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Implementation of intrinsic lumped parameter modeling into computational fluid dynamics studies of cardiopulmonary bypass

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ABSTRACT

Stroke and cerebral hypoxia are among the main complications during cardiopulmonary bypass (CPB). The two main reasons for these complications are the cannula jet, due to altered flow conditions and the sandblast effect, and impaired cerebral autoregulation which often occurs in the elderly. The effect of autoregulation has so far mainly been modeled using lumped parameter modeling, while Computational Fluid Dynamics (CFD) has been applied to analyze flow conditions during CPB. In this study, we combine both modeling techniques to analyze the effect of lumped parameter modeling on blood flow during CPB. Additionally, cerebral autoregulation is implemented using the Baroreflex, which adapts the cerebrovascular resistance and compliance based on the cerebral perfusion pressure.

The results show that while a combination of CFD and lumped parameter modeling without autoregulation delivers feasible results for physiological flow conditions, it overestimates the loss of cerebral blood flow during CPB. This is counteracted by the Baroreflex, which restores the cerebral blood flow to native levels. However, the cerebral blood flow during CPB is typically reduced by 10–20% in the clinic. This indicates that either the Baroreflex is not fully functional during CPB, or that the target value for the Baroreflex is not a full native cerebral blood flow, but the plateau phase of cerebral autoregulation, which starts at approximately 80% of native flow.

1. Introduction

Cardiopulmonary bypass (CPB) remains a standard technique in cardiac surgery. However, cerebral hypoxia and an increased risk for strokes are among the main complications during CPB. The cannula jet has been identified as one of the main reasons due to altered flow conditions in the aortic arch and the sandblast effect (Kapetanakis et al., 2004; Scarborough et al., 2003; Verdonck et al., 1998). It has also been shown that the risk for strokes is significantly increased for patients with impaired cerebral autoregulation (Ono et al., 2012). Since many of the elderly suffer from impaired autoregulation (van Beek et al., 2008), cardiac surgeons face the challenge of an ageing population with the potential to further increase the risk for neurological complications during CPB.

Due to the nature of CPB, it is hardly possible to study the underlying mechanisms for these complications in vivo and the analysis is currently limited to in vitro or in silico data (Fukuda et al., 2009; Kaufmann et al., 2009a, 2009b; Laumen et al., 2010; Markl et al., 2007; Tokuda et al., 2008). Numerical simulations provide the possibility of analyzing CPB conditions and have been widely applied to study these phenomena, but they are strongly affected by the boundary conditions assumed during CPB, which differ from clinically accessible physiological values (Fukuda et al., 2009; Kaufmann et al., 2009a, 2009b; Tokuda et al., 2008). While the first studies in this field used simple pressure boundary conditions, more recent analyses used boundary conditions based on peripheral resistance (Assmann et al., 2012; Benim et al., 2011; Gallo et al., 2012; Kaufmann et al., 2012a, 2012b).

In addition to the usual limitations of in vitro and in silico analyses, hardly any of these studies have included the complex mechanisms of cerebral autoregulation, which is defined as the body's intrinsic ability to provide sufficient cerebral blood flow despite changes in cerebral perfusion pressure (Bellapart et al., 2010; van Beek et al., 2008). The cerebral autoregulation acts by adapting the cerebrovascular resistance (Panerai, 1998) and compliance to changes in perfusion pressure, which is achieved by myogenic, neurogenic or metabolic mechanisms (Chillon and Baumbach, 2002; Paulson et al., 1990). Thereby, the cerebral blood flow is kept constant over a range of cerebral perfusion pressures.

In Kaufmann et al. (2012a, 2012b), we presented a mathematical approach to incorporate static cerebral autoregulation into Computational Fluid Dynamics (CFD) studies of CPB by using the relationship between the cerebral perfusion pressure and the cerebral blood flow. While this already delivers feasible results based on the level of autoregulation and cerebral perfusion pressure, this method lacks the time-dependent adaptability of cerebrovascular resistance and compliance.

