrial reservoir (P_{ar}) and the windkessel (P_{wk}). Fluid flow rate is measured immediately upstream of the shunt model (Q_{sh}), in the superior vena cava (Q_{svc}) and in the right pulmonary artery (Q_{rpa}) with an ultrasonic transit time flowmeter (H110, Transonic Systems Inc., Ithaca, NY, USA).
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Figure 2.1. The schematic hydraulic mock loop set-up at continuous conditions. CP: centrifugal pump, WK1: windkessel, PR: porous resistances, AR: atrial reservoir, RP: rollerpump, FR: fluid reservoir, SM: shunt model, SVC: superior vena cava, LPA, RPA: left and right pulmonary artery. The positions of the pressure ($1 = P_{\text{svc}}, 3 = P_{\text{rpa}}, P_{\text{par}}, P_{\text{wk}}$) and flow ($1 = Q_{\text{svc}}, 2 = Q_{\text{sh}}, 3 = Q_{\text{rpa}}$) measurements are shown. The colours of the lines represent the oxygenation in a physiological situation: red for oxygenated blood and blue for deoxygenated blood.

Figure 2.2. Schematic detail of the mock loop set-up: the fluid reservoir with inlet, outlet and overflow section.

Figure 2.3. The hydraulic mock loop set-up at continuous conditions. The distensible shunt model is shown.
2.1.2 Pulsatile condition

At pulsatile condition, the hydraulic mock loop set-up is almost the same as at continuous condition, but for some adaptations (figure 2.4). The centrifugal pump is replaced by a ventricular assist device system (VAD) (Thoratec™, Berkeley, California, USA), which supplies the SPS with a pulsatile flow. Both pumps withdraw the fluid from the atrial reservoir. Also a second windkessel (WK2) and a shunt resistance (SR) are added. These are necessary to control the pulsatile flow.

The flow through the SPS is determined by the following factors: the pressure and vacuum that control the ventricular pump, the shunt resistance and the compliance supplied by the windkessel. Also the length of the SPS, the preload and the afterload play a role. The length of the tubes, which connected the VAD with the rest of the set-up, are kept as short as possible so the effects of inertia can be neglected. The rest of the set-up determines the preload and afterload. So only the first four factors are used to obtain the desired SPS flow.

Two criteria are taken into account when setting the VAD, the pulsatility index (PI) and the average resistance index (RI). These indices are calculated as follows [Woo, 2002]:

\[
PI = \frac{Q_{\text{max}} - Q_{\text{min}}}{Q} \quad (2.1)
\]

\[
RI = \frac{Q_{\text{max}}}{Q_{\text{min}}} \quad (2.2)
\]

where: \(Q_{\text{max}}\) is the maximum flow, \(Q_{\text{min}}\) is the minimum flow and \(Q\) is the time-averaged flow.

Clinical data from Pennati [2000] and equation 2.1 and 2.2 are used to calculate the PI and the RI for the systemic-to pulmonary artery shunt in children with a hypoplastic left heart syndrome. The PI and RI are 0.7 and 2.1 respectively.

In the pulsatile set-up the porous resistances do not determine the pulmonary resistance anymore, see §4.1.1. An extra porous resistance is added and all are opened to minimise their influence on the pulmonary resistance. Two clamps were attached to the right and left pulmonary arteries (\(R_{\text{rpa}}\) and \(R_{\text{lpa}}\)). The pulmonary resistance is obtained by setting these clamps.

In addition to the pressures and flows measured at continuous conditions, also the flow (\(Q_{\text{lpa}}\)) and the pressure (\(P_{\text{lpa}}\)) at the LPA are recorded. The flows are measured with the same ultrasonic transit time flowmeter. The pressures are recorded with pressure transducers (accuracy ± 5 mmHg). These devices are connected to a computer device system (LabView 5.1) in order to collect the pulsatile tracings.
Analysis of the fluid dynamics in the SVC – PA anastomosis with an additional SPS

Figure 2.4. The schematic hydraulic mock loop set-up at pulsatile conditions. Instead of the centrifugal pump the VAD is implemented. Also a windkessel and resistance are added. VAD: ventricular assist device system, SR: shunt resistance, WK1 and WK2: windkessels, PR: porous resistances, \( R_{rpa} \) and \( R_{lpa} \): right and left pulmonary resistance, AR: atrial reservoir, RP: rollerpump, FR: fluid reservoir, SM: shunt model, SVC: superior vena cava, LPA, RPA: left and right pulmonary artery. The positions of the pressure (1 = \( P_{svc} \), 3 = \( P_{rpa} \), 4 = \( P_{lpa} \), \( P_{ar} \), \( P_{wk} \)) and flow (1 = \( Q_{svc} \), 2 = \( Q_{sh} \), 3 = \( Q_{rpa} \), 4 = \( Q_{lpa} \)) measurements are shown. The colours of the lines represent the oxygenation in a physiological situation: red for oxygenated blood and blue for deoxygenated blood.

Figure 2.5. The hydraulic mock loop set-up at pulsatile conditions. The clamps are not attached to the pulmonary arteries yet.

2.2 Shunt models

Two different configurations of the connection of the systemic-to-pulmonary shunt with the PA are used. In shunt model 1 (SM1), the SPS is connected directly at an angle of 45° to the left pulmonary artery and in shunt model 2 (SM2), the SPS is connected perpendicularly to the LPA at a distance of 1.8 cm from the anastomosis between the superior vena cava and the pulmonary artery (SVC-PA anastomosis) (figure 2.6). The inner diameter of the SVC-PA-
Analysis of the fluid dynamics in the SVC – PA anastomosis with an additional SPS

anastomosis is 1.0 cm and the inner diameter of the SPS tunnel is 0.5 cm. The lengths of the shunts are 3.4 and 3.9 cm respectively. Figure 2.6 shows the rigid shunt models.

Figure 2.6, Schematic representation of the rigid shunt models: a) In shunt model 1 (SM1), the SPS is connected directly at an angle of 45° to the left pulmonary artery. b) In shunt model 2 (SM2), the SPS is connected perpendicularly to the LPA at a distance of 1.8 cm from the SVC-PA anastomosis.

The distensible shunt models (DSM1 and DSM2) are partly made of Silcrothane® vascular prosthesis. This material is a patented polymer, which contains silicone. The material properties can be adapted by changing the percentage of silicone. In this study Silcrothane with 20% silicone is used for the SVC part of the shunt model and Silcrothane with 40% silicone is used for the PA part to obtain different Young moduli for the different vessels [Kull, 2002]. The SPS consists of the material Gore-Tex® (Fallstaff, AZ, USA). In DSM1, the SPS is not exactly connected to the SVC-PA-anastomosis, but to the SVC at a distance of 0.8 cm upwards from this anastomosis. The inner diameter of the superior vena cava is not zero but 1.2 cm. The rest of DSM1 and DSM2 has the same dimensions as the rigid shunt models.

2.3 Experimental protocol

The experiments were conducted at room temperature. Water (dynamic viscosity $\mu = 1.0$ cP, density $\rho = 1.0 \times 10^3$ kg/m$^3$) was used as the working fluid. During all tests, the pressure in the atrial reservoir was kept constant.

2.3.1 Preliminary tests: setting the pulmonary resistance for the continuous conditions

First, the pulmonary resistance was set for the continuous conditions. The pulmonary resistance is determined by the pressure drop between the right pulmonary artery and the atrial reservoir. At the continuous conditions, the porous resistances were used to set the pulmonary resistance.

Little experimental data on the haemodynamics in patients with a bi-directional Glenn shunt (BDG) are available. Clinical studies by Mavroudis [1999] and Stellin [2002] gave a range for the $P_{svc}$ of 11-16 mmHg. A problem was the large variety in age, physiology and pathology of the patients in postoperative follow-up studies. In the absence of data on the haemodynamics in patients immediately after a BDG placement, it was assumed that $P_{svc} = 10-12$ mmHg at $Q_{svc} = 1.0$ l/min was a normal value. One porous resistance was set to obtain these values for the pressure and the flow. The other porous resistance was set to obtain a situation with a high $P_{svc} = 15-17$ mmHg at $Q_{svc} = 1.0$ l/min was chosen.

The flow-pressure relationships of the porous resistances were determined by measuring $P_{wk}$ and $P_{ar}$ at constant $Q_{svc}$. Four series of subsequent runs were performed varying $Q_{svc}$ within the range of 0.4-1.0 l/min, with steps of 0.20 l/min. To investigate if the flow-pressure relationship of the resistance depends on time, the experiments were repeated two weeks after the first measurements. At this time, three series of subsequent runs were performed varying $P_{svc,0}$ = 7, 10, 15 and 20 mmHg. At the second measurements, also $P_{rpa}$ was recorded and the characteristics of the whole pulmonary resistance were determined. In order to evaluate the
obtained $P_{svc}$ at $Q_{svc} = 1.0$ l/min with the two pulmonary resistances, the $Q_{svc} - P_{svc}$ relationship was determined.

