KaHMo: A Patient Specific Model of Human Cardiac Fluid Dynamics

Torsten Schenkel, Sven Donisi, Herbert Oertel
Institute for Fluid Mechanics, University of Karlsruhe

Introduction

The Institute for Fluid Mechanics at the University of Karlsruhe works on a numerical model of the simulation of the fluid dynamics in the human heart. This modular model will eventually cover the whole heart with the adjacent large vessels. At the moment the focus is on the left ventricular flow.

Setting itself apart from other works in cardiac function simulations, which mostly cover electrodynamics or structural dynamics approaches or are based on generic data, the Karlsruhe Heart Model (KaHMo) is based on patient specific MR data. This allows the simulation of a specific heart and evaluation of different hearts, e.g. healthy hearts with different ejection fractions, or the differences between the function of the healthy and the diseased heart as well as pre- and post-surgical comparison.

Methodology

The KaHMo is based on a general separation of the heart into active and passive parts. While the heart muscle is exerting forces onto the blood and thus forces the flow and can therefore be considered active, the valves and vessels are moved by the forces imposed by the blood flow and are treated as passive. This separation and the treatment of the myocardium in the ventricle walls as active is what makes a patient specific model possible, since then there is no need for a fluid structure interaction approach which would rely on information about the muscle fiber structure not obtainable in vivo. What we can acquire by standard non-invasive measurement and diagnosis is the geometry and the movement of the ventricle walls. In our work this information is gained by MR (magnetic resonance) imaging.

From the MR dataset the inner surface of the ventricle (fluid volume) is reconstructed for several points of time during one heart cycle. From this surface a numerical grid is generated for every time step present in the measurement between which the ventricle's motion is interpolated.

The real heart valves are passive elements that are moved by the pressure forces exerted by the flow. The movement of the valve is governed by complicated, non-linear and non-isotropic structural mechanics. To our knowledge there is no fluid structure interaction approach currently, which is able to handle this kind of problem. In order to be able to simulate the ventricle flow the valves are modeled by a simplified 2-D projected opening area. Valve opening times are based on the change of the ventricular volume over time. The resulting velocity profiles some diameters behind the valve are matched to the velocity profiles measured by MR flux or Echo Doppler.

The non-Newtonian properties of the fluid are taken into account using a modified cross model by Perktold adapted to the properties of blood by as measured by Liepsch.

Boundary conditions for the numerical model are firstly the movement of the wall as prescribed, and secondly relative pressure boundaries at the inflow and outflow tracts. While the venous pressure shows only small pulsation and is modeled as a constant relative pressure of 500 Pa, the aortic pressure is highly dependent on the flow itself and cannot be prescribed. The effect of the circulatory system is currently modeled in
a highly simplified way using a velocity dependent pressure drop in the outflow tract. This resistance is adapted to produce the physiological pressure.

The flow is simulated using the commercial Computational Fluid Dynamics (CFD) code StarCD©. The models used are unsteady, laminar, viscous flow with shear rate dependent viscosity and moving grid formulation. Differencing methods: Monotone Advection and Reconstruction Scheme (MARS) space and Euler implicit time discretisation, Pressure Implicit with Splitting of Operators (PISO) algorithm.

Results
In early diastole the inflow through the mitral orifice produces a so-called ring vortex. This vortex is formed briefly after the opening of the mitral valve. In the further process of the diastole this ring vortex grows asymmetrically and bifurcates. In the given ventricle this results in a main vortex with a counter clockwise rotation and a second vortex in the apex, which assists the flushing of old blood out of the ventricle's tip. At the end of the filling the flow orientation is towards the aortic tract and thus prepared for ejection.

The ring vortex configuration might be expected as in the unsteady, accelerated inflow through a small orifice into a large container with stagnant flow. This initially symmetrical elongated torus travels and grows in axial direction while growing radially with a sinistral preference. This right hand dominance leads to a secondary twist which rotates the vortex itself until the former right half of it fills almost the whole ventricular cavity. This one sided preference can be explained by the asymmetry in the ventricles movement, where the outer myocardium moves to a higher degree than the septum wall.

In later diastole additional vortices are induced by the main vortex. These counter rotating smaller vortices are of considerably lower velocity and kinetic energy and dissolve into the main vortex during initial systole during which the whole vortex structure disintegrates.

The outflow of blood into the aorta is formed as a jet flow, with a velocity maximum in the centre of the aortic valve.

Conclusion and Prospects
The method was proven to be an effective means for evaluation of intraventricular flow for a specific heart. Work currently under progress on the application of the method on ventricles with an aneurysm after myocardial infarction before and after surgical aneurysmectomy will provide insights in the fluid mechanical efficiency of state of the art surgical techniques.