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1D Modelling of Blood Flow in Human Vascular Network

Soroush Safaei[a,c], **Chris Bradley**[a] **and Vinod Suresh**[a,b]

[a] *Auckland Bioengineering Institute (ABI),* [b] *Department of Engineering Science, University of Auckland, New Zealand,* [c] *soroush.safaei@auckland.ac.nz*

OBJECTIVES

The goal of this project is to develop an anatomically based, one-dimensional model of circulatory bed to:

- Provide boundary conditions for 3D models of blood flow by defining upstream and downstream flow and pressure.
- Develop pharmacokinetic models of drug transport and metabolism

STRUCTURE

Arteries are assumed to be straight long tubes with elastic walls:

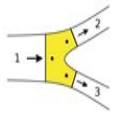
$$P - P_{ext} = \beta\left(\sqrt{A} - \sqrt{A_0}\right)$$

By integrating the continuity and longitudinal momentum equations of the Navier-Stokes equations over the cross sectional area, we obtain:

$$\frac{\partial Q}{\partial t} + \frac{\partial(\alpha Q^2/A)}{\partial x} + \frac{A}{\rho}\frac{\partial P}{\partial x} + 8\pi\nu\frac{Q}{A} = 0 \qquad \frac{\partial A}{\partial t} + \frac{\partial Q}{\partial x} = 0$$

Arterial bifurcations are modelled by imposing conservation of mass & momentum in combination with calculation of the Riemann invariants of the system:

$$\frac{Q_1}{A_1} + 4A_1^{1/4}\sqrt{\frac{\beta_1}{2\rho}} = W_1^1 \qquad \frac{Q_2}{A_2} - 4A_2^{1/4}\sqrt{\frac{\beta_2}{2\rho}} = W_2^2 \qquad \frac{Q_3}{A_3} - 4A_3^{1/4}\sqrt{\frac{\beta_3}{2\rho}} = W_3^3$$



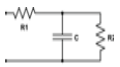
$$p_1 + \frac{1}{2}\rho\left(\frac{Q_1}{A_1}\right)^2 = p_2 + \frac{1}{2}\rho\left(\frac{Q_2}{A_2}\right)^2 \qquad p_1 + \frac{1}{2}\rho\left(\frac{Q_1}{A_1}\right)^2 = p_3 + \frac{1}{2}\rho\left(\frac{Q_3}{A_3}\right)^2 \qquad Q_1 = Q_2 + Q_3$$

Navier-Stokes Equation — 1D Model — Characteristics System

Peripheral Model — 0D Model — Heart Model

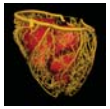
A terminal impedance representing the distal network of each truncated artery is prescribed. This terminal impedance is modelled using a three element windkessel model. The pressure and flow at the truncated arteries are related according to:

$$P + R_2C\frac{\partial P}{\partial t} = P_v + (R_1 + R_2)Q + R_1R_2C\frac{\partial Q}{\partial t}$$

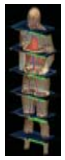


The arterial tree is coupled to the left ventricle of the heart. The left ventricular model is based on the time-varying elastance $E(t)$ which describes the variation of left ventricular pressure p_{lv} and volume V_{lv} during a cardiac cycle:

$$E(t) = \frac{p_{lv}(t)}{V_{lv}(t) - V_0}$$



IMPLEMENTATION



Anatomical data from the Visible Human Project are used to create the geometry for the circulatory model.
(www.nlm.nih.gov/research/visible)

The FEM is implemented in OpenCMISS which is an object based, modular, open source, library of routines written in Fortran 95/2003 designed to solve complex bioengineering.
(www.OpenCMISS.org)

The 0D models are implemented in CellML which is an XML based language designed to encode lumped parameter models represented by systems of ordinary differential equations and nonlinear algebraic equations. (www.CellML.org)

