CPM Specifications Document
Valvar Pulmonic Stenosis:

OSMSC 0136_0000

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Version 1

Open Source Medical Software Corporation

1. Clinical Significance & Condition

Pulmonary arteries connect blood flow from the heart to the lungs in order to oxygenate blood before being pumped through the body. The main pulmonary artery (MPA) starts at the right ventricle of the heart and divides into the left (LPA) and right pulmonary arteries (RPA), which branch out into the lungs. The main pulmonary artery in healthy subjects has an average diameter of 2.72 cm. Anatomical differences in MPA diameter have also been documented between genders, with a mean MPA diameter of 2.77 cm for males and 2.64 cm for females [1].

Examples of complications seen in the pulmonary arteries include pulmonary hypertension and pulmonary embolisms. Pulmonary arterial hypertension (PAH) is a chronic disease that occurs when the blood vessels between the heart and lungs narrow and harden, increasing the pressure in the pulmonary arteries [2]. The increased resistance makes it difficult for the heart to pump blood to the lungs, adding strain to and weakening the right ventricle. PAH is a serious condition, with a median survival of less than 3 years if left untreated [3],causing over 15,000 deaths and 260,000 hospital visits in the United States in 2002 [4].

Significant vascular remodeling is observed in PAH patients, with larger proximal pulmonary arteries and more convoluted branches when compared to healthy patients (Figure 1). In a study examining three-dimensional hemodynamics of the pulmonary arteries, PAH patients were found to have an average main pulmonary diameter of 3.5 ± 0.5 cm, where healthy patients had an average of 2.7 ± 0.1 cm [3].

A pulmonary embolism is another condition seen the pulmonary arteries, involving one or more arteries being blocked by a blood clot. The blood clots typically originate elsewhere in the body and travel to the pulmonary arteries. The effects of a pulmonary embolism can be quiet severe, with the first sign being sudden death in 25% of pulmonary embolism cases [5]. However, prompt application of anti-clogging medication can help avoid mortality and further complications [6].

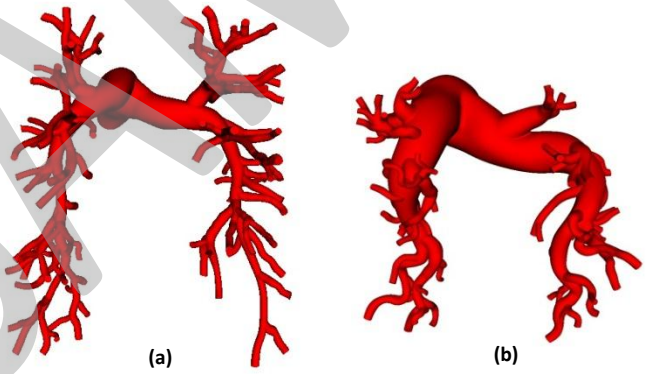


Figure 1 (a) Healthy pulmonary arteries, (b) PAH pulmonary arteries

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
0136_0000	MR	1.1000	0.5469	0.5469	112	512	512	123.20	280.01	280.01

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

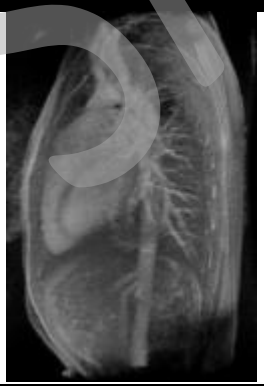
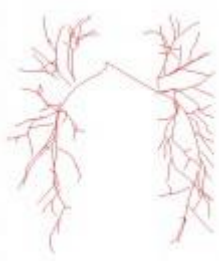
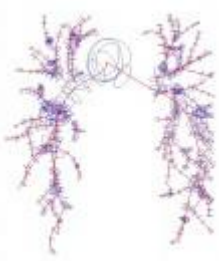
Table 2 – Available patient-specific clinical data

OSMSC ID	Age	Gender	Height (m)	Weight (kg)	BSA (m^2)	CO (L/min)	CI (L/min/m^2)	HR (bpm)	Psys (mmHg)	Pdia (mmHg)
0136_0000	10	M	1.38	30	1.1	4.39	4.03	77	95	71

3. Anatomic Model Description

Anatomic models were created using customized SimVascular software (Simtk.org) and the image data described in Section 2. The models extend from the main pulmonary artery to various levels of branching in the left and right pulmonary arteries. 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: OSMSC0136 subID: 0000 Age: 10 Gender: M				

Details of anatomic models, such has number of outlets and model volume, can be seen in Table 4 – Anatomic Model details

OSMSC ID	Inlets	Outlets	Volume (cm^3)	Surface Area (cm^2)	Vessel Paths	2-D Segmentations

0136_0000	1	85	61.614609	311.590676	94	530

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Table 4 – Anatomic Model details

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0136_0000	1	85	61.614609	311.590676	94	530

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] P. D. Edwards, R. K. Bull and R. Coulden, "CT measurement of main pulmonary artery diameter," *The British Institue of Radiology,* vol. 71, pp. 1018-1020, 1998.

[2] U.S. National Library of Medicine, "Pulmonary Hypertension," National Institutes of Health, 4 January 2012. [Online]. Available: http://www.nlm.nih.gov/medlineplus/pulmonaryhypertension.html#cat22. [Accessed January 2012].

[3] B. Tang, *Quantification of Three-Dimensionsal Hemodynamic Conditions in the Human Abdominal Aorta and Pulmonary Arteries with Application to Shear-Mediated Gene Transcription in Endothelial Cell Culture,* PhD Dissertation, Department of Mechanical Engineering, Stanford University, Stanford, CA, USA, June 2007.

[4] A. Hyduk, J. B. Croft, C. Ayala, K. Zheng, Z.-J. Zheng and G. A. Mensah, "Pulmonary Hypertension Surveillance: United States, 1980-2002," *Morbidity and Mortality Weekly Report ,* vol. 54, no. SS05, pp. 1-28, 2005.

[5] Ceneters for Disease Control and Prevention, "Deep Vein Thrombosis/Pulmonary Emoblism: Data & Statistics," 21 September 2011. [Online]. Available: http://www.cdc.gov/ncbddd/dvt/data.html. [Accessed 24 January 2012].

[6] Mayo Clinic, "Pulmonary Embolism," 27 September 2011. [Online]. Available: http://www.mayoclinic.com/health/pulmonary-embolism/DS00429. [Accessed 19 January 2012].

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).

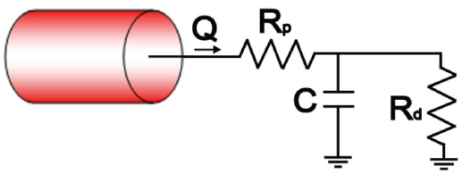

Figure 2 - Windkessel model

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.