Rhode, S., Paul, M., Martens, E. and Campbell, D.F. (2011) *Blood flow dynamics in patient specific arterial network in head and neck.* In: 2nd International Conference on Mathematical and Computational Biomedical Engineering - CMBE2011, 30 Mar - 1 Apr 2011, Washington, D.C., USA.

2nd International Conference on Mathematical and Computational Biomedical Engineering - CMBE2011
March 30 - April 1, 2011, Washington D.C, USA
P. Nithiarasu and R. Löhner (Eds.)

Blood flow dynamics in patient specific arterial network in head and neck

S. Rhode*, M. C. Paul†, E. Martens and D. F. Campbell*****
*†School of Engineering, University of Glasgow, Glasgow G12 8QQ, UK, *st.rhode@gmail.com,
†Manosh.Paul@glasgow.ac.uk
**Department of Mechanical Engineering, University of Applied Sciences, 76133 Karlsruhe, Germany
***Department of Oral & Maxillofacial Surgery, Queen Margaret's Hospital, Fife, UK

SUMMARY

This paper shows a steady simulation of blood flow in the major head and neck arteries as if they had rigid walls, using patient specific geometry and CFD software FLUENT®. The Artery geometry is obtained by CT–scan segmentation with the commercial software ScanIP™. A cause and effect study with various Reynolds numbers, viscous models and blood fluid models is provided. Mesh independence is achieved through wall y^+ and pressure gradient adaption. It was found, that a Newtonian fluid model is not appropriate for all geometry parts, therefore the non–Newtonian properties of blood are required for small vessel diameters and low Reynolds numbers. The k–ω turbulence model is suitable for the whole Reynolds number range.
Key Words: *patient specific model, blood flow dynamics, non–Newtonian model*

1 INTRODUCTION

A better understanding of patient specific blood flow should inform pharmacodynamic simulations of regional drug concentrations, for instance intra arterial catheter placement positioning in chemotherapy for cancer of the head and neck. The ongoing development in computation resources allow engineers to simulate blood flow in vessels in a more realistic way then one decade ago. In contrast to ideal geometry, where many assumptions are required, the patient specific geometry, which is obtained from CT or MRI–scans [1, 2], offers detailed knowledge of flow dynamics including three dimensional vortices. This paper contains a cause and effect study of boundary conditions and material models for a steady simulation with rigid walls and provides conclusions, how to ensure the quality of CFD simulations in a patient specific model.

2 STEADY SIMULATION

The blood flow simulation in head and neck vessels with patient specific geometry was started with a model of the right common carotid artery, shown in Fig. 1. The geometry does not contain the major neck veins, hence the pressure value is not directly comparable with blood pressure measures. It is desirable to include as many geometrical details as possible for further studies, meaning major neck veins and a model including smaller peripheral arterial and venous blood vessels. The Reynolds number was calculated on a cross section near the pressure inlet bc with the velocity magnitude. The geometry was created with the software ScanIP™[3]. A patient's head