ACKNOWLEDGEMENTS

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REFERENCES

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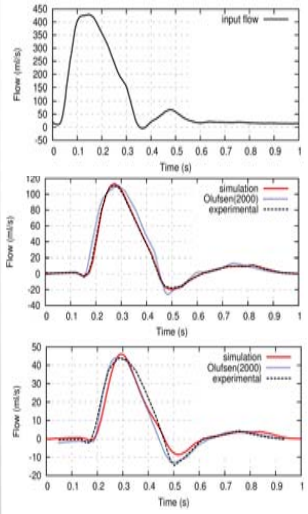
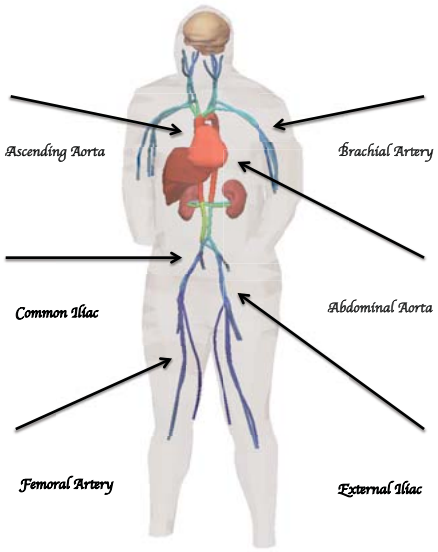
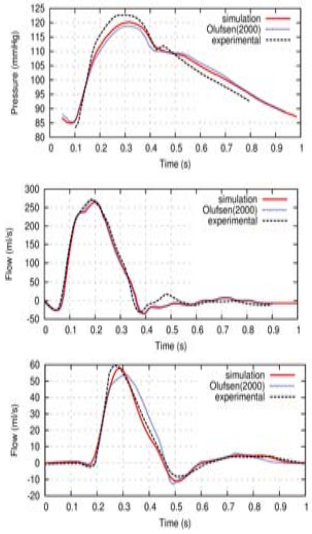
[2] Abernethy, D. R. (1989). The pharmacokinetic profile of amlodipine. *American heart journal, 118*(5), 1100-1103.

RESULTS



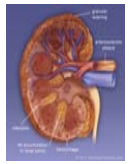


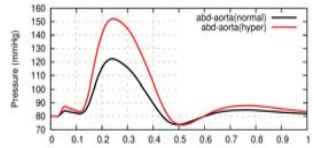


APPLICATIONS

Modelling Renovascular Hypertension

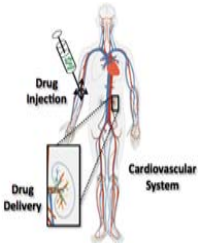
In renovascular hypertension high blood pressure is caused by the kidneys' hormonal response to narrowing (stenosis) of the renal arteries. When functioning properly this hormonal axis regulates blood pressure. Due to low local blood flow, the kidneys increase blood pressure of the entire circulatory system for compensation.

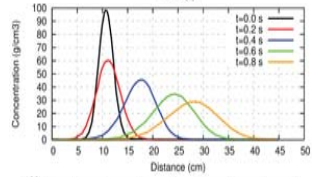




Pharmacokinetic Modelling (PBPK)

1D models of the circulatory system will be useful in physiologically based pharmacokinetic modelling (PBPK). By coupling the flow with advection-diffusion equations, we can develop PBPK models to determine drug distribution in particular vascular beds. A system of differential equations for drug concentration can be written which depends on blood flows, pulmonary ventilation rate, organ volumes, metabolism etc. Model can be used to determine optimum dosage and regional clearance on the basis of physiological factors.





Amlodipine (Calcium Channel Blocker)

As a case study, we examined the effect of distribution and excretion of amlodipine used to treat renovascular hypertension. Metabolism and excretion of amlodipine are simulated using PBPK model for the kidneys. Figure on top right shows drug concentration during 48 hours. Amlodipine lowers blood pressure by relaxing arterial smooth muscles. We decreased the wall elasticity of the lower arteries by 25% to model the amlodipine effect. These effects gradually decreased as the drug was excreted through the kidneys. Figure on down right shows effect of amlodipine on blood pressure in the arterial system.