2.3.2 Experiments with rigid shunt models at continuous conditions
The first measurements were performed with the rigid shunt models (figure 2.6) at continuous conditions. The centrifugal pump supplied the SPS with a continuous flow (figure 2.1). Each test condition was identified by the imposed values for the upstream pressure in the superior vena cava, $(P_{svc}, 0)$, the flow through the systemic-to-pulmonary shunt, $(Q_{sh})$ and the pulmonary resistance (NPR and HPR). Subsequent runs were performed varying $P_{svc, 0}$ at different levels: 10 mmHg, 15 mmHg and 20 mmHg. The different constant pressure levels were obtained by varying the height of the fluid reservoir. At each run, the height of the fluid reservoir was kept constant while $Q_{sh}$ was varied within the range of 0.0-1.0 l/min, with steps of 0.25 l/min. To obtain $Q_{sh} = 0$ l/min, the centrifugal pump was switched off and the proximal end of the shunt was cross-clamped. The pulmonary resistance, which was obtained in the preliminary tests, was varied from a normal (NPR) to a high value (HPR). When each test condition was reached, pressure and flow measurements $P_{svc}, P_{rpa}, P_{ar}, Q_{svc}$ and $Q_{rpa}$ were recorded. The change in terms of percentage of $Q_{tot} (Q_{svc} + Q_{sh}), P_{svc}$ and $P_{rpa}$ were determined and the flow distribution through the pulmonary arteries was regarded.

2.3.3 Experiments with distensible shunt models at continuous conditions
Next, instead of the rigid shunt models, distensible shunt models (figure 2.6) were tested, also at continuous conditions and with the set-up as in figure 2.1. Subsequent runs were performed varying $P_{svc, 0}$ at two levels, 15 and 10 mmHg, to determine the effect of the SPS on the flow and the pressure in the SVC and the PA's. The pulmonary resistance was kept constant at the normal value.

2.3.4 Preliminary tests: setting the pulmonary resistance for the pulsatile conditions
The hydraulic mock loop set-up was changed according to figure 2.4. The centrifugal pump is replaced by a ventricular assist device system (VAD). The VAD supplies the SPS with a pulsatile flow. The pulmonary resistances were set by using two clamps: one at the right and one at the left pulmonary artery. The starting point was the same as at the continuous conditions: $P_{svc} = 10-12$ mmHg at $Q_{svc} = 1.0$ l/min. The clamps were set in such a way that these values were obtained and that the flow in the left pulmonary artery was equal to the flow in the right pulmonary artery. The flow-pressure relation from the pulmonary arteries and the total pulmonary resistance were determined as follows. $P_{rpa}, P_{lpa}, P_{wk}$ and $P_{ar}$ were recorded at three subsequent runs with 6 heights of the fluid reservoir (FR) in the range of $Q_{svc} = 0.4 - 1.4$ l/min.

2.3.5 Experiments with rigid shunt models at pulsatile conditions
Also in the case of a pulsatile SPS flow, the effect of the SPS in two different configurations on the flow and pressure in the SVC and PA's was examined. The fluid reservoir was set such that $P_{svc} = 10-12$ mmHg and $Q_{svc} = 1.0$ l/min while $Q_{sh} = 0$ l/min. In subsequent runs the mean SPS flow was varied, $Q = 0.5, 1.0$ and $1.4$ l/min. When each test condition was reached, pressure and flow measurements $P_{svc}, P_{rpa}, P_{lpa}, P_{ar}, Q_{svc}, Q_{rpa}$ and $Q_{lpa}$ were recorded during 5 seconds. The flows and pressures were averaged over 6 cycles to obtain mean values. The influence of $Q_{sh}$ on these averaged values was regarded and the flow distributions were calculated.
2.4 Computational study

Computational fluid dynamics (CFD) simulations were performed, to improve the understanding of the local fluid dynamics in the shunt models. The simulations were carried out by means of Fluent 6.0 (Fluent Inc., Lebanon, NH, USA). Fluent uses the finite volume method.

2.4.1 Simulations

Two different sets of simulations were performed at steady flow conditions with conduit diameters, angles and fluid properties as tested in vitro. Two 3-dimensional meshes with the same dimensions as used in vitro for the two different used shunt models are created in Gambit (Fluent Inc., Lebanon, NH, USA) (figure 2.7).

![Figure 2.7, 3-dimensional meshes of the rigid shunt models. a) SM1 and b) SM2.](image)

Because of symmetry only half of the shunt model is defined. The shunt models contain two inlets, the SVC and the SPS, and two outlets, the RPA and the LPA. In both outlets, the resistances (the porous jumps) are defined. These porous jumps represent the resistance of the left and right pulmonary artery and are used to simulate the pressure drop between the entrance of the right and left pulmonary artery and the windkessel. The right porous jump is set according to the following equations [Manuals Fluent]. The left porous jump is taken equal to the right one.

Experimental: \[ \Delta p = aQ^2 + bQ = aCSA^2v^2 + bCSAv \] (2.3)

Porous jump: \[ \Delta p = \left(C \frac{1}{2} \rho v^2 + \frac{\mu}{\gamma} v\right)\Delta m \] (2.4)

This results in: \[ aCSA^2 = C \frac{1}{2} \rho \Delta m \quad \text{and} \quad bCSA = \frac{\mu}{\gamma} \Delta m \] (2.5)

with a and b parameters for the pressure-flow relationship, CSA the cross sectional area, \( \rho \) the pressure, Q the flow, v the mean velocity, \( \rho \) the fluid density, \( \mu \) the fluid viscosity, \( \gamma \) the permeability porous jump, \( \Delta m \) the “thickness” porous jump and C a constant.

Experimental data are used to determine the relationship between the pressure at the entrance of the right PA and the windkessel and the flow through the RPA, equation 2.3. This relationship is used to determine values for C, \( \gamma \) and \( \Delta m \). The permeability of the porous jump, \( \gamma \), is chosen to be unity. The “thickness” of the porous jump, \( \Delta m \), and the constant, C,
can be calculated with equation 2.5. The thickness of the porous jump is virtual, because the resistance has to fit within the dimensions of the defined mesh.

### 2.4.2 Protocol

First, the shunt model fluid dynamics based on in vitro obtained data were simulated. The input values for the SPS flow were taken 0.25 and 1.0 l/min. The pressure in the SVC had to be set at 10 mmHg. However, with the pressure as input, it was much more difficult to find a stable solution and to reach convergence. So instead of the pressure the flow through the vena cava which was obtained during the experiments as described in §2.3.2 was used as an input value. The values for the porous jumps were calculated with data obtained during the experiments as described in §2.3.1, as described in §2.4.1. The input for the outlet was the windkessel pressure which was calculated using the pressure in the RPA (obtained from experiments, §2.3.2) and the pressure-flow relationship for the right PA (equation 2.3).

Second, a higher resistance (higher values for the porous jumps) was chosen as an input for the CFD model. The effect on the shunt model fluid dynamics of the total pulmonary resistance implemented in the pulmonary arteries was investigated. The input values for the SPS flow were again 0.25 and 1.0 l/min. The relationship between the pressure at the entrance of the right pulmonary artery and the atrial reservoir and the flow through the PA was used as equation 2.3. With this relation the variables for the porous jumps were calculated. Although the vena cava flow as the inlet input was better than the vena cava pressure, the vena cava pressure was defined (10 mmHg), because there were no experiments done with these values for the pulmonary resistances. Therefore, also the windkessel pressure was unknown and was set to zero.

The pressure contours, the particle pathlines, the velocity vectors and the flow distribution were evaluated.
3 Results

The fluid dynamics in two different shunt models were investigated. Several in-vitro experiments and numerical simulations were performed:

- **Continuous flow through the SPS:**
  Preliminary experiments were necessary to set and characterise the porous resistances and to characterise the pulmonary resistance. The effect of ageing of the porous resistances was regarded. Also it was evaluated if the pulmonary resistance led to the desired values for the pressure in the superior vena cava ($P_{svc}$) and the flow in the vena cava ($Q_{svc}$).
  After the preliminary experiments the influence of the SPS flow ($Q_{sh}$) on the flows and pressures in the SVC and right and left pulmonary artery (RPA and LPA) were regarded. This was done for both a rigid and a distensible shunt model.

- **Pulsatile flow through the SPS:**
  First, in a preliminary experiment the clamps at the PA’s were set and the pulmonary resistance was characterised. Then at different $Q_{sh}$, the $P_{svc}$, $P_{rpa}$, $P_{lpa}$, $Q_{svc}$, $Q_{rpa}$ and $Q_{lpa}$ were recorded during 5 seconds for the rigid shunt models. The flows and pressures during 6 cycles were averaged to obtain mean values.

- **The CFD models:**
  The first CFD model was based on the experiments with continuous SPS flow and the rigid shunt models and for the second model the pulmonary resistance was increased. The two configurations of the rigid shunt models were tested at $Q_{sh} = 0.25$ and $1.0$ l/min.

3.1 Results for continuous flow

3.1.1 Results preliminary experiments to set the pulmonary resistances

By setting the porous resistances, the pulmonary resistance is set. The flow-pressure relationship ($P_{wk}$-$P_{ar}$ versus $Q_{svc}$) of the porous resistances is shown in figure 3.1.1. Two series of measurements were done to investigate the effect of ageing. The experimental data and the second order interpolation are depicted.