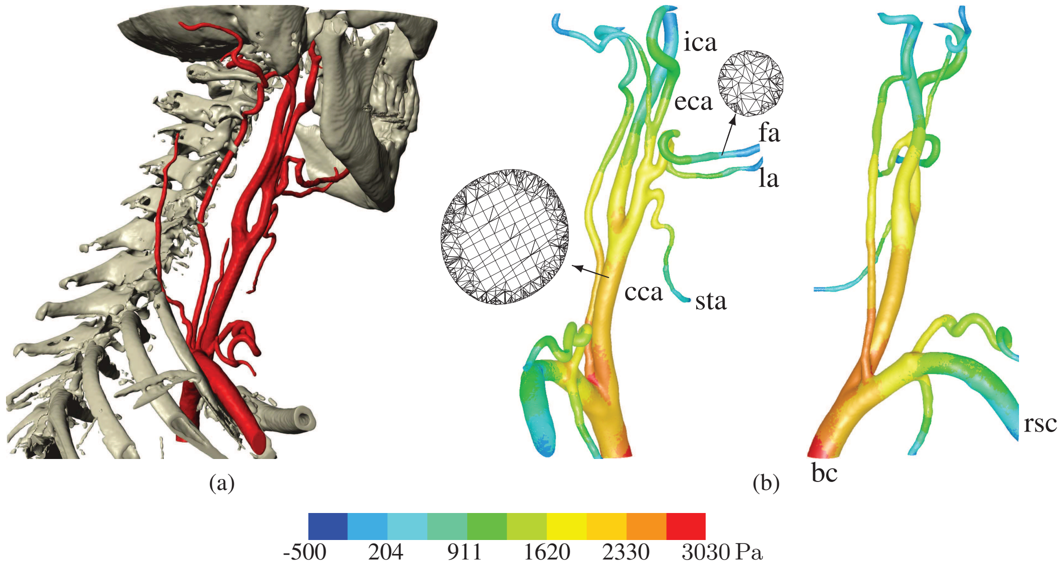


Figure 1: Artery and skull after segmentation (a). Total pressure on the wall for the highest Reynolds number $Re = 3972$. $k-\omega$ turbulence and blood as Newtonian medium was used (b). The pressure inlet is bc–brachiocephalic artery. All the other limits are pressure outlets, rsc–right subclavian artery, cca–common carotid artery, sta–superior thyroid artery, la–lingual artery, fa–facial artery, eca–external carotid artery and ica–internal carotid artery.

and neck CT–Scans were segmented to generate a volume mesh. Using gray scale values, vessels were marked throughout the slices and segmented into a volume. The volume was imported into GAMBIT [4] for remeshing. The CFD simulation was solved by FLUENT®. Blood flow in the human body is pulsatile. In general, a transient CFD simulation takes a very long computational time and requires extensive memory and disk capacity in comparison with a steady simulation. A cause and effect study is compulsory to set up a realistic transient simulation with appropriate parameters. Therefore, it is good to begin with a steady simulation to study the effects of various meshes, turbulence models and material models.

The mesh consists of hexahedron elements in the artery core and tetrahedron elements for the vessel wall. Due to this separation, the core elements have a perfect quality, the skewness being close to zero. The focus was on the tetrahedron elements next to the wall. To resolve the boundary layer, it is required to create a fine mesh next to the wall. The mesh refinement and independence test was done by wall y^+ adaption and pressure gradient adaption in FLUENT®. For the most critical case with the maximum Reynolds number, a value of $y^+ = 5.9$ was achieved, which is slightly above the recommendation of $y^+ < 5$, found in [5] and [6, chapter 12.11.1]. The impact on the simulation results, during the mesh adaption process, was monitored by the velocity magnitude of the bc–inlet and ica–, fa–outlets, shown in Fig. 2. After finding an appropriate mesh, a series of simulations was made. It was decided to vary the Reynolds number in eight steps at the pressure inlet bc in a typical range $676 \leq Re \leq 3972$, given by [7, chapter 3] for the brachiocephalic artery. In order to simplify the simulation, fluid structure interaction was not considered, hence rigid walls were set up for this model.

First, the fluid model of blood was Newtonian and the $k-\omega$ turbulence model was used. In [6, chapter 7.2.2] best practice guidelines are described for an approximate setup for the turbulent kinetic energy k and specific dissipation rate ω for fully developed turbulent pipe flows. The range of Re extends from laminar over transitional to turbulent, measured by the critical Reynolds number for pipe flows $Re = 2300$. However, in pulsating blood flow it is rather improbable that full developed turbulent flow will occur. Neither the entrance length nor the time are long enough

