Modelling of the mechanobiological adaptation to vascular occlusion in the arterial tree

Bachelor’s Thesis

Author: Jaime Rodríguez María

Supervisor: Stefan Lindström
AGENDA

1. INTRODUCTION
2. METHOD
3. DISEASES
4. CONCLUSIONS
1. Introduction
Why modelling cardiovascular diseases?

- 2012 - 17.5 millions of people, 31% of the global deaths
- Prediction of their development → G&R

Polycythemia  Thrombosis
Atherosclerosis  Aneurysms  Stroke
Anemia  Arrhythmia
Coronary heart disease  Cerebral edema
Calcification  Abnormal aging
Hemorrhage  Dementia  Cerebral ischemia
Arteriosclerosis
2. Method
Simplifications (1/2)

\[ A^k(t) = A^k(0)Q^k(t) + \int_0^t a^k(\tau)q^k(t - \tau)d\tau \]
Simplifications (2/2)

Middle cerebral artery

Hagen-Poiseuille’s law: \( u = \frac{\Delta p \pi r_i^4}{8L\mu} \)

Kirchhoff’s law: \( \nabla \bar{u} = 0 \)

Murray’s law: \( \dot{r}_i^3 = Ku \)
LIMITATIONS

• Aorta – curvature & turbulent flow
• Arterial wall – uniformity in composition
• Arterial tree – 2D, symmetric & w/o crossing branches
• Internal chemical processes
• Certain diseases (aneurysms)
• Experimental data
Boundary conditions & set up (1/4)

- Local cerebral blood flow (MCA) = 20 ml/min
- Pressure drops − 0 Pa at the first node
- Wall shear stress − \( \tau_W = \frac{4f \mu_0 u}{\pi r_i^3} \); \( \hat{\tau}_W = \frac{4\mu_0}{\pi K} \)
- Vascular occlusion − \( u_{factor}(t) = \frac{1}{\pi} \left( \frac{\pi}{2} - \tan \left( \frac{t-t_d}{\tau} \right) \right) \)

\[
\begin{align*}
    u_{out,1}(t) &= u_{out,1}(0)(R + (1 - R)u_{factor}(t)) \\
    u_{out,2}(t) &= u_{out,2}(0)(R + (1 - R)u_{factor}(t)) \\
    u_{out,3}(t) &= u_{out,1}(0) + u_{out,3}(0) - u_{out,1}(t) \\
    u_{out,4}(t) &= u_{out,2}(0) + u_{out,4}(0) - u_{out,2}(t)
\end{align*}
\]
Boundary conditions & set up (2/4)

- Double bifurcation
Boundary conditions & set up (3/4)

- Same length for each segment ($L = 3$ mm)
Boundary conditions & set up (4/4)

• Control of the simulation through 4 parameters:
  a) \( t_d \) – when the sudden change occurs
  b) \( \tau \) – how fast the transition takes places
  c) \( T \) – duration of the simulation
  d) \( R \) – percentage of blood flow left
Final model
3. DISEASES
Thrombosis

- Continued lines – Vascular occlusion ($\mathcal{R} = 0.3$)
- Dashed lines – Vascular occlusion ($\mathcal{R} = 0.6$)
Calcification

- Continued lines – Vascular occlusion ($\mathcal{R} = 0.5$)
- Dashed lines – Vascular occlusion ($\mathcal{R} = 0.5$) & calcification (stiffer collagen)
Cerebral Ischemia and Edema (1/4)

- Cerebral edema (hypertension)
CEREBRAL ISCHEMIA AND EDEMA (2/4)

- Cerebral ischemia (hypotension)
CEREBRAL ISCHEMIA AND EDEMA (3/4)

- Cerebral edema \( (p_0 = 130 \text{ mmHg}) \) & vascular occlusion \( (\mathcal{R} = 0.3) \)
Cerebral ischemia and edema (4/4)

- Cerebral isquemia ($p_0 = 50$ mmHg) & vascular occlusion ($\mathcal{R} = 0.3$)
Anemia & Polycythemia (1/3)

- Blue line – Polycythemia (hypercoagulability)
- Green line – Anemia (hypocoagulability)
Anemia & Polycythemia (2/3)

- Polycythemia (hypercoagulability) & vascular occlusion ($\mathcal{R} = 0.3$)
• Anemia (hypocoagulability) & vascular occlusion ($R = 0.3$)
4. Conclusions
CONCLUSIONS

• In thrombosis, thickness & lumen radius change at the same time

• Calcification increases wall stresses during G&R

• Diseases related to pressure
  — Thickness changes faster than lumen radius
  — Ischemia distinguished from edema by looking at thickness

• Diseases related to viscosity
  — Both thickness & lumen radius change at the beginning of G&R
  — Polycythemia more difficult to detect than anemia
  — Huge risk if medication
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