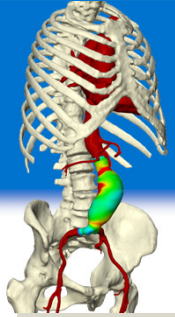

The influence of including the renal arteries in a CFD simulation of the blood flow through an abdominal aortic aneurysm (AAA)

STUDENT PROJECT
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Introduction

For a better understanding of the origin of an abdominal aortic aneurysm (AAA) and furthermore for predicting future changes in the aneurysm, CFD simulations of the blood flow through the investigated part of the aorta can be very helpful. For this kind of simulation the renal arteries are usually being neglected. The object of this project is to investigate the influence of the renal arteries on the flow. For this, two simulation models are created - one with renal arteries and one without. To consider patient specific influences on the simulation, two different patient-specific CT scans have been used leading to four different models in total.

Methods

The lumen geometry of the AAA is segmented from a CT scan in *Mimics* and subsequently smoothed. Two versions of the AAA are being segmented, one with the renal arteries and one without. For a more stabilized outflow, all outlets are extended with cylinders of a length three times the diameter of the arteries. The final volume is meshed in *Harpoon* with a base level size of about 1.53. While the main aortic surface is meshed on this level, level 2 is chosen for the upper renal artery and level 3 for the lower renal artery to avoid stability issues. For an improved resolution of the wall shear stress, a boundary layer with an initial cell height of 0.1, 4 layers and an expansion of 1.35 is added. This leads to a total amount of cells of 183 642 for model a) without renal arteries and 244 607 with renal arteries, for model b) of 165 529 and 204 483, respectively.

For the inflow boundary condition a Womersley inflow profile is used, representing a pulsatile flow rate of 4.0 liters/min. According to **[1]**, Windkessel boundary conditions are set at the outlets to model the behavior of the flow downstream the outlets. For the renal arteries a combined outflow of 20% of the total outflow is assumed. The simulation is performed with the in-house code *baci*.

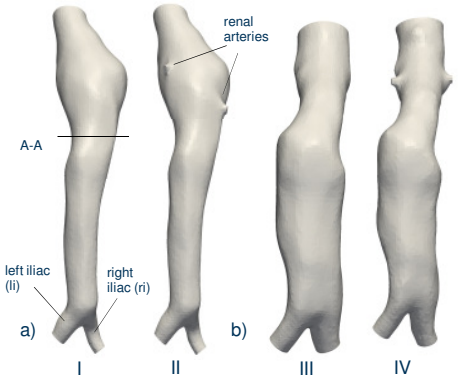

Figure 1 *Geometries of the two patient specific AAAs, both with and without renal arteries.*

Results

Model IV does not converge, even after several attempts with different mesh level sizes in the perimeter of the renal arteries. Inside the renal arteries, both pressure and flow velocity quickly reach unrealistically large values. Because of this, only models I and II will be thoroughly discussed hereafter. Model III will be neglected, as there are no simulation results available for comparison.

Flow rates

The flow through the inlet and outlet patches is given in table 1 for the maximum blood flow at t = 1.12s. The values are in agreement with continuity. In addition, the flow through the outlets of the renal arteries matches the prescribed boundary condition, i.e. that 20% of the blood flows out through the two renal arteries. The reduction of the blood flow through the iliac arteries is equally distributed to both arteries.

	Inlet	Left Iliac	Right Iliac	Left Renal Artery	Right Renal Artery
Model I	162 339	72 531	89 731	-	-
Model II	162 030	57 743	71 951	14 838	17 331

Table 1 *Flow rate [mm^3/s] at the iliac arteries and the renal arteries*

Velocity profile

The velocity profile of models I and II are compared at different slices of the model. Figure 2 shows the same profile characteristic for both iliac arteries, only the value of the velocity differs (also see flow rates). In contrast, during the systolic phase there are differences in the velocity profiles at the slice below the renal arteries (A-A). These vortex structures are caused by the flow deflection of the renal arteries. These effects are significant in the area of the renal arteries but are dissolved at the iliac arteries.