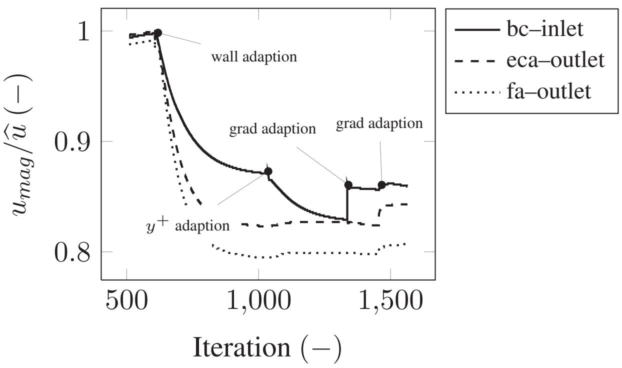
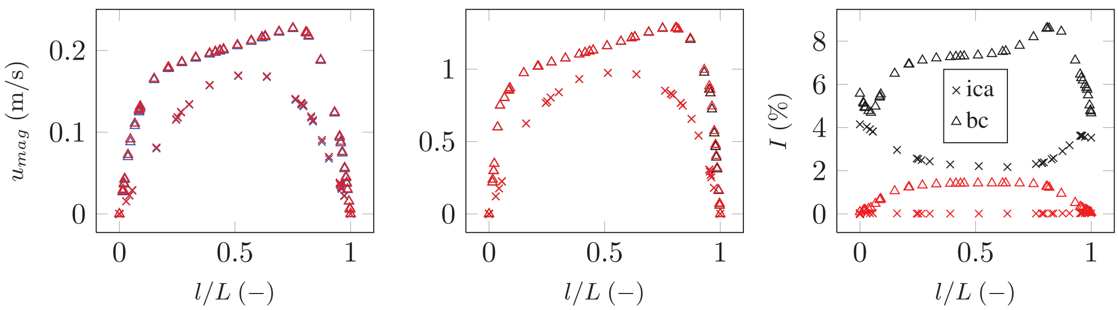


Figure 2: The mesh quality is examined by monitoring mass averaged velocity at pressure inlet and two pressure outlets, using the $k-\omega$ turbulence model, taking blood as a Newtonian medium with the highest Reynolds number, $Re = 3972$. The velocity is normalised for each variable with their maximum value. The first mesh adaption for all cells adjacent to the wall results in decreasing velocity values. The change after the wall y^+ adaption in the smaller arteries (eca and fa–outlet) is marginal, the maximum value of wall y^+ is 5.9. The following velocity gradient adaption was stopped when the value change was below 3 %.

for a fully established turbulent flow. Nevertheless low turbulent effects will be present and have to be taken into account. In a transient simulation, choosing between laminar and turbulent is required in the pre processing. Later on, simulations were done with laminar viscous model and the non–Newtonian power law fluid model. The difference between the $k-\omega$ model in a standard or SST version to laminar viscous model is negligible for low Reynolds numbers, Fig. 3a. The $k-\omega$ model is appropriate for low Reynolds number flow with standard or SST option. The difference between both $k-\omega$ models on high Reynolds number is marginal, Fig. 3b.



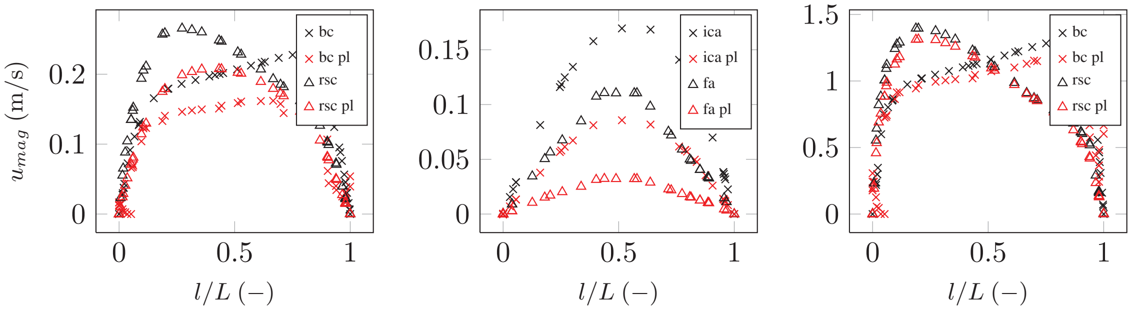
(a) $Re = 676$, black: $k-\omega$, blue: laminar, red: SST $k-\omega$

(b) $Re = 3972$, black: $k-\omega$, red: SST $k-\omega$

(c) black: $Re = 3972$, red: $Re = 676$

Figure 3: Velocity profiles of line probe ica and bc for low and high Reynolds numbers with various viscous model approaches, Fig. (a) and (b). In (a), the difference between booth k–ω models and laminar viscous model is negligible, contrary to (b). In Fig. (c) the turbulence intensity is shown. The blood fluid model is Newtonian.