![Figure 3.1.1](image-url)

**Figure 3.1.1.** Pressure difference between the windkessel and the atrial reservoir ($P_{wk}$-$P_{ar}$) as a function of the flow through the porous resistances, with normal (NPR) and high (HPR) pulmonary resistances at September 24 and October 9. Experimental data (marks) and second order interpolation (—).
An increase of the flow results in an increase of the pressure difference between the windkessel and the atrial reservoir. The higher is the pulmonary resistance, the higher is this pressure difference.

Figure 3.1.2 shows the flow – pressure relationships for the total normal and high pulmonary resistances, determined by the pressure difference between $P_{rpa}$ and $P_{ar}$.

**Figure 3.1.2,** Pressure difference between the right pulmonary artery and the atrial reservoir ($P_{rpa} - P_{ar}$) as a function of the flow through the superior vena cava, with normal (NPR) and high (HPR) pulmonary resistances. Experimental data (marks) and second order interpolation (---).

Figure 3.1.3 shows the experimental data and the linear interpolation of $P_{svc}$ versus $Q_{svc}$ with normal and high pulmonary resistances. This figure is used to evaluate the starting-point of the experiments, normal: $P_{svc} = 10-12$ mmHg and high: $P_{svc} = 15-17$ mmHg at $Q_{svc} = 1.0$ l/min.

**Figure 3.1.3,** $P_{svc}$ versus $Q_{svc}$ with normal (NPR) and high (HPR) pulmonary resistances. Experimental data (marks) and linear interpolation (---).
An increase of the $Q_{svc}$ leads to an increase of the $P_{svc}$. A higher pulmonary resistance results in a higher $P_{svc}$ than a lower pulmonary resistance.

### 3.1.2 Results of fluid dynamics with two different rigid shunt models

The influence of the flow through the SPS ($Q_{sh}$) on the flows in the SVC ($Q_{svc}$) and the total flow ($Q_{tot} = Q_{sh} + Q_{svc}$) through the pulmonary arteries is regarded. Figures 3.1.4 and 3.1.5 show the $Q_{sh}$-$Q_{svc}$ and the $Q_{sh}$-$Q_{tot}$ relationships for SM1 and SM2 with normal and high pulmonary resistance at $P_{svc,\theta} = 10$, 15 and 20 mmHg.

**Figure 3.1.4.** $Q_{svc}$ (—) and $Q_{tot}$ (---) versus $Q_{sh}$ through SM1 and SM2, with normal pulmonary resistance at $P_{svc,\theta} = 10$, 15 and 20 mmHg.

**Figure 3.1.5.** $Q_{svc}$ (—) and $Q_{tot}$ (---) versus the flow through SM1 and SM2, with high pulmonary resistance at $P_{svc,\theta} = 10$, 15 and 20 mmHg.
Table 3.1.1 lists the change in terms of percentage of the total flow $Q_{tot}$ and of $Q_{svc}$ at $Q_{sh} = 1.0 \text{ l/min}$ with SM1 and SM2, at $P_{svc, 0} = 10, 15$ and $20 \text{ mmHg}$ with normal and high pulmonary resistance with respect to the total flow with the SPS cross-clamped.

**Table 3.1.1, Change in terms of percentage of $Q_{tot}$ and $Q_{svc}$ at $Q_{sh} = 1.0 \text{ l/min}$ with respect to the flow with SPS crossclamped.**

<table>
<thead>
<tr>
<th>$P_{svc, 0}$ (mmHg)</th>
<th>SM1</th>
<th></th>
<th></th>
<th>SM2</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>10</td>
<td>15</td>
<td>20</td>
<td>10</td>
<td>15</td>
<td>20</td>
</tr>
<tr>
<td>% change $Q_{tot}$ with NPR</td>
<td>23.7</td>
<td>15.3</td>
<td>13.0</td>
<td>20.3</td>
<td>14.4</td>
<td>12.6</td>
</tr>
<tr>
<td>% change $Q_{tot}$ with HPR</td>
<td>17.3</td>
<td>11.3</td>
<td>13.8</td>
<td>16.4</td>
<td>4.2</td>
<td>12.0</td>
</tr>
<tr>
<td>% change $Q_{svc}$ with NPR</td>
<td>-87.4</td>
<td>-58.8</td>
<td>-46.1</td>
<td>-88.4</td>
<td>-58.6</td>
<td>-46.5</td>
</tr>
<tr>
<td>% change $Q_{svc}$ with HPR</td>
<td>-161.4</td>
<td>-105.9</td>
<td>-78.0</td>
<td>-165.5</td>
<td>-110.3</td>
<td>-78.3</td>
</tr>
</tbody>
</table>

Figures 3.1.6 to 3.1.9 show the influence of $Q_{sh}$ on the flow distribution in the pulmonary arteries. The $Q_{lpa}$ and $Q_{rpa}$ versus $Q_{sh}$ through SM1 and SM2 with normal and high pulmonary resistance at $P_{svc, 0} = 10, 15$ and $20 \text{ mmHg}$ are depicted.

**Figure 3.1.6, $Q_{rpa}$ and $Q_{lpa}$ versus $Q_{sh}$ through SM1 with normal pulmonary resistance at $P_{svc, 0} = 10, 15$ and $20 \text{ mmHg}$.**
Figure 3.1.7, $Q_{rpa}$ and $Q_{lpa}$ versus $Q_{sh}$ through SM1 with high pulmonary resistance at $P_{svc, 0} = 10, 15$ and $20$ mmHg.

Figure 3.1.8, $Q_{rpa}$ and $Q_{lpa}$ versus $Q_{sh}$ through SM2 with normal pulmonary resistance at $P_{svc, 0} = 10, 15$ and $20$ mmHg.
Analysis of the fluid dynamics in the SVC – PA anastomosis with an additional SPS

Figure 3.1.9, $Q_{rpa}$ and $Q_{lpa}$ versus $Q_{sh}$ through SM2 with high pulmonary resistance at $P_{svc, 0} = 10, 15$ and $20$ mmHg.

Tables 3.1.2 a and b list the flow distribution at $Q_{sh} = 0, 0.5$ and $1.0$ l/min with normal and high pulmonary resistances.

**Table 3.1.2 a.** The flow distribution at $P_{svc, 0} = 10, 15$ and $20$ mmHg with normal pulmonary resistance.

<table>
<thead>
<tr>
<th>Normal pulmonary resistance</th>
<th>SM1</th>
<th></th>
<th>SM2</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>$Q_{sh}$ (l/min)</td>
<td>0</td>
<td>0.5</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>$Q_{rpa}/Q_{lpa}$ ($P_{svc, 0} = 10$ mmHg)</td>
<td>0.97</td>
<td>1.49</td>
<td>-19.17</td>
<td>1.04</td>
</tr>
<tr>
<td>$Q_{rpa}/Q_{lpa}$ ($P_{svc, 0} = 15$ mmHg)</td>
<td>0.91</td>
<td>1.21</td>
<td>3.00</td>
<td>1.06</td>
</tr>
<tr>
<td>$Q_{rpa}/Q_{lpa}$ ($P_{svc, 0} = 20$ mmHg)</td>
<td>0.92</td>
<td>1.13</td>
<td>1.91</td>
<td>1.08</td>
</tr>
</tbody>
</table>

**Table 3.1.2 b.** The flow distribution at $P_{svc, 0} = 10, 15$ and $20$ mmHg with high pulmonary resistance.

<table>
<thead>
<tr>
<th>High pulmonary resistance</th>
<th>SM1</th>
<th></th>
<th>SM2</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>$Q_{sh}$ (l/min)</td>
<td>0</td>
<td>0.5</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>$Q_{rpa}/Q_{lpa}$ ($P_{svc, 0} = 10$ mmHg)</td>
<td>0.93</td>
<td>4.76</td>
<td>-2.44</td>
<td>1.09</td>
</tr>
<tr>
<td>$Q_{rpa}/Q_{lpa}$ ($P_{svc, 0} = 15$ mmHg)</td>
<td>0.94</td>
<td>1.80</td>
<td>-6.46</td>
<td>1.06</td>
</tr>
<tr>
<td>$Q_{rpa}/Q_{lpa}$ ($P_{svc, 0} = 20$ mmHg)</td>
<td>0.92</td>
<td>1.30</td>
<td>9.07</td>
<td>1.08</td>
</tr>
</tbody>
</table>

Figure 3.1.10 shows the $P_{svc}$ and $P_{rpa}$ as function of the $Q_{sh}$ through SM1 with the normal pulmonary resistance at $P_{svc, 0} = 10, 15$ and $20$ mmHg. The developments of $P_{svc}$ and $P_{rpa}$ as function of $Q_{sh}$ through SM2 and with high pulmonary resistance are quite similar and are shown in Appendix C.
Analysis of the fluid dynamics in the SVC – PA anastomosis with an additional SPS

![Graph showing fluid dynamics](image)

**Figure 3.1.10.** $P_{\text{svc}}$ and $P_{\text{rpa}}$ versus $Q_{\text{sh}}$ through SM1 with normal pulmonary resistance at $P_{\text{svc}, 0} = 10, 15$ and $20$ mmHg.

