Modelling of Blood Flow in a Vascular System

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Introduction

- **Stroke** is the 3rd largest cause of death in Australia
  - 1/3 of stroke patients (around 16,000 people) will die in first 12 months
- **Stroke** is an acute neurological injury caused by a sudden **loss of blood** supply to the brain
- **Hemorrhagic Stroke** and **Ischemic Stroke**
How does it occur?

Hemorrhagic Stroke

- Aneurysm

Ischemic Stroke

- Thrombus

- Embolus

- Atherosclerosis

80%
Hypotheses

- Research works have been done
- Causative factors *(plaque formation)*:
  - Low shear stress
  - Reversed flow shear
  - Local wall static pressure
- Causative factors *(plaque growth)*:
  - High shear stress
Carotid Bifurcation
Problem

- Surgery can be done for removing the plaque
- Factors need to consider:
  - Degree of narrowing
  - Growth of the plaque
  - Possibility of fragmentation of the plaque
- Require Knowledge of hemodynamics parameters
- Difficult to measure and Unknown for Medical Doctor
Objectives

- Provide **detailed Hemodynamics** data of individual patient for Medical Doctor

- Attempt to **correlate** the hemodynamics parameter to the **plaque formation/growth**

- Enable better Hemodynamics **Analysis** for **Stroke diagnostics**
Generation of Carotid Bifurcation Model (based on in vivo MRI angiograms)
Carotid bifurcation model

Original Stenosed Model  Modified Healthy Model

CCA  ECA  ICA

CCA  ECA  ICA

Original Stenosed Model  Modified Healthy Model
Model Assumptions

- Blood – incompressible, Newtonian fluid
  - Density – 1176 kg/m³
  - Viscosity – 0.004 Pa s

- Artery Wall – Rigid Boundary
  - Non-elastic and impermeable

- $k$-$\varepsilon$ turbulence model
Computational Model

89,501 number of element  59,502 number of element
Boundary Conditions

- Pulsatile Pressure Waveform at the inlet
- Flow Waveform at three outlets

Flow Rate
Pressure
Waveform input as boundary conditions
Results

- **Transient simulation** was performed for 6 complete cardiac cycle

- Physical Time Step: 1/100 of cardiac cycle (0.092 seconds)

- **Convergence criterion** based on RMS residual was set as $1.0 \times 10^{-4}$
Comparison of Wall Shear Stress distribution
WSS in Stenosed Model
Time-dependent WSS fluctuation at the narrowest point
Observations

- Maximum Wall Shear Stress: 90.5 pa
- Fluctuate periodically from 10.0 to 90.5 pa
- High level of wall shear stress constantly “peeling” off the plaque which increase the risk of having embolytic stroke
WSS in Healthy Model

Posterior View

Anterior View
Observations

- Low wall shear stress was found at the outer wall region.

- Distribution of the WSS is in accordance with the location of the plaque.

- Low WSS values are possibly correlated to localization of atherosclerotic lesions.
Streamline of Blood Flow

Maximum Velocity: 4.66 m/s

Maximum Velocity: 1.27 m/s
Observations

- Maximum velocity is almost 4 times higher than the healthy model
- Blood flow inside the stenosed model appears more chaotic
- Recirculation flow was found after the stenosis (sudden expansion)
- Longer residence time may promote platelet accumulation (plaque growth)
Blood Flow Structure in Stenosed Model
Observations

- Highly non-uniform blood flow was formed due to the sudden congestion and expansion.

- Slow moving/recirculation region were found after the plaque.

- Highly shewed and non-uniformed flow structure results a non-uniformed WSS distribution.
Blood Flow Structure in Healthy Model
Observations

- Flow is highly skewed towards the flow divider walls
- Slow flow zones were found at the outer wall of ECA and ICA opposite to the divider
- During deceleration phase, flow was more disturbed and the slow flow zones were larger
Conclusion

- Anatomically realistic Carotid Bifurcation Model have been successfully generated and incorporated with CFD simulation
- Blood flow in the stenosed and healthy model were simulated and compared
- Distribution of the WSS and location of slow flow regions in the healthy model is in accordance with the location of the plaque
- Flow in the healthy model is highly skewed towards the flow divider walls
Further Works

- More in vivo models will be investigated
Further Works (continue)

- Elasticity of artery wall will be considered
  - Wall dispensability may effect WSS distribution

- Different blood flow conditions
  - Different flow rate, blood pressure
The End