MODELLING STENOSIS DEVELOPMENT IN THE CAROTID ARTERY AT THE EARLY STAGES OF STENOSIS DEVELOPMENT

A.C.Stamou[1], J.M.Buick[2]

[1] Faculty of Engineering and Computing, Coventry University

[2] School of Engineering, Anglesea Building, Portsmouth

Objectives of the talk

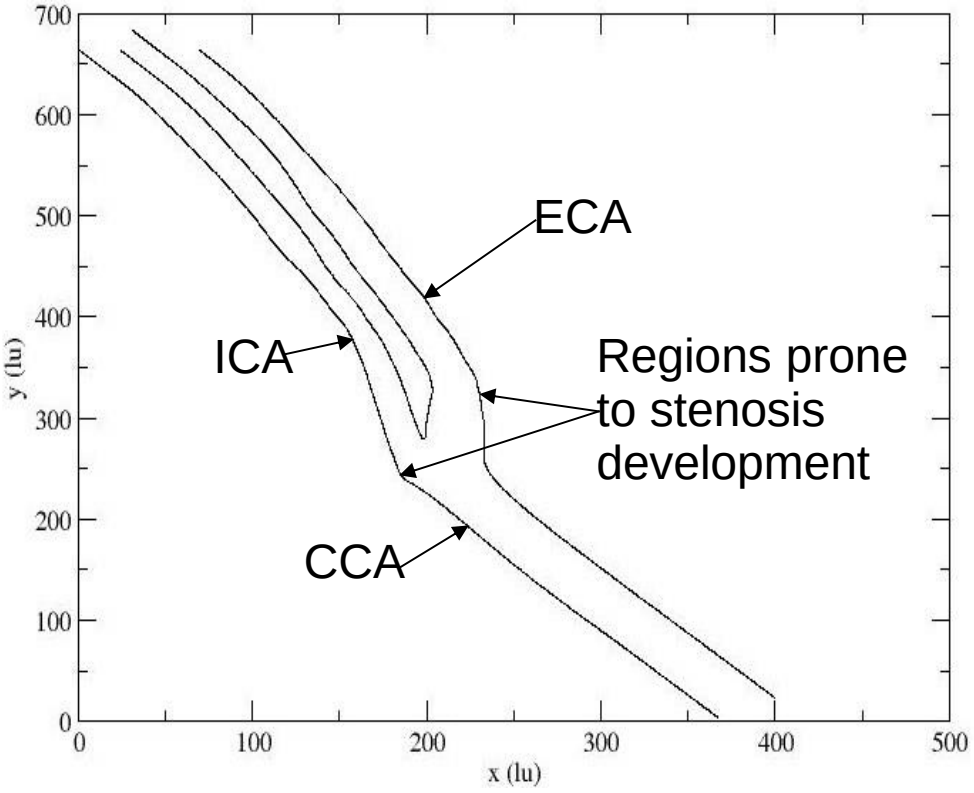
- Presentation outline
- Description of the problem
- Methodology
- Lattice Boltzmann method
- Stenosis growth model
- Results
- Conclusion

Presentation outline

- In the present work, the position of the development of a stenosis in the carotid artery has been simulated based on regions of **stagnation in blood flow**.
- The interest is focused on:
  - the blood flow properties, computed here using the Lattice Boltzmann Method,
  - prediction of the initial growth of the stenosis, based on regions of blood stagnation at the artery wall structure,
  - variation of the haemodynamic properties with the growth of the stenosis.

ATHEROSCLEROSIS AND SUSCEPTIBILITY TO ATHEROSCLEROSIS

- Atherosclerosis, one of the major causes of strokes. It is a disease which
  - is likely caused by the lipoprotein accumulation accompanied with endothelial cell damage [1,2] on the wall surface,
  - manifests on areas of high curvature and bifurcations.[1,2]
- These atherosclerotic-prone-sites are often characterized by:
  - low endothelial stress [3, 4](endothelial stress: the applied shear stress on the walls because of the fluid movement),
  - disturbed flow [2] (flow characterized by high oscillations of the shear stress from the predominant flow direction [5]),
  - stagnant flow. [6]