Table 3.1.3 lists the increase in terms of percentage of the $P_{\text{svc}}$ and $P_{\text{rpa}}$ at $Q_{\text{sh}} = 1.0$ l/min through SM1 and SM2 with respect to the pressures with SPS cross-clamped, at $P_{\text{svc}, 0} = 10, 15$ and $20$ mmHg with normal and high pulmonary resistance.

**Table 3.1.3, Increase in terms of percentage of $P_{\text{svc}}$ and $P_{\text{rpa}}$ at $Q_{\text{sh}} = 1.0$ l/min with respect to pressures with SPS cross-clamped.**

<table>
<thead>
<tr>
<th>normal pulmonary resistance</th>
<th>SM1</th>
<th>SM2</th>
</tr>
</thead>
<tbody>
<tr>
<td>$P_{\text{svc}, 0}$ (mmHg)</td>
<td>10</td>
<td>15</td>
</tr>
<tr>
<td>increase $P_{\text{svc}}$ (%)</td>
<td>11.1</td>
<td>11.1</td>
</tr>
<tr>
<td>increase $P_{\text{rpa}}$ (%)</td>
<td>21.4</td>
<td>15.6</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>high pulmonary resistance</th>
<th>SM1</th>
<th>SM2</th>
</tr>
</thead>
<tbody>
<tr>
<td>$P_{\text{svc}, 0}$ (mmHg)</td>
<td>10</td>
<td>15</td>
</tr>
<tr>
<td>increase $P_{\text{svc}}$ (%)</td>
<td>9.8</td>
<td>7.5</td>
</tr>
<tr>
<td>increase $P_{\text{rpa}}$ (%)</td>
<td>22.1</td>
<td>14.2</td>
</tr>
</tbody>
</table>
3.1.3 Results of fluid dynamics with two different distensible shunt models

The influence of the flow through the SPS \( Q_{sh} \) in the distensible shunt models (DSM1 and DSM2) on the flows in the SVC \( Q_{svc} \) and the total flow \( Q_{tot} = Q_{sh} + Q_{svc} \) through the pulmonary arteries is regarded. Figures 3.1.11 and 3.1.12 show the \( Q_{sh}-Q_{svc} \) and the \( Q_{sh}-Q_{tot} \) relationships for DSM1 and DSM2 with the normal pulmonary resistance at \( P_{svc,0} = 10 \) and 15 mmHg. Table 3.1.4 gives the change in terms of percentage of \( Q_{tot} \) and of \( Q_{svc} \) at \( Q_{sh} = 1.0 \) l/min.

![Graph showing \( Q_{tot} \) and \( Q_{svc} \) vs. \( Q_{sh} \) for DSM1 and DSM2 with normal pulmonary resistance at \( P_{svc,0} = 10 \) and 15 mmHg.]

Figure 3.1.11, \( Q_{svc} \) (—) and \( Q_{tot} \) (---) versus the flow through DSM1 and DSM2, with normal pulmonary resistance at \( P_{svc,0} = 10 \) and 15 mmHg.

**Table 3.1.4.** Change in terms of percentage of \( Q_{tot} \) and \( Q_{svc} \) at \( Q_{sh} = 1.0 \) l/min with respect to the flow with SPS crossclamped.

<table>
<thead>
<tr>
<th>( P_{svc,0} ) (mmHg)</th>
<th>DSM1</th>
<th>DSM2</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>10</td>
<td>15</td>
</tr>
<tr>
<td>% change ( Q_{tot} )</td>
<td>19.9</td>
<td>16.1</td>
</tr>
<tr>
<td>% change ( Q_{svc} )</td>
<td>-97.3</td>
<td>-64.5</td>
</tr>
</tbody>
</table>

Figures 3.1.12 and 3.1.13 show the influence of \( Q_{sh} \) on the flow distribution in the pulmonary arteries. The \( Q_{lpa} \) and \( Q_{rpa} \) versus \( Q_{sh} \) through DSM1 and DSM2 with normal pulmonary resistance at \( P_{svc,0} = 10 \) and 15 mmHg are depicted.
Analysis of the fluid dynamics in the SVC – PA anastomosis with an additional SPS

Figure 3.1.12, $Q_{rpa}$ and $Q_{lpa}$ versus $Q_{sh}$ through DSM1 with normal pulmonary resistance at $P_{svc, 0} = 10$ and $15$ mmHg.

Figure 3.1.13, $Q_{rpa}$ and $Q_{lpa}$ versus $Q_{sh}$ through DSM2 with normal pulmonary resistance at $P_{svc, 0} = 10$ and $15$ mmHg.

Table 3.1.5 lists the flow distribution at $Q_{sh} = 0$, $0.5$ and $1.0$ l/min through the distensible shunt models.

**Table 3.1.5. The flow distribution at $P_{svc, 0} = 10$ and $15$ mmHg with distensible shunt models.**

<table>
<thead>
<tr>
<th>Normal pulmonary resistance</th>
<th>DSM1</th>
<th></th>
<th></th>
<th>DSM2</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>$Q_{sh}$ (l/min)</td>
<td>0</td>
<td>0.5</td>
<td>1</td>
<td>0</td>
<td>0.5</td>
<td>1</td>
</tr>
<tr>
<td>$Q_{rpa}/Q_{lpa}$ ($P_{svc, 0} = 10$ mmHg)</td>
<td>1.02</td>
<td>0.91</td>
<td>0.73</td>
<td>0.32</td>
<td>0.40</td>
<td>0.49</td>
</tr>
<tr>
<td>$Q_{rpa}/Q_{lpa}$ ($P_{svc, 0} = 15$ mmHg)</td>
<td>0.98</td>
<td>1.07</td>
<td>0.89</td>
<td>0.28</td>
<td>0.29</td>
<td>0.38</td>
</tr>
</tbody>
</table>
Figures 3.1.14 and 3.1.15 show the $P_{svc}$ and $P_{rpa}$ as a function of the $Q_{sh}$ through DSM1 and DSM2 with the normal pulmonary resistance at $P_{svc, 0} = 10$ and 15 mmHg.

The maximum increases of $P_{svc}$ and $P_{rpa}$ at maximum shunt flow ($Q_{sh} = 1.0$ l/min) are listed in table 3.1.6.

Table 3.1.6, Increase in terms of percentage of $P_{svc}$ and $P_{rpa}$ at $Q_{sh} = 1.0$ l/min through DSM1 and DSM2 with respect to pressures with SPS crossclamped.

<table>
<thead>
<tr>
<th>$P_{svc, 0}$ (mmHg)</th>
<th>DSM1</th>
<th>DSM2</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>10</td>
<td>15</td>
</tr>
<tr>
<td>increase $P_{svc}$ (%)</td>
<td>12.4</td>
<td>10.0</td>
</tr>
<tr>
<td>increase $P_{rpa}$ (%)</td>
<td>21.2</td>
<td>18.6</td>
</tr>
</tbody>
</table>
3.2 Results for pulsatile flow

3.2.1 Results preliminary experiment to set the pulmonary resistance with clamps

The set-up was changed for the experiments with a pulsatile flow through the SPS, §2.1.2. Under pulsatile conditions, the pulmonary resistance was set with one clamp on each pulmonary artery. Figure 3.2.1 shows the experimental data and the second order interpolation of the flow-pressure relationship ($P_{rpa} - P_{wk}$ versus $Q_{rpa}$) of the pulmonary arteries.

\[ y = 10.1x^2 + 9.9x - 4.1 \]
\[ R^2 = 0.97 \]

Figure 3.2.1, Pressure difference between the windkessel and the atrial reservoir ($P_{rpa} - P_{wk}$) as a function of the flow through the pulmonary arteries with the clamps. Experimental data (marks) and second order interpolation (—).

Figure 3.2.2 gives the flow-pressure relationship for the total pulmonary resistance, determined by the pressure difference between $P_{rpa}$ and $P_{ar}$.

\[ y = 5.4x^2 + 3.4x - 3.0 \]
\[ R^2 = 1.0 \]

Figure 3.2.2, Pressure difference between the right pulmonary artery and the atrial reservoir ($P_{rpa} - P_{ar}$) as a function of the flow through the superior vena cava, with normal (NPR) and high (HPR) pulmonary resistances. Experimental data (marks) and second order interpolation (—).
3.2.2 Results of fluid dynamics with two different rigid shunt models

Figure 3.2.3 shows an example of the recorded flow tracings during the pulsatile experiments.

![Flow tracings with SM2](image)

Figure 3.2.3, Flow tracings with SM2, at mean $P_{svc, 0} = 11.9$ mmHg, at mean $Q_{sh} = 0.53$ l/min.

The influence of mean flow through the shunt model 1 and 2 on the mean flows through the SVC, the RPA and the LPA are depicted in figures 3.2.4 and 3.2.5.

![Mean flows through SM1](image)

Figure 3.2.4, mean $Q_{tot}$, $Q_{svc}$, $Q_{rpa}$ and $Q_{lpa}$ versus the mean flow through SM1 at pulsatile conditions.
Figure 3.2.5, mean $Q_{tot}$, $Q_{svc}$, $Q_{rpa}$ and $Q_{lpa}$ versus the mean flow through SM2 at pulsatile conditions.