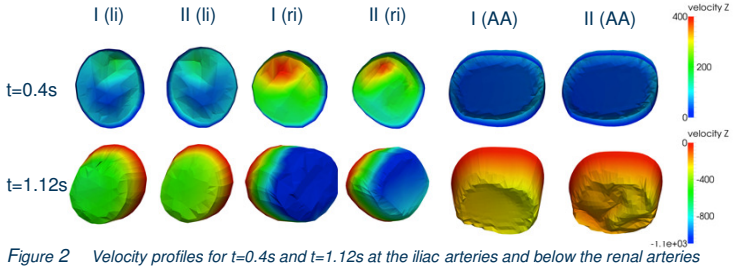

Figure 2 *Velocity profiles for t=0.4s and t=1.12s at the iliac arteries and below the renal arteries*

Wall shear stress

The wall shear stress is investigated at t = 0.4s, the maximum of the backflow during the diastolic phase of the heart cycle and at t = 1.12s, the maximum flow rate. The maximum wall shear stress of model I is located at the right iliac artery for both timesteps. For model II, it is at the base of the renal arteries. Calculated values of the wall shear stresses at the right iliac artery for both models as well as at the left renal artery for model II is given in table 2.

The wall shear stresses at the left iliac artery decreases for model II – the difference to model I is 33.4% for the diastolic phase and 43.4% for the systolic phase. However, this can be explained with the decreased blood flow through the iliac arteries for model II, as 20% of the blood is leaving the domain through the renal arteries, which is not considered in model I (see table 1). Hence, reduced wall shear stresses are in line with the expectations.

Analyzing the wall shear stress for model II, one can see that the maximum values at the renal arteries exceed the maximum values at the iliac arteries by a factor up to 10. This has to be considered, as according to **[2]** the tissue can be expected to be damaged permanently if the wall shear stress reaches values of about 37.9 ± 8.5 Pa for an hour or more.

In the rest of the domain the differences of the wall shear stress are mostly negligible.

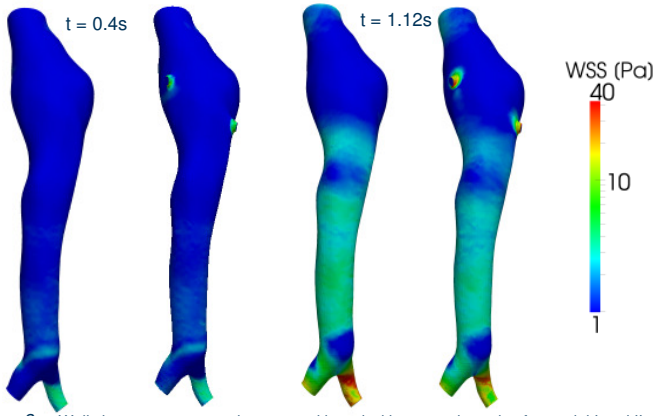

Figure 3 *Wall shear stress at two timesteps with and without renal arteries for model I and II*

	Right iliac artery, I	Right iliac artery, II	Left renal artery, II
t = 0.4s	4.97 Pa	3.31 Pa	35.30 Pa
t = 1.12s	70.56 Pa	39,93 Pa	133.75 Pa

Table 2 *Maximum wall shear stresses at the left iliac artery for both models and the upper renal artery for model II*

Conclusion

The simulations show the increasingly unstable behavior of the models including the renal arteries. While for models I and II a stable simulation has been achieved after several iterations, this has not been possible for model IV – all created meshes diverge. In addition, the required number of cells to model the domain increases largely, e.g. about 33.2% or about 60 965 cells for model II compared to model I, leading to increasing computational times.

Comparing the results of models I and II, the differences caused by adding the renal arteries are negligible in major parts of the domain. However, the maximum WSS and flow rate, especially at the iliac arteries, largely decrease. As the flow rate in model I is not adapted to the additional outflow through the renal arteries in model II, the larger wall shear stresses in model I are a direct result of the larger flow rates in the iliac arteries. However, the renal arteries have a strong influence on the flow profile only in the area of the renal arteries but not downstream in the iliac arteries.

Overall, the simulation shows that the assumption to neglect the renal arteries is reasonable. However, one uncertainty is the wall shear stress near the renal arteries. The values reach critical values over most of the heart cycle according to **[2]**, leading to permanent tissue damages. This has to be further analyzed to assure, whether these large values are physically determined or simply caused by numerical errors.

REFERENCES:
[1] Ismail, Mahmoud, Wall, Wolfgang A., Gee, Michael W. (2013), Adjoint-based analysis of windkessel parameters for patient-specific vascular models, Journal of Computational Physics.
[2] Fry, Donald L. (1967), Acute Vascular Endothelial Changes Associated with Increased Blood Velocity Gradients, Journal of Circulation Research.

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