Carotid artery (ICA, ECA, CCA: External Carotid Artery, Internal Carotid Artery, Common Carotid Artery)

LATTICE BOLTZMANN METHOD

- **LBM equation-Kinematic behaviour**

- $f(\vec{x}, t)$ is the particle distribution function along the i^{th} direction
- Evolution equation

$$f_i(\vec{x} + \overrightarrow{e_i}, t + 1) = f_i(\vec{x}, t) + \Omega_i(\vec{x}, t)$$

where $\overrightarrow{e_i}$ is the vector along link i,

- the collision operator is

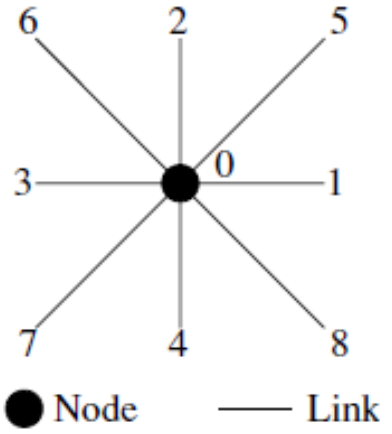
$$\Omega_i(\vec{x}, t) = -\frac{1}{\tau}(f_i(\vec{x}, t) - {f_i}^{eq}(\vec{x}, t))$$

and the equilibrium distribution function is

$${f_i}^{eq} = w_i \rho \left(1 + 3\overrightarrow{e_i} \cdot \vec{u} + \frac{9}{2}(\overrightarrow{e_i} \cdot \vec{u})^2 - \frac{3}{2}u^2\right)$$

where w_i $(0 < w_i < 1)$ is weight function and the density and the velocity are given by:

$\rho = \sum_{i=0}^{8} f_i$ and $\rho\vec{u} = \sum_{i=0}^{8} f_i \overrightarrow{e_i}$



The D2Q9 lattice

STENOSIS GROWTH MODELLING (CRITERIA)

- The velocity magnitude along the wall is considered of significant effect on the simulation of the growth of the stenosis.

- However, the maximum stagnation of the flow is considered in our work as the criterion for the growth of the stenosis, with stagnation index:

$$SI = N/T$$

- where T is the period of the cardiac pulse and N is the number of time-steps in a period when u_1, the local velocity magnitude at the 1 grid length from the wall, is less than 1% of the average u_1 over the whole of the wall during the previous period.