Figures 3.2.6 and 3.2.7 show the mean $P_{svc}$, $P_{rpa}$ and $P_{lpa}$ versus the mean flow through respectively shunt model 1 and model 2.

Figure 3.2.6, mean $P_{svc}$, $P_{rpa}$ and $P_{lpa}$ versus the mean flow through SM1 at pulsatile conditions.
Analysis of the fluid dynamics in the SVC – PA anastomosis with an additional SPS

3.3 Results computational study
Two different sets of simulations were performed at steady flow conditions. First, the shunt model fluid dynamics based on in vitro obtained data was simulated. Second, a higher resistance was chosen as an input value for the CFD model.

3.3.1 The shunt model fluid dynamics based on in vitro obtained data
The relationship between the pressure difference between the $P_{\text{rpa}}$ and $P_{\text{wk}}$ and the flow through the RPA is given in the figure 3.3.1.

The second order interpolation of the pressure-flow relationship is:

$$ p_{\text{rpa}} - p_{\text{wk}} = 0.9Q_{\text{rpa}}^2 + 0.6Q_{\text{rpa}} $$

(3.1)
With equations 2.6-8 the parameters of the porous jump were calculated:

**Table 3.3.1, parameters of the porous jumps**

<table>
<thead>
<tr>
<th>γ (m²)</th>
<th>Δm (m)</th>
<th>C (1/m)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>0.36·10⁶</td>
<td>1.55·10⁻⁵</td>
</tr>
</tbody>
</table>

During the experiments as described in §2.3.2 at a superior vena cava pressure of 10 mmHg the value of the flow through the vena cava was as given in table 3.3.2. The pressure in the outlet, the windkessel, was determined using the $p_{pa}$ at $p_{svc} = 10$ mmHg and equation 3.1, see also table 3.3.2.

**Table 3.3.2, values for the vena cava flow and $P_{wk}$ at $P_{svc, 0} = 10$ mmHg.**

<table>
<thead>
<tr>
<th></th>
<th>$Q_{svc}$ (l/min)</th>
<th>$P_{wk}$ (mmHg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>SM1, $Q_{sh} = 0.25$ l/min</td>
<td>0.72</td>
<td>9.76</td>
</tr>
<tr>
<td>SM1, $Q_{sh} = 1.0$ l/min</td>
<td>0.11</td>
<td>10.27</td>
</tr>
<tr>
<td>SM2, $Q_{sh} = 0.25$ l/min</td>
<td>0.72</td>
<td>9.89</td>
</tr>
<tr>
<td>SM2, $Q_{sh} = 1.0$ l/min</td>
<td>0.11</td>
<td>10.90</td>
</tr>
</tbody>
</table>

With these input values the simulations were done. First, the particle pathlines for shunt model 1 and 2 at the two SPS flows ($Q_{sh} = 0.25$ and 1.0 l/min) are shown.

*Figure 3.3.2, Particle path lines for shunt model 1 and 2 at the two SPS flows ($Q_{sh} = 0.25$ and 1.0 l/min). The different colours represent the different particles.*
Second, the contours of the static pressure are depicted.

![Shunt model 1](image1)

![Shunt model 2](image2)

$Q_{sh} = 0.25 \text{ l/min}$

$Q_{sh} = 1.0 \text{ l/min}$

*Figure 3.3.3, the contours of the static pressure for shunt model 1 and 2 at $Q_{sh} = 0.25$ and $Q_{sh} = 1.0 \text{ l/min.}$*
Figure 3.3.4 gives the velocity vectors.

**Shunt model 1**

\[ Q_{sh} = 0.25 \text{ l/min} \]

**Shunt model 2**

\[ Q_{sh} = 1.0 \text{ l/min} \]

*Figure 3.3.4, Velocity vectors for shunt model 1 and 2 at \( Q_{sh} = 0.25 \) and \( Q_{sh} = 1.0 \text{ l/min} \).*
3.3.2 The shunt model fluid dynamics with a higher resistance

The following flow-pressure relationship holds for the total pulmonary resistance.

\[ y = 18.7x^2 + 2.1x \]

\[ R^2 = 0.98 \]

![Graph of flow-pressure relationship](image)

Figure 3.3.5, the relationship between the pressure difference \( P_{rpa} - P_{ar} \) and the flow through the PA.

The second order interpolation of the pressure-flow relationship is as follows:

\[ p_{rpa} - p_{ar} = 18.7 Q_{rpa}^2 + 2.1 Q_{rpa} \quad (3.2) \]

The parameters for the porous jumps were:

<table>
<thead>
<tr>
<th>( \gamma ) (m(^2))</th>
<th>( \Delta m ) (m)</th>
<th>( C ) (1/m)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>1.39 \times 10^6</td>
<td>8.72 \times 10^{-5}</td>
</tr>
</tbody>
</table>

With \( p_{svc} = 10 \) mmHg and \( p_{wk} = 0 \) mmHg, the simulations led to the following results.
Analysis of the fluid dynamics in the SVC – PA anastomosis with an additional SPS

Shunt model 1

![ Particle path lines for shunt model 1 at Q_{sh} = 0.25 l/min. ]

Shunt model 2

![ Particle path lines for shunt model 2 at Q_{sh} = 1.0 l/min. ]

Figure 3.3.6, Particle path lines for shunt model 1 and 2 at the two SPS flows (Q_{sh} = 0.25 and 1.0 l/min). The different colours represent the different particles.
Second, the contours of the static pressure are depicted.

\[ Q_{sh} = 0.25 \text{ l/min}, \text{ both shunt models have the same colour coded bar.} \]

\[ Q_{sh} = 1.0 \text{ l/min} \]

*Figure 3.3.7, the contours of the static pressure for shunt model 1 and 2 at } Q_{sh} = 0.25 \text{ and } Q_{sh} = 1.0 \text{ l/min.} *
Figure 3.3.8 gives the velocity vectors.

**Shunt model 1**

- $Q_{sh} = 0.25$ l/min
- $Q_{sh} = 1.0$ l/min

**Shunt model 2**

- $Q_{sh} = 0.25$ l/min
- $Q_{sh} = 1.0$ l/min

*Figure 3.3.8, Velocity vectors for shunt model 1 and 2 at $Q_{sh} = 0.25$ and $Q_{sh} = 1.0$ l/min.*
4 Discussion

4.1 Continuous flow

4.1.1 Preliminary experiments to set the pulmonary resistances

Figure 3.1.1 shows that the flow-pressure relationships of the porous resistances are well described by a second order approximation ($R^2$ in range from 0.97 to 1.0). The effects of bending of the flow between the windkessel and the atrial reservoir before it enters the porous resistances explain a part of this non-linearity.

To characterise the porous resistances (NPR and HPR), the slopes of the flow–pressure ($Q_{svc}$ vs. $dP_{pr}$) relationships at $Q_{svc} = 0.5, 1.0$ and $1.5$ l/min were determined (table 4.1). After two weeks, the resistances were decreased a little. An explanation is a change in the properties of the porous material during the experiments. During the experiments with the SPS connected, the minimum flow through the normal pulmonary resistances was approximately 1.0 l/min. It means that the decrease in the slope was maximum 8.2% in two weeks. Regarding these results, it can be concluded that the resistance does not change significantly when all corresponding measurements are done at one day.

<table>
<thead>
<tr>
<th>$Q_{svc}$ (l/min)</th>
<th>NPR</th>
<th>HPR</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>0.5</td>
<td>1.0</td>
</tr>
<tr>
<td>Slope $Q_{svc}$-$dP_{pr}$ (mmHg/l·min), date: Sep-24</td>
<td>5.9</td>
<td>9.8</td>
</tr>
<tr>
<td>Slope $Q_{svc}$-$dP_{pr}$ (mmHg/l·min), date: Oct-09</td>
<td>4.6</td>
<td>9.0</td>
</tr>
<tr>
<td>difference in slope $Q_{svc}$-$dP_{pr}$ (%) between Oct-09 and Sep-24</td>
<td>-22.0</td>
<td>-8.2</td>
</tr>
</tbody>
</table>

Figure 3.1.2 shows that the flow-pressure relationships of the total pulmonary resistances ($Q_{svc}$-$dP_{tpr}$) are well described by a second order approximation ($R^2 = 0.9705$ to 0.9964).

Table 4.2 compares the slopes of the porous and the total pulmonary resistances at $Q_{svc} = 0.5, 1.0$ and $1.5$ l/min with NPR and HPR.

<table>
<thead>
<tr>
<th>$Q_{svc}$ (l/min)</th>
<th>NPR</th>
<th>HPR</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>0.5</td>
<td>1.0</td>
</tr>
<tr>
<td>Slope $Q_{svc}$-$dP_{pr}$ (mmHg/l·min)</td>
<td>4.6</td>
<td>9.0</td>
</tr>
<tr>
<td>Slope $Q_{svc}$-$dP_{tpr}$ (mmHg/l·min)</td>
<td>5.1</td>
<td>9.7</td>
</tr>
<tr>
<td>contribution PR/TPR (%) (Porous Res./Total pulm. Res.)</td>
<td>90.2</td>
<td>92.3</td>
</tr>
</tbody>
</table>

From table 4.2, it appears that the total pulmonary resistances were almost completely determined by the porous resistances. It is concluded that the porous resistances can be used to control the pulmonary resistance.