Blood is a non–Newtonian fluid. Its viscosity depends on the shear rate. For small shear rates, the viscosity increases. Approximately from $\dot{\gamma} > 100\,\mathrm{s}^{-1}$, the viscosity is constant as for a Newtonian fluid, dealt with in [8, chapter 1.2.2]. In [9, chapter 6.4.2], different fluid models are introduced with typical parameters for blood. Every model has advantages and disadvantages, and a comparison is studied in [10]. It was decided to use the power law model for a comparison between Newtonian and non–Newtonian fluid. Fig. 4 shows the influence of the viscous assumption on the simulation results. For small vessels on small Reynolds numbers it is not valid to use a Newtonian model, shown by Fig. 4b. The influence decreases with higher Reynolds numbers and increasing vessel diameter, Fig. 4a and (c). Further studies with various non–Newtonian models are required.



(a) Inlet pressure 150 Pa

(b) Inlet pressure 150 Pa

(c) Inlet pressure 3000 Pa

Figure 4: Velocity profiles with various blood models. Black markers show blood as a Newtonian medium, red markers as non–Newtonian power law. The non–Newtonian approach results in lower velocity values, due to more viscosity on low shear rate. This effect is more relevant in low Reynolds number flow and in small vessels. The difference between both material models is approximately $50\,\%$, shown in Fig. (b). In contrast, Fig. (c) shows approximately similar functions. The length of line–bc is $L = 0.0121\,\mathrm{m}$, of line–rsc $L = 0.0093\,\mathrm{m}$, of line–ica $L = 0.0057\,\mathrm{m}$ and of line–fa $L = 0.0029\,\mathrm{m}$.

3 CONCLUSIONS

A mesh independent solution is produced with wall y^+ and pressure gradient adaption in FLUENT®. A maximum value of $y^+ = 5$ is desirable. The $k - \omega$ turbulence model is able to provide approximately similar results as the laminar viscous model for low Reynolds numbers. For further simulations the Newtonian fluid model approach is not desirable. The small arteries with low Reynolds number values require a non–Newtonian fluid model with increasing viscosity on low shear rates. A study of the fluid structure interaction is necessary to estimate the influence of the vessel deformation on the simulation results.

REFERENCES

[1] C. A. Taylor and C. A. Figueroa. Patient-specific modeling of cardiovascular mechanics. *Annu Rev Biomed Eng*, 11:109–134, 2009.

[2] T. Schenkel, S. Krittian, K. Spiegel, S. Höttges, M. Perschall, and H. Oertel. The Karlsruhe Heart Model KaHMo: A modular framework for numerical simulation of cardiac hemodynamics. In *IFMBE Proceedings Volume 25/4, World Congress on Medical Physics and Biomedical Engineering, September 7-12, 2009, Munich, Germany*, pages 615–618. Springer, September 2009.

[3] Simpleware LTD, Innovation Centre Rennes Drive Exeter EX4 4RN, UK. *ScanIP™, +ScanFE™ and +ScanCAD™ Reference Guide*, 2010.

[4] Fluent Incorporated, Centerra Resource Park 10 Cavendish Court Lebanon, NH 03766. *Gambit 2.4 User's Guide*, 2007.

[5] S. M. Salim and S. C. Cheah. Wall y + Strategy for Dealing with Wall–bounded Turbulent Flows. In *Proceedings of the International MultiConference of Engineers and Computer Scientists 2009 Vol II*, IMECS, Hong Kong, March 2009. ISBN 978-988-17012-7-5.

[6] Fluent Incorporated, Centerra Resource Park 10 Cavendish Court Lebanon, NH 03766. *Fluent 6.3 User Guide*, 2007.

[7] Y. C. Fung. *Biodynamics : Circulation*. Springer, Berlin, 1984. ISBN 3-540-90867-6 ; 0-387-90867-6.

[8] K. B. Chandran, A. P. Yoganathan, and S. E. Rittgers. *Biofluid Mechanics : The Human Circulation*. Taylor & Francis Group, Boca Raton, Fl, 2006. ISBN 978-0-8493-7328-2.

[9] L. Formaggia. *Cardiovascular Mathematics : Modeling and simulation of the circulatory system*. MS&A ; 1SpringerLink : Bücher. Springer-Verlag Milan, Milano, 2009. ISBN 978-88-470-1152-6. In: Springer-Online.

[10] M. M. Molla and M. C. Paul. LES of non–Newtonian physiological blood flow in a model of arterial stenosis, 2010. Submitted for publication in Medical Engineering & Physics.