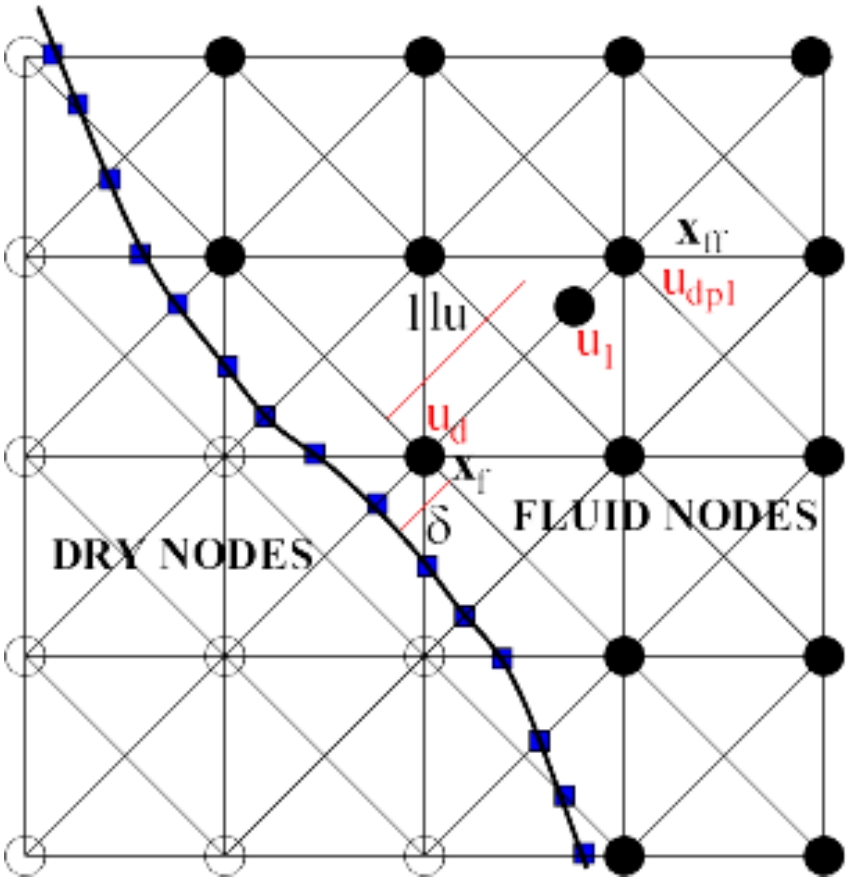
CALCULATION OF THE VELOCITY MAGNITUDE

- The simulation of the growth of the stenosis is accomplished assuming an extrapolation scheme that supports the movement-boundary mechanism.

- The velocity magnitude locally at the 1 grid space from the wall is worked out, by:

$$u_1 = (1-\delta)^* u_{dp_1} + \delta * u_d$$

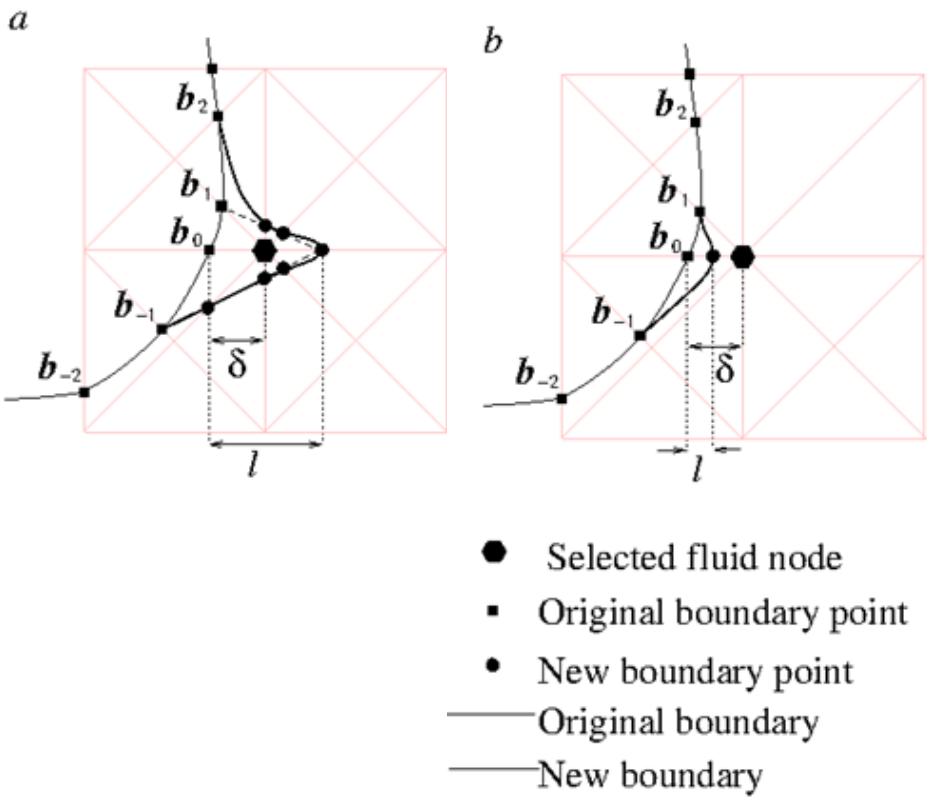
  - δ: distance of the first fluid node x_f
  - from the wall. u_{dp1}, u_d, u_1:velocities
  - at x_{ff}, x_f and one grid length (1 lu) from the wall.