Clinical data show a range from 2 to 15 mmHg/l·min [Migliavacca, 2000] for the pulmonary resistances. The values for the normal pulmonary resistance are within this range at a $Q_{svc}$ varying from 0 to 1.5 l/min. The values for the high pulmonary resistance are a little higher for flows higher than 1.0 l/min, but this is acceptable.

Figure 3.1.3 shows that the $P_{svc}$ - $Q_{svc}$ relationships are well described by a linear interpolation ($R^2 = 0.97$ and 0.95). The normal pulmonary resistance resulted in a $P_{svc}$ of 10.2 mmHg, while with the high pulmonary resistance a $P_{svc}$ of 16.8 mmHg was obtained at $Q = 1.0$ l/min. The
goal was to set the pulmonary resistances at a normal and high value to obtain a normal (10-12 mmHg, see §2.3.1) and a high (15-17 mmHg) P_{svc} at Q_{svc} = 1.0 l/min. So the pulmonary resistance satisfies these target values.

### 4.1.2 Fluid dynamics with two different rigid shunt models

Figures 3.1.4 and 3.1.5 show that an increase in Q_{sh} led to a linear increase of Q_{tot}. The maximum increase of Q_{tot} was 23.7 % (table 3.1.1). With a normal pulmonary resistance, an increase of P_{svc,0} led to a smaller increase in terms of percentage of Q_{tot}. This trend can not be confirmed with the results of the high pulmonary resistance. Furthermore, an increase of Q_{sh} led to a strong linear decrease of Q_{svc} at all different conditions (maximally 165 %, table 3.1.1). A lower P_{svc,0} and a higher resistance led to a larger decrease in terms of percentage of Q_{svc}. At high pulmonary resistance, at P_{svc,0} = 10 and 15 mmHg, the flow through the superior vena cava became even negative. Also, a higher P_{svc,0} led to a higher Q_{svc} and Q_{tot} and an increase in pulmonary resistance resulted in a decrease of the Q_{svc} and Q_{tot}. However, the slope of the Q_{sh}-Q_{svc} and Q_{sh}-Q_{tot} relationships did not change significantly for these different conditions.

Comparing the results from SM1 and SM2, there was no significant difference between the Q_{sh}-Q_{svc} relationships. Also, it is remarkable that the results strongly resembled the calculated results of the simplified ejector model (figure 1.6) at the domain 0 < Q_{sh} < 1.0 l/min. So, a different angle or position of the SPS did not effect the flow from the superior vena cava.

However, the configuration of the shunt model did influence the flow partition (figures 3.1.6 to 3.1.9). With SM1, an increase of Q_{sh} resulted in an increase of Q_{rpa} and a decrease of Q_{lpa} which appeared to be dependent on the P_{svc,0} (figure 3.1.6). A lower value of P_{svc,0} had more influence on the increase and decrease of respectively Q_{rpa} and Q_{lpa}. Regarding figures 3.1.6 and 3.1.7 at Q_{sh} = 1.0 l/min, it is remarkable that an increase of P_{svc,0} led to a small increase of Q_{rpa} (about 0.15 l/min) while Q_{lpa} and Q_{svc} differed strongly (about 0.9 l/min).

At high pulmonary resistance, the Q_{rpa} and Q_{lpa} were lower (figure 3.1.7) than at normal pulmonary resistance, except at Q_{sh} = 1.0 l/min and P_{svc,0} = 10 mmHg. The increase of Q_{rpa} and the decrease of Q_{lpa} were stronger than with the normal resistance. At P_{svc,0} = 10 and 15 mmHg, the Q_{lpa} at high resistance became even negative.

The Q_{rpa} and Q_{lpa} in SM1 were dependent on the value of the P_{svc,0} and the resistances, which led to a large range of flow distributions (-19.17 to 4.76, table 3.1.2 a and b). At Q_{sh} = 0.5 l/min, an increase in the P_{svc,0} and a decrease in resistance led to a decrease of the flow distribution. At Q_{sh} = 1.0 l/min, this trend was not observed. The flow distribution became negative when there was backflow in left lung.

Figures 3.1.8 and 3.1.9 show that in contrast to SM1, with SM2 the Q_{rpa} and Q_{lpa} both slowly increased when Q_{sh} increased. This difference between SM1 and SM2 is explained by the difference in geometry. In SM1, the SPS is connected at an angle of 45°, so more flow goes through the RPA. In SM2, the SPS is connected perpendicularly, which led to a more homogenous flow distribution. A higher P_{svc,0} increased the flows through the lungs, while a higher resistance decreased these flows. The flow distribution did not change as much as with SM1 (table 3.1.2 a) at the different conditions. By varying the P_{svc,0} and the pulmonary resistance, the flow distribution in SM2 was about unity (table 3.1.2 b).

Figure 3.1.10 shows that an increase of the Q_{sh} resulted in a low increase of the P_{svc} and P_{rpa}. At maximum Q_{sh} of 1.0 l/min, at P_{svc,0} = 10 mmHg with normal pulmonary resistance, the P_{svc} increased maximally 11.1 % (about 1.7 mmHg) and the P_{rpa} 21.4 % (about 2.2 mmHg) (table 3.1.3). This is acceptable in the physiological situation. The development of the P_{svc} did not differ significantly from the development of P_{rpa}. The pressure drop between P_{rpa} and P_{svc} at the shunt models was in the range of 0 to 1 mmHg. According to figure 3.1.10 and appendix C, the developments did hardly depend on the P_{svc,0} and the pulmonary resistance.
4.1.3 Fluid dynamics with two different distensible shunt models

Figure 3.1.11 shows that there was no significant difference in the developments of the flows between DSM1 and DSM2. However, the absolute values of the flow differed at the same \( P_{svc,0} \) and resistance, which could be due to the different resistances of the shunt models. At the different conditions, \( Q_{tot} \) did not increase much (maximally approximately 20 %, table 3.1.4) with respect to the corresponding strong decrease of \( Q_{svc} \) (97 %). At maximum \( Q_{sh} \) with DSM2 at \( P_{svc,0} = 10 \text{ mmHg} \), \( Q_{svc} \) became even negative. It was expected that at a low pressure the shape of the anastomosis is more oval and at high pressure the anastomosis is more circular and has a larger area, which should result into different fluid dynamics. However, the trend of the \( Q_{sh}-Q_{svc} \) and \( Q_{sh}-Q_{tot} \) relationships did not differ significantly at different \( P_{svc,0} \). Furthermore, the distensible shunt models showed the same slope of the flows as the rigid shunt models and thus also these results strongly resembled the calculated results of the simplified ejector model (figure 1.6) at the domain \( 0 < Q_{sh} < 1.0 \text{ l/min} \). From these results, it appears that a distensible anastomosis does not change the flow in the superior vena cava in the pressure measurement range.

The flow distribution in the distensible shunts did not resemble with the flow distribution in the rigid shunts. Especially between SM1 and DSM1, the behaviour was completely different. With DSM1, \( Q_{lpa} \) increased while in SM1 the \( Q_{lpa} \) decreased when \( Q_{sh} \) increased (figure 3.1.6 and 3.1.12). It is remarkable that with DSM2, there was a low flow distribution (0.32 and 0.28, table 3.1.5) although the shunt was cross-clamped. Furthermore there is no unambiguous relation between the \( P_{svc,0} \) and the flow distribution in the two shunt models. This is probably due to a difference in resistance between the right and left connections of the shunt models. It was difficult to connect the shunt models within the test set-up, because the material (Silcrothan) was easily damaged.

Table 3.1.6 shows that with DSM1, the pressure increase of SVC (12.4 % and 10.0 %) and RPA (21.2 % and 18.6 %) and the SVC-RPA pressure drop (approximately 2 mmHg, figure 3.1.14) were acceptable in the physiological situation. The \( P_{rpa} \) with DSM2 (figure 3.1.15) was lower than with DSM1. This agrees with the result that more flow goes to the left than to the right pulmonary artery in DSM2 in comparison with DSM1.

As mentioned before, backflow in the left lung occurred with the rigid SM1 (figure 3.1.7). Of course, this is a non-physiological situation. Physiologically, after the division into the right and left pulmonary artery, each pulmonary artery supplies the flow to the respective lung. The blood from the LPA can not flow into the RPA and vice versa. However, in the experiments the position of the largest contribution to the pulmonary resistance, namely the porous resistances are situated behind the windkessel. The pulmonary arteries have a much lower resistance. Therefore, the fluid flowed much easier from the RPA to the LPA instead of in the direction of the porous resistance, resulting in a circulation flow in the pulmonary artery. For this reason, the experimental set-up was changed at pulsatile conditions. Two clamps are situated at the pulmonary arteries to force the flow in the direction of the atrial reservoir.

