CPM Specifications Document
Healthy Aortofemoral – Lower Extremity:

OSMSC 0004_0001, 0159_0001

May 1, 2013
Version 1

Open Source Medical Software Corporation

1. Clinical Significance & Condition

Studying the hemodynamics of the vasculature distal to the abdominal aorta may be important in understanding common diseases in peripheral arteries downstream of the thoracic aorta. Diseases in the peripheral vasculature affect millions of people in the U.S and can have a profound effect on daily quality of life.

Peripheral arterial disease is the build-up of fatty tissue, or atherosclerosis, in lower extremity arteries. By 2001 at least 10 million people in the U.S were estimated to have peripheral arterial disease. The prevalence of peripheral arterial occlusive disease increases with age and can increase to up to 20% of the population in the geriatric population [1]. Up to 4 million people in the U.S suffer from intermittent claudication causing pain in the legs during exercise. Atherosclerotic occlusive disease of the lower extremity arteries is a major cause of walking impairment, pain, ulcerations and gangrene.

Renal artery stenosis can have a prevalence of up to 45% in selective populations, specifically populations with other vascular disease. Prevalence can be from 1-6% in hypertensive patients to 30-45% in patients with aortoiliac occlusive disease or abdominal aortic aneurysms [1]. It is most often caused by atherosclerosis in the renal arteries and is often undetected until symptoms become severe. The most common symptom of renal artery stenosis is hypertension, which can have significant effects on the entire vasculartur. Up to 24% of patients with renal insuffiency, which can lead to end-stage renal disease renal disease, had renal artery stenosis, suggesting that renal artery stenosis may play an important role in kidney failure [1].

2. Clinical Data

Patient-specific volumetric image data was obtained to create physiological models and blood flow simulations. Details of the imaging data used can be seen in Table 1. See Appendix 1 for details on image data orientation.

Table 1 – Patient-specific volumetric image data details (mm)

OSMSC ID	Modality	Voxel Spacing			Voxel Dimensions			Physical Dimensions		
		R	A	S	R	A	S	R	A	S
0004_0001	CT	0.5859	0.5859	0.9000	512	512	1448	300	300	1303.2
0159_0001	CT	0.7285	0.7285	2.5000	512	512	539	373	373	1347.5

Available patient-specific clinical data collected can be seen in Table 2.

Table 2 – Available patient-specific clinical data

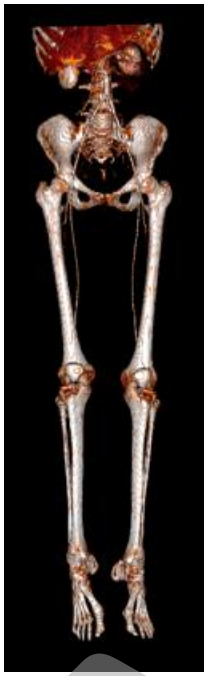
OSMSC ID	Age	Gender	Height (m)	Weight (kg)	BSA (m2)	Psys	Pdia
0004_0001	23	F	-	-	-	-	-
0159_0001	40	F	1.524	55.33	1.84	120	80

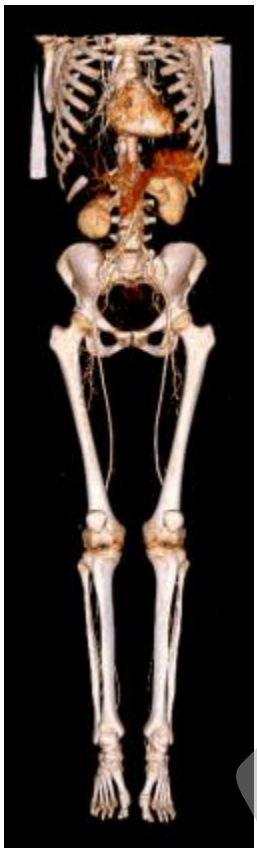
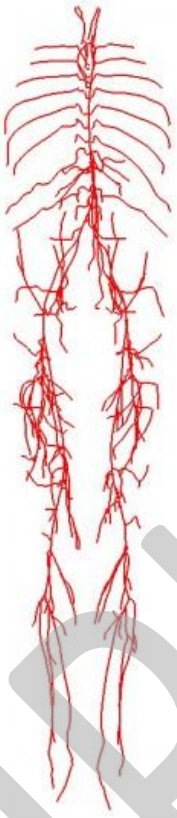
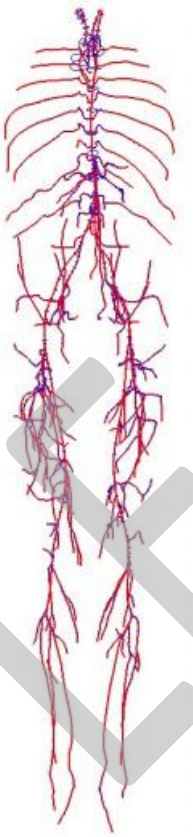
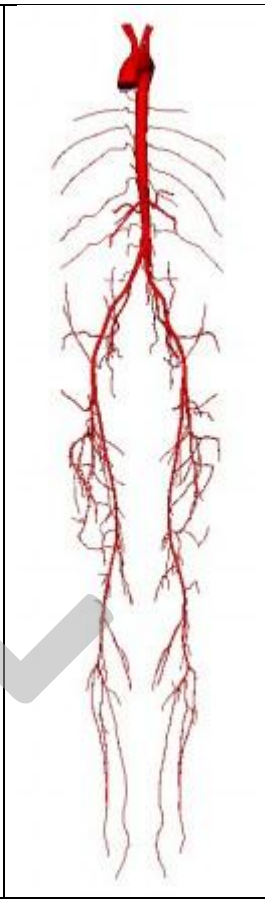
3. Anatomic Model Description