Arterial grid

APPLYING THE MOVING BOUNDARY MECHANISM

- Picking out the point satisfying the mechanical criterion (stagnation)
- Applying the illustrated boundary-movement scheme at the first fluid node from the wall
- Comparing the δ and l (the distance the boundary moves, here set to 0.5)
  - $\delta \leqq l$: the selected fluid node x_f turns to wall (case a)
  - $\delta > l$: the selected fluid node x_f remains a fluid node (case b)
- Working out the new δ'
  - $\delta' = l - \delta$ (case a)
  - $\delta' = \delta - l$ (case b)
- Set up the new boundary as shown



Movement-boundary mechanism

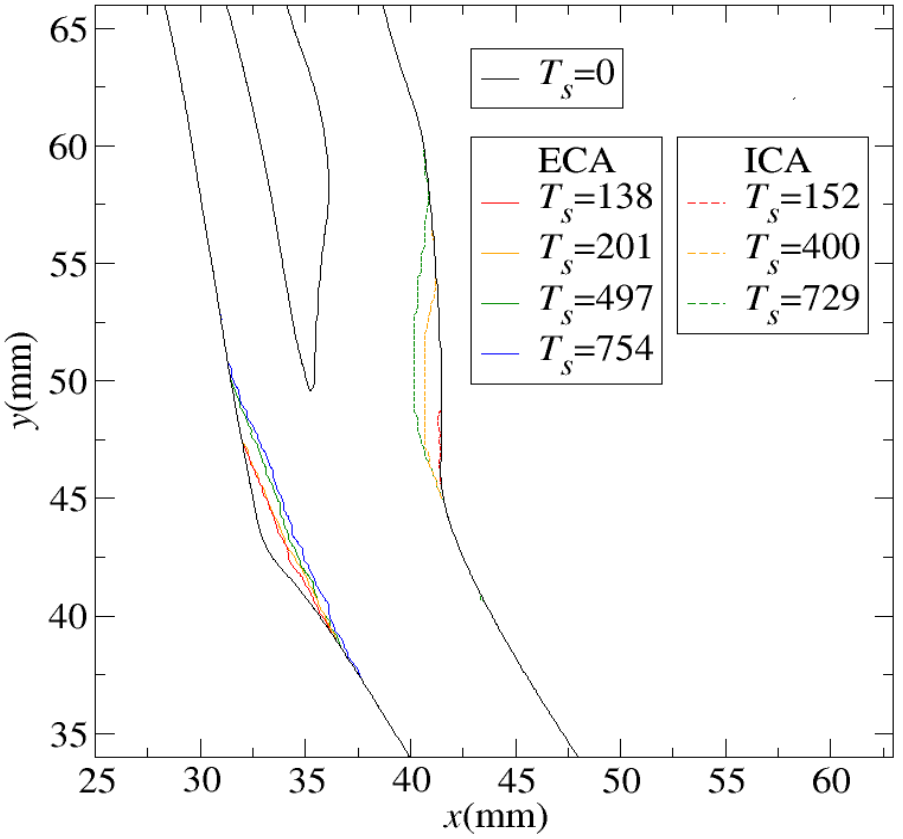
RESULTS ANALYSIS

The presentation of the results is structured as follows:

- Stenosis development in layers
- Effect of stenosis growth on the blood flow along the wall
- Flow profiles