4.2 Pulsatile flow

4.2.1 Preliminary experiments to set the pulmonary resistances with clamps

Figures 3.2.1 and 3.2.2 show that the pressure difference was negative for low flows. The pressure difference was not zero but 4 and 3 mmHg respectively at zero flow. The pressure transducers were calibrated but were not able to measure the pressures precisely within a mmHg. So inaccuracy of the pressure transducers can explain these pressure differences at zero flow.

\[ Q_{rpa} = 0.5 \text{ l/min} \], the slope of the flow – pressure relationship (figure 3.2.1) is 19.95 mmHg/l/min. Assumed is that the left and right pulmonary artery have the same resistance.
Because the right and left PA are parallel, equation 4.1 is used to calculate the total resistance of the pulmonary arteries (R_{pa}).

\[
\frac{1}{R_{pa}} = \frac{1}{R_{rpa}} + \frac{1}{R_{lpa}} \tag{4.1}
\]

It follows that the resistance of the pulmonary arteries is 9.98 mmHg/l·min. The total pulmonary resistance (figure 3.2.2) at Q_{svc} = 1.0 l/min is 14.27 mmHg/l·min. This means that the clamps in the pulmonary arteries mainly determine the total pulmonary resistance.

### 4.2.2 Fluid dynamics with two different rigid shunt models at pulsatile conditions

Figure 3.2.3 shows that the maxima and minima of the pulsatile tracings were not completely constant. A variation of the tracing itself was observed, due to external disturbances.

Although it is difficult to compare the results with the continuous conditions, the same trend of Q_{svc} and Q_{tot} was observed (figure 3.2.4 and 3.2.5). Also with SM1 and SM2 at pulsatile conditions with resistance clamps in the pulmonary arteries, Q_{tot} increased and Q_{svc} decreased strongly when Q_{sh} increased. The offsets (Q_{sh} = 0 l/min) of the Q_{svc} were different, due to inaccuracies in the pressure measurements (about 5 mmHg) and perturbations in the pulmonary resistances.

The flow was more homogeneously distributed (figure 3.2.4, Q_{rpa} \approx Q_{lpa}) than at continuous conditions with shunt model 1. An increase of Q_{sh} resulted in a decrease of Q_{lpa} at continuous and in an increase of Q_{rpa} at pulsatile conditions. This difference can be explained by the position of the pulmonary resistances. As mentioned before, at continuous conditions, the pulmonary resistance was determined by the porous resistances, which led to recirculation in the lungs. At pulsatile conditions, the clamps in the pulmonary arteries determined the pulmonary resistances. No recirculation occurred, so Q_{lpa} did not decrease when Q_{sh} increased. Figure 3.2.5 shows that there was no significant difference between the flow distribution with SM2 at pulsatile and continuous conditions.

It is remarkable that the flow through the right and left pulmonary arteries did not exactly agree with the total entering flow. A possible explanation are inaccuracies in the measurements, it was difficult to obtain an exact reproducibility.

The pressures in the SVC and the LPA did not differ between the conditions with SM1 and SM2 (figures 3.2.6 and 3.2.7). However, the P_{rpa} became higher with SM1 than with SM2 which agreed with the result that with SM1 more flow went to the right than to the left in contrast with SM2 where more flow went to the left than to the right pulmonary artery.

It can be concluded that the pulsatility character of the flow did not influence the global fluid dynamics and that the clamps in the pulmonary arteries almost mainly determined the flow distribution. So, to the conclusion drawn in section 4.1.2, that the configuration of the shunt models mainly determines the flow distribution, must be added that the resistance of the pulmonary arteries determine what the influence of the configuration is.
4.3 Computational fluid model at continuous conditions

4.3.1 The shunt model fluid dynamics based on in vitro obtained data

Three-dimensional computational models have allowed insight in the local fluid dynamics. The results of the simulations based on in vitro data (§3.3.1) showed much resemblance with the results from experimental work (§3.1). Table 4.3 gives an overview of both results.

Table 4.3, Results of experiments and simulations.

<table>
<thead>
<tr>
<th>SM1</th>
<th>$Q_{sh}$ (l/min)</th>
<th>$P_{svc}$ (mmHg)</th>
<th>$P_{sh}$ (mmHg)</th>
<th>$P_{rpa}$ (mmHg)</th>
<th>$Q_{rpa}$ (l/min)</th>
<th>$Q_{lpa}$ (l/min)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Experiment</td>
<td>0.25</td>
<td>10.41</td>
<td>10.70</td>
<td>10.26</td>
<td>0.50</td>
<td>0.47</td>
</tr>
<tr>
<td>Simulation</td>
<td>10.38</td>
<td>10.41</td>
<td>10.28</td>
<td>0.50</td>
<td>0.47</td>
<td></td>
</tr>
<tr>
<td>Experiment</td>
<td>1.0</td>
<td>11.12</td>
<td>16.0</td>
<td>11.98</td>
<td>1.12</td>
<td>-0.007</td>
</tr>
<tr>
<td>Simulation</td>
<td>10.91</td>
<td>10.71</td>
<td>11.16</td>
<td>0.72</td>
<td>0.39</td>
<td></td>
</tr>
</tbody>
</table>

With the experiments, it was investigated whether there is a difference between the effects of SM1 and SM2. A difference between the SM1 and SM2 is noticed, both in the experiments as in the numerical simulations. However, both models do not predict a good effect in a patient. The data obtained with the simulations agree much with the experimental results. When table 4.3 at $Q_{sh} = 0.25$ l/min is regarded, it is noticed that for this low SPS flow the flow distribution was approximately equal for the LPA and the RPA. The particle path lines in figure 3.3.2 show how this was established. It is remarkable that the SPS flow had the tendency to go to the LPA and most of the SVC flow went to the RPA. The SPS flow appears to be blocked by the SVC flow. This is different at $Q_{sh} = 1.0$ l/min. The particle path lines were less organised. The SPS flow in SM1 went more to the RPA than to the LPA and it seems that no fluid of the SVC flowed to the LPA. This resulted in an unequal flow distribution between the RPA and the LPA. There was less difference in SM2, due to the parallel geometry. At $Q_{sh} = 1.0$ l/min with both shunt models, the situation appears to be opposite from the low SPS flow, the SPS flow blocked the SVC flow. This is also confirmed by the velocity vectors, figure 3.3.4. The fluid from the SPS has the highest velocity at both shunt models at both conditions ($Q_{sh} = 0.25$ and 1.0 l/min). The particle path lines show that next to the outlet of the SPS vortices develop and the velocity vectors show the low velocity of the fluid at these areas. These vortices form the restriction in SM2 for the fluid coming from the SVC. In SM1 the SVC fluid appears to be more restricted by the high velocity of the SPS flow.

The contours of the static pressure (figure 3.3.3) confirm that the pressure drop between the SVC and the PA’s is very low, see also table 4.3. Where the SPS flow enters the PA, a high pressure developed.

For SM1 at $Q_{sh} = 1.0$ l/min counts that the results from the simulation differ from the experimental results. This is explained by a small difference in the geometry of the SM1 and the mesh of SM1. The mesh had the same dimensions as the experimental used SM except for the corner between the SVC and the SPS. Here the mesh was a little rounded off, so that all volume elements necessary for the finite volume method (FVM) were properly shaped. With these rounded corner the gap size of the SPS outlet was a little larger. The velocity of the fluid was lower, compared with the original SM, resulting in a different flow distribution between the experiments and the simulations.
The difference in flow distribution between the RPA and the LPA at SM1 and to a lesser degree at SM2 at $Q_{sh} = 1.0 \text{ l/min}$ might be due to the low resistance of the PA’s. The majority of the resistance is defined after the PA and during the experiments even a reversed flow was observed at the LPA. To investigate if a placement of the largest contribution of the pulmonary resistance in the PA gives another flow distribution as a result, the simulations with a higher resistance were done.

### 4.3.2 The shunt model fluid dynamics with a higher resistance

Table 4.4 confirms what was expected: the resistance in the PA mainly determines the flow distribution. With the applied resistance there is no significant difference between the two shunt models.

<table>
<thead>
<tr>
<th>$Q_{sh}$ (l/min)</th>
<th>$P_{sh}$ (mmHg)</th>
<th>$P_{lpa}$ (mmHg)</th>
<th>$P_{rpa}$ (mmHg)</th>
<th>$Q_{lpa}$ (l/min)</th>
<th>$Q_{rpa}$ (l/min)</th>
<th>$Q_{svc}$ (l/min)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.25</td>
<td>9.88</td>
<td>9.65</td>
<td>9.65</td>
<td>0.66</td>
<td>0.66</td>
<td>1.06</td>
</tr>
<tr>
<td>SM1</td>
<td>9.81</td>
<td>9.63</td>
<td>9.68</td>
<td>0.66</td>
<td>0.66</td>
<td>1.07</td>
</tr>
<tr>
<td>SM2</td>
<td>10.57</td>
<td>9.85</td>
<td>9.80</td>
<td>0.66</td>
<td>0.66</td>
<td>0.32</td>
</tr>
</tbody>
</table>

The particle pathlines (figure 3.3.6) showed the same pattern as in the previous simulations. Also figure 3.3.8 does not differ much from the previously obtained results, but the velocity in the SVC was higher. This is obvious, because $Q_{svc}$ was higher. The pressure drop between the SVC and the PA’s was low in this case (figure 3.3.7).