Anatomic models were create using customized SimVascular software (Simtk.org) and the image data described in Section 2. The models extend from the ascending and descending aorta all the way to the talocrural region, with various levels of branching inbetween. See Appendix 2 for a description of modeling methods. See **Error!**

Reference source not found. for a visual summary of the image data, paths, segmentations and solid model constructed.

Table 3 – Visual summary of image data, paths, segmentations and solid model.

OSMSC ID	Image Data	Paths	Paths and Segmentations	Model
ID: OSMSC0004 subID: 0001 Age: 23 Gender: F				

ID: OSMSC0159 subID: 0001 Age: 40 Gender: F				

Details of anatomic models, such has number of outlets and model volume, can be seen in Table 4.

Table 4 – Anatomic Model details

OSMSC ID	Inlets	Outlets	Volume (cm^3)	Surface Area (cm^2)	Vessel Paths	2-D Segmentations
0004_0001	1	20	59.34647	522.0463	20	590
0159_0001	1	149	151.9397	1104.816	149	3137

4. Physiological Model Description

In addition to the clinical data gathered for this model, several physiological assumptions were made in preparation for running the simulation. See Appendix 3 for details.

5. Simulation Parameters & Details

No simulation results available.

6. Simulation Results

No simulation results available.

7. References

[1] W. R. Hiatt, A. T. Hirsch and J. Regensteiner, Peripheral Artery Disease Handbook, Boca Raton, FL: CRC Press LLC, 2001.

Appendix

1. Image Data Orientation

The RAS coordinate system was assumed for the image data orientation. Voxel Spacing, voxel dimensions, and physical dimensions are provided in the Right-Left (R), Anterior-Posterior (A), and Superior-Inferior (S) direction in all specification documents unless otherwise specified.

2. Model Construction

All anatomic models were constructed in RAS Space. The models are generated by selecting centerline paths along the vessels, creating 2D segmentations along each of these paths, and then lofting the segmentations together to create a solid model. A separate solid model was created for each vessel and Boolean addition was used to generate a single model representing the complete anatomic model. The vessel junctions were then blended to create a smoothed model.

3. Physiological Assumptions

Newtonian fluid behavior is assumed with standard physiological properties. Blood viscosity and density are given below in units used to input directly into the solver.

Blood Viscosity: 0.04 g/cm•s^2
Blood Density: 1.06 g/cm^3

4. Simulation Parameters

Conservation of mass and Navier-Stokes equations were solved using 3D finite element methods assuming rigid and non-slip walls. All simulations were ran in cgs units and ran for several cardiac cycles to allow the flow rate and pressure fields to stabilize.

5. Outlet Boundary Conditions

5.1 Resistance Methods

Resistances values can be applied to the outlets to direct flow and pressure gradients. Total resistance for the model is calculated using relationships of the flow and pressure of the model. Total resistance is than distributed amongst the outlets using an inverse relationship of outlet area and the assumption that the outlets act in parallel.

5.2 Windkessel Model

In order to represent the effects of vessels distal to the CFD model, a three-element Windkessel model can be applied at each outlet. This model consists of proximal resistance (R_p), capacitance (C), and distal resistance (R_d) representing the resistance of the proximal vessels, the capacitance of the proximal vessels, and the resistance of the distal vessels downstream of each outlet, respectively (Figure 1).

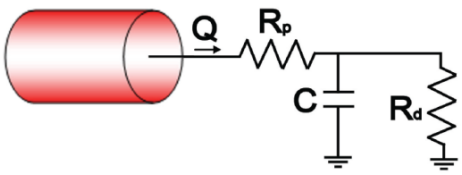


Figure 1 - Windkessel model

First, total arterial capacitance (TAC) was calculated using inflow and blood pressure. The TAC was then distributed among the outlets based on the blood flow distributions. Next, total resistance (R_t) was calculated for each outlet using mean blood pressure and PC-MRI or calculated target flow ($R_t=P_{mean}/Q_{desired}$). Given that $R_t=R_p+R_d$, total resistance was distributed between R_p and R_d adjusting the R_p to R_t ratio for each outlet.