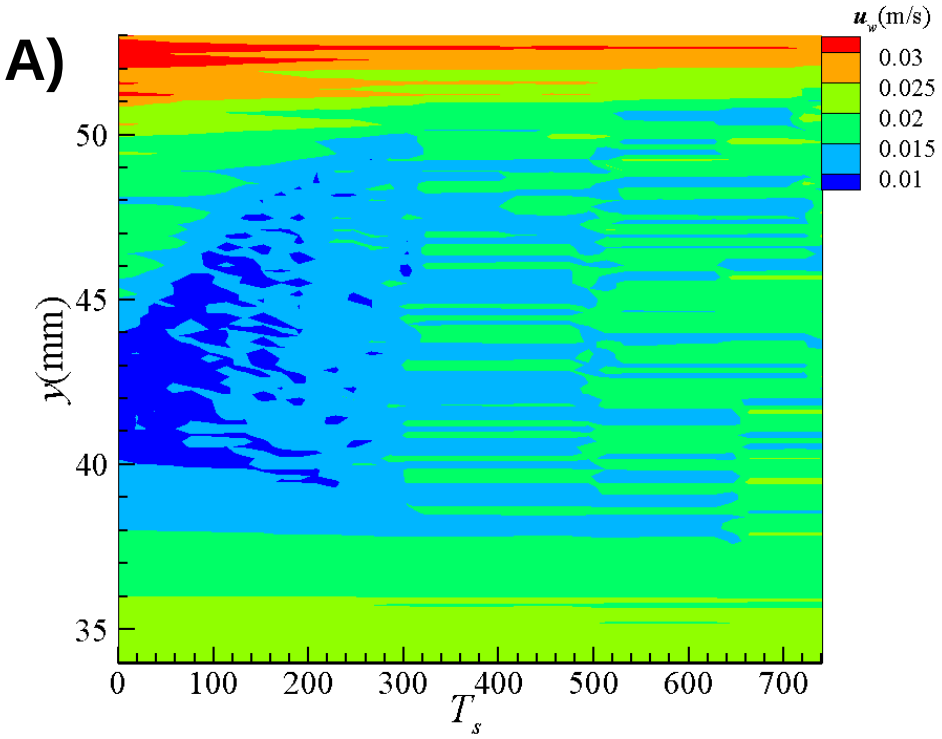
STENOSIS DEVELOPMENT

- The stenosis is formed in layers built up on the outer walls of the ECA and ICA.
- The stenosis layers are built up consecutively on either the same arterial wall or on the opposite wall.
- The growth occurs on the ECA below the bifurcation.
- In the ICA the stenosis occurs from slightly below the bifurcation and extends significantly in to the ICA.

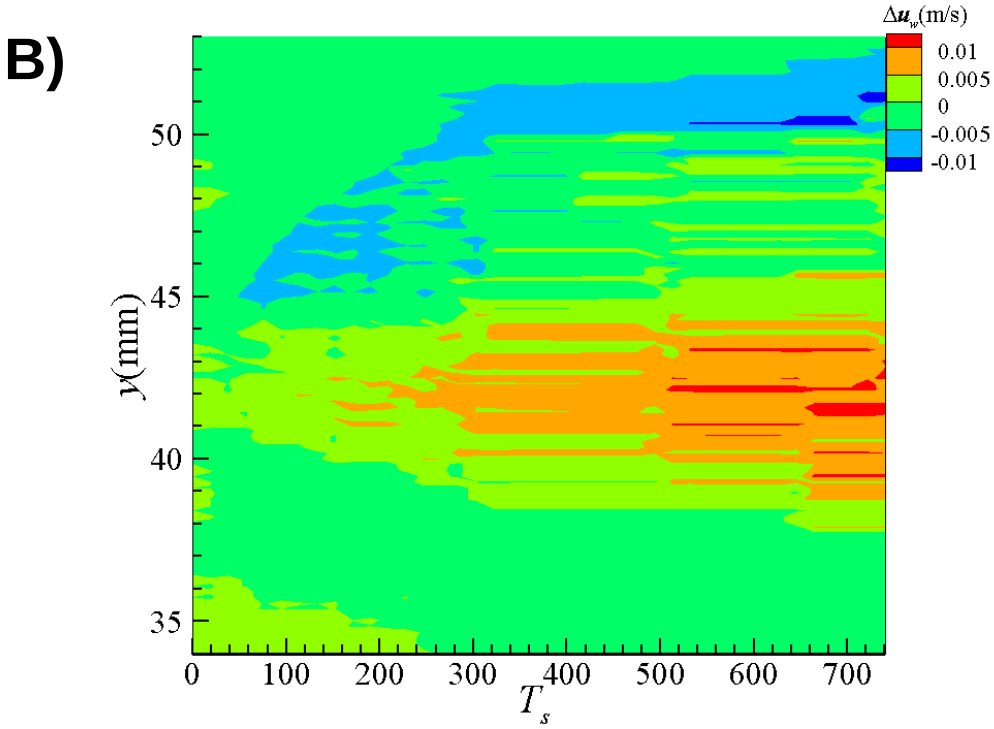


Stenosis growth in layers with T_s, the number of grid sites, originally in the blood flow region, which have been enclosed by the growth of the stenosis.

CHANGE IN THE WALL VELOCITY WITH STENOSIS GROWTH



A) Velocity magnitude along the ECA