### 4.4 Recommendations

During some of the performed experiments, a backflow in the superior vena was observed. It is not likely that this would have happened in vivo. In the physiological situation, there is a complete upper body with a complex regulation system of its pressures and resistances, but of course in vitro it is impossible to model the complete circulation exactly without simplifications. Some recommendations to improve the experimental set-up are given.

In the present set-up, the flow through the SPS is imposed. Physiologically, the pressure generated by the heart is the driving force. Then, the flow is dependent on the impedance of the pulmonary circulation and the resistance (determined by the diameter) and the pressure drop of the SPS. A windkessel system after the centrifugal pump should be added which represents the systemic circulation. When the shunt is branched off after the pump, the effect of a pressure-forced shunt can be investigated.

At the pulsatile conditions, the pressure transducers were not accurate enough to measure the small pressure drops. More accurate pressure transducers are recommended in further research.

The diameter of the shunts in the experiments was 0.5 cm. However, the diameter of the shunts, used in surgical operations depends on different parameters, like the age and the condition of the baby and the preference of the surgeon. Smaller diameters result in higher velocities, which may lead to an improvement of the ejector. Further research with different shunt diameters is recommended.
5 Conclusion

In vitro experiments and CFD simulations were performed to investigate the fluid dynamics in different shunt models. The in vitro experiments consisted of three parts: continuous systemic-to-pulmonary shunt (SPS) flow with rigid and distensible shunt models and pulsatile SPS flow with rigid shunt models. The first CFD model was based on the experiments with continuous SPS flow and the rigid shunt models. With the second model the pulmonary resistance was increased.

At all conditions, with an increasing SPS flow the pressure in the superior vena cava (Psvc) did not become too high (maximum increase of 13.5%). However, the total flow in the pulmonary arteries (Qtot) did not increase much either (maximally 23.7%) with respect to the corresponding strong decrease (87.4%) of the flow in the superior vena (Qsvc). A higher pressure in the superior vena cava led to a higher Qsvc and a higher Qtot and a high pulmonary resistance resulted in the opposite, but the slope of the flow-pressure relationships was not affected.

The CFD model confirmed what the experiments showed: the flow from the SVC was blocked by the flow from the SPS. At low Qsh, the Qsvc flowed mostly into the right pulmonary artery (RPA) and directed the Qsh into the left pulmonary artery (LPA). At high Qsh, the SPS flow blocked the Qsvc and the Qsvc became very low or even negative. Qsh went to both the LPA and the RPA and the Qsvc only went to the PA.

The pulmonary artery resistance and the configuration of the shunt model determined the flow direction. At the experiments with only continuous flows, the major part of the pulmonary resistance was defined after the pulmonary arteries. The resistance in the pulmonary arteries was low and in shunt model 1, a recirculation in the pulmonary arteries developed. At pulsatile conditions the pulmonary resistance was constructed in a more realistic way and the flow distribution between the RPA and the LPA was not influenced by the configuration of the shunt models anymore.

The SPS did not work as an ejector. The results of the experiments and simulations strongly resembled the calculated results of the simplified ejector model (figure 1.6) at the domain 0 < Qsh < 1.0 l/min. The increase of Qtot was small, because Qsvc was blocked instead of entrained. The expected influence of a more physiological configuration on the effect of the ejector was hardly noticeable. The differences in angle and position of the SPS did not result in a preference for one of the two shunt models. Also the distensibility did not improve the effect of the ejector, neither did the pulsatile flow through the SPS.
Acknowledgements

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References


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Appendix A Fontan operation

Two other examples with the final stage of the Fontan procedure are shown: the double outlet right ventricle and tricuspid atresia. In double outlet right ventricle, the two great arteries (aorta and pulmonary artery) both originate from the right ventricle and blood from the left ventricle passes across a ventricular septal defect into the right the total ventricle to reach the great arteries. To create the total cavopulmonar connection, the Fontan operation is performed extracardiacally. The superior (SVC) and inferior (IVC) venae cavae are both connected to the right pulmonary artery (RPA), but a Gore-Tex conduit is used to connect the IVC to the RPA extracardiacally (figure a.1).

"Extracardiac" FONTAN

Figure a.1, The total cavopulmonary connection with double outlet right ventricle. The superior and inferior venae cavae are both connected to the pulmonary artery, but a Gore-Tex conduit is used to connect the IVC to the RPA extracardiacally.

In tricuspid atresia, there is no opening from the right atrium into either ventricle, and in the majority of patients there is no identifiable valve tissue or valve remnant. At the fontan procedure the SVC and IVC are both connected to the pulmonary artery (figure a.2). Oxygen-poor blood is thus directed into the lungs rather than to the left atrium. A patch is placed to prevent blood passing from the right to the left atrium although sometimes a small hole (a fenestration) is deliberately left.

Figure a.2, The total cavopulmonary connection with tricuspid atresia. The superior and inferior venae cavae are both connected to the pulmonary artery. The atrium septal defect is closed to prevent blood passing from the right to the left atrium.
Appendix B  Ejector

In §1.2 the liquid-liquid ejector is regarded (figure 1.5). In this appendix is shown how mass balance and momentum balance lead to equation 1.1.

- Mass balance. At steady state:
  \[ w_1 = w_2 \quad \text{or} \quad \rho_1 \langle \vec{v}_1 \rangle A_1 = \rho_2 \langle \vec{v}_2 \rangle A_2 \]  \hspace{1cm} (B.1)

Subscript 1: variables at surface 1 \((A_1 \ [m^2])\), subscript 2: variables at surface 2 \((A_2 \ [m^2])\),
w: mass flow \([kg/s]\), \(\rho\): density \([kg/m^3]\), v: velocity \([m/s]\)

For an incompressible fluid and from the geometry:
\[ \rho_1 = \rho_2 \quad \text{and} \quad A_1 = A_2 \] \hspace{1cm} (B.2)

Equation (B.1) can be written as:
\[ \frac{\langle \vec{v}_1 \rangle}{\langle \vec{v}_2 \rangle} = \frac{\int_0^{2\pi} \int_0^R v_{\text{sh}} r dr d\theta + \int_0^{2\pi} \int_0^R \beta v_{\text{sh}} r dr d\theta}{\int_0^{2\pi} \int_0^R r dr d\theta} = v_{\text{sh}} (\alpha + \beta - \alpha \beta) \] \hspace{1cm} (B.3)

\(\alpha\): factor for the cross sectional area, \(\beta\): factor for the velocity, \(r\): radius \([m]\), \(R\): radius \([m]\), \(\theta\): angle \([\text{radians}]\), \(v_{\text{sh}}\): velocity in shunt \([m/s]\)

- Momentum balance. When \(F\) is neglected, the component of the momentum balance in the flow direction:
  \[ w_1 \frac{\langle \vec{V}_2 \rangle^2}{\langle \vec{V}_1 \rangle} - w_2 \frac{\langle \vec{V}_2 \rangle^2}{\langle \vec{V}_1 \rangle} + p_1 A_1 - p_2 A_2 = 0 \] \hspace{1cm} (B.4)

\(p_1\): pressure at surface 1, \(p_2\): pressure at surface 2.

The ratios of averages are (assuming flat profiles):
\[ \frac{\langle \vec{V}_2 \rangle^2}{\langle \vec{V}_2 \rangle} = v_{\text{sh}} (\alpha + \beta - \alpha \beta) \] \hspace{1cm} (B.5)
\[ \frac{\langle \vec{V}_1 \rangle^2}{\langle \vec{V}_1 \rangle} = \frac{\alpha v_{\text{sh}}^2 + (1-\alpha)(\beta v_{\text{sh}})^2}{\alpha v_{\text{sh}} + (1-\alpha)(\beta v_{\text{sh}})} = v_{\text{sh}} (\alpha + \beta - \alpha \beta) \] \hspace{1cm} (B.6)

Substituting (B.5) in (B.1):
\[ w_1 = w_2 = (\alpha + \beta - \alpha \beta) \rho v_{\text{sh}} A \] \hspace{1cm} (B.7)

Substituting (B.7) in (B.4) and after some rearrangement:
\[ p_2 - p_1 = \rho v_{\text{sh}}^2 \left( \alpha^2 + \beta^2 - 2\alpha^2 \beta + \alpha^2 \beta^2 + 2\alpha \beta - 2\alpha \beta^2 \right) \] \hspace{1cm} (B.8)

This is the expression for the pressure increase resulting from the mixing of the two streams.
Appendix C  Results: flow-pressure relationships

Figure c.1, \( P_{svc} \) and \( P_{rpa} \) versus \( Q_{sh} \) through SM2 with normal pulmonary resistance at \( P_{svc, 0} = 10, 15 \) and 20 mmHg.

Figure c.2, \( P_{svc} \) and \( P_{rpa} \) versus \( Q_{sh} \) through SM1 with high pulmonary resistance at \( P_{svc, 0} = 10, 15 \) and 20 mmHg.

Figure c.3, \( P_{svc} \) and \( P_{rpa} \) versus \( Q_{sh} \) through SM2 with high pulmonary resistance at \( P_{svc, 0} = 10, 15 \) and 20 mmHg.