A way of including this mechanism is by coupling lumped parameter simulations of the circulatory system into CFD.

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Groundbreaking in this field is the model presented by Kim et al. (2009), who developed and implemented autoregulation mechanisms into CFD simulations of physiological flows and tested them during exercise. This model also includes the Baroreflex (Kim et al., 2010), which acts on the resistance, compliance, heart rate and cardiac contractility. However, Kim et al. only incorporated lumped parameter modeling for physiological conditions. Since the heart is clamped during CPB, the model cannot be applied to CPB without adaptations. In this study, we partly adapt this approach to develop an intrinsic lumped parameter model within our CFD model and apply it to CFD studies of flow conditions during CPB.

The model creation and simulation setup was based on Kaufmann et al. (2012a, 2012b). A 3-dimensional CAD model of the human vascular system including aorta and greater vessels was created from imaging data. For simulations of CPB conditions, a 24 FR standard CPB outflow cannula was placed virtually in the CAD model. Prior to the numerical studies, a PIV study was initially performed to validate the effect of vascular resistance during CFD for physiological and CPB conditions. After validation, a simple lumped parameter model was created according to Kim et al. (2010, 2009) and implemented into the CFD studies. It was tested for physiological conditions and then applied to CPB simulations. Furthermore, the baroreflex was implemented to analyze the effect of cerebral autoregulation on cerebral blood flow during CPB.

2. Materials and methods

2.1. Basic model creation and mesh generation

The process of model building and mesh generation was the same as previously reported (Kaufmann et al., 2012a, 2012b). Based on imaging data, a 3-dimensional model of the human cardiovascular system was generated. The final model consisted of the aortic arch including the descending aorta and the subclavian arteries to represent systemic circulation. The cerebral vessels were represented by the carotid and vertebral arteries. For analysis of CPB conditions, a 24 FR standard CPB outflow cannula was virtually placed in the ascending aorta, representing a standard positioning technique during CPB.

A tetrahedral mesh was generated within the geometry (ICEM CFD 14.0, Ansys Germany Inc., Otterfing, Germany). Three layers of prismatic elements were furthermore created around the boundaries representing the vessel walls to resolve the boundary layers of the flow. A mesh independency study with element numbers between 0.6 and 11.1 million was initially performed to minimize discretization errors. The final mesh consisted of approximately three million elements.

2.2. Simulation setup

Using these boundary conditions, transient numerical simulations of blood flow in the cardiovascular system were performed using commercial software (ANSYS CFX 14.0, Ansys Germany Inc., Otterfing, Germany). A Specified Blend Factor of 0.75 was set as the advection scheme. With this scheme, at least 75% of all mesh elements are solved second-order-accurate, while up to 25% may be solved first-order-accurate. The turbulence settings were set according to Kaufmann et al., (2012a, 2012b) using the Shear Stress Transport turbulence model implemented in ANSYS CFX. The time step size was set to 0.01 s. Up to 3 internal iterations were solved per time step until the average changes in the transport equations were smaller than the specified convergence target of 1e-4.

The vessel walls were considered rigid with a no slip conditions and blood was modeled as a non-Newtonian fluid. The hematocrit was set to 44%, which resulted in a density of 1056.4 kg/m^3.

The time for one cardiac cycle was set to 0.8 s with a systolic time of 0.3 s. The inlet representing the heart was set using a simplified inlet profile. For analysis of CPB conditions, a continuous flow of 5 l/min through the CPB outflow cannula was set instead of the pulsatile flow from the native heart.

2.3. Validation

Validation of the model is performed with an approach similar to a previous study (Laumen et al., 2010). The main goal was to validate the effect of vascular resistances within CFD simulations.

Using a slightly adapted CAD model of the cardiovascular system, a 3-dimensional model of this system was printed using rapid prototyping. The model was fixated in a box which was filled with silicone (Elastosil® RT601, Wacker Chemie). After hardening of the silicone, the printed core was removed to create a transparent model of the cardiovascular system. The process of model building is shown in Fig. 1.

In addition to this model, throttles were crafted to set a pressure drop behind each vessel. Since all throttles had the same resistance, a non-physiological flow distribution is expected. The results are therefore discussed only in terms of comparison between experiments and simulations and not in terms of physiological feasibility.

Two models were crafted: one with native flow from the heart and one with an implemented cannula for CPB. Using these models, Particle Image Velocimetry (PIV) studies were performed to validate the flow fields obtained in-silico.

Stereo 3D PIV was performed for both models in focal planes with a distance of 2.5 mm between. The results were averaged over 300 images in each plane.

For simplification, the same pressure drop was assumed for all vessels, resulting in non-physiological flow distributions. The flow in the PIV experiments was also averaged to allow a steady state analysis in the experiments. Additionally, geometric adaptations to the regular model were necessary. To achieve a good agreement in boundary conditions, separate validation simulations were performed in the same geometry and with the same pressure drop as in PIV. Quantitative comparison of experimental and numerical results was performed using significance tests.

A water–glycerol mixture of 56.4/43.6 (m_{Gl}/m_W) tempered at 45 °C was used. Thereby, the refractive index of the silicone ($n=1.4095$) could be nearly matched and an average Newtonian blood viscosity was achieved in the test fluid. The PIV setup is depicted in Fig. 2.

2.4. Implementation of lumped parameter modeling

The simplest way to describe the flow through a vessel using lumped parameter modeling is an electric circuit of one vascular compliance C and i parallel resistances R_i, for each represented vessel, with i being the individual vessel. This lumped parameter model leads to an ordinary differential equation (ODE) which is given in which is given in (1).

$$\dot{p}(t)+\frac{1}{RC}p(t)=\frac{1}{C}q(t) \tag{1}$$

This ODE can be solved for p in each time step n by using the Eq. (2), with q^n being the total flow. Using (2), the pressure at each vessel outlet is given dependent on the vascular resistance and compliance.

$$p^n=p^{n-1}e^{-(\Delta t/RC)}+q^nR(1-e^{-(\Delta t/RC)}) \tag{2}$$

The resistance in each vessel is given in (3).

$$R_i=\frac{R_{ves}}{n_i} \tag{3}$$

R_{ves} is the total resistance and n_i is the ideal percentage of cardiac output flowing through vessel i. The values for n_i and R_{ves} are calculated based on the idealized mass flow to each vessel from Laumen et al. (2010). Using Eqs. (2) and (3) as well as the blood density ρ, the blood flow to each vessel can be calculated as shown in (4).

$$\dot{m}_i=\frac{p(t)}{R_i}\rho \tag{4}$$

This mass flow calculation can be applied as a boundary condition to each individual outgoing vessel.

After the intrinsic lumped parameter model was established for physiological flow conditions, it was applied to the pathological flow conditions during cf-CPB.

During CPB, the cerebral vessels were modeled using the aforementioned lumped parameter approach, while the outlets representing the systemic circulation (descending aorta and subclavian arteries) were modeled using the Darcy Weißbach Eq. (5) according to Kaufmann et al. (2012a, 2012b), which calculates the pressure drop based on the normal velocity U_n, the fluid density ρ and a dimensionless loss coefficient L, resulting in the model setup shown in Fig. 3.

$$\Delta P=\frac{1}{2}L\,\rho\,U_n^2 \tag{5}$$

2.5. Baroreflex

One of the key aspects of autoregulation is the baroreflex, which allows adaptation of the cerebrovascular resistance and compliance based on the cerebral perfusion pressure. If the desired pressure differs from the actual pressure, the sympathetic and parasympathetic nervous system start to change R and C in order to restore sufficient cerebral blood flow. A way of modeling this has been presented by Kim et al. (2010). The sympathetic activity n_s and parasympathetic activity n_p are following equations (6a) and (6b) (Ottesen et al., 2004):

$$n_s=\frac{1}{(1+(\overline{p}/p_t))^{\nu}} \tag{6a}$$

$$n_p=\frac{1}{(1+(\overline{p}/p_t))^{-\nu}} \tag{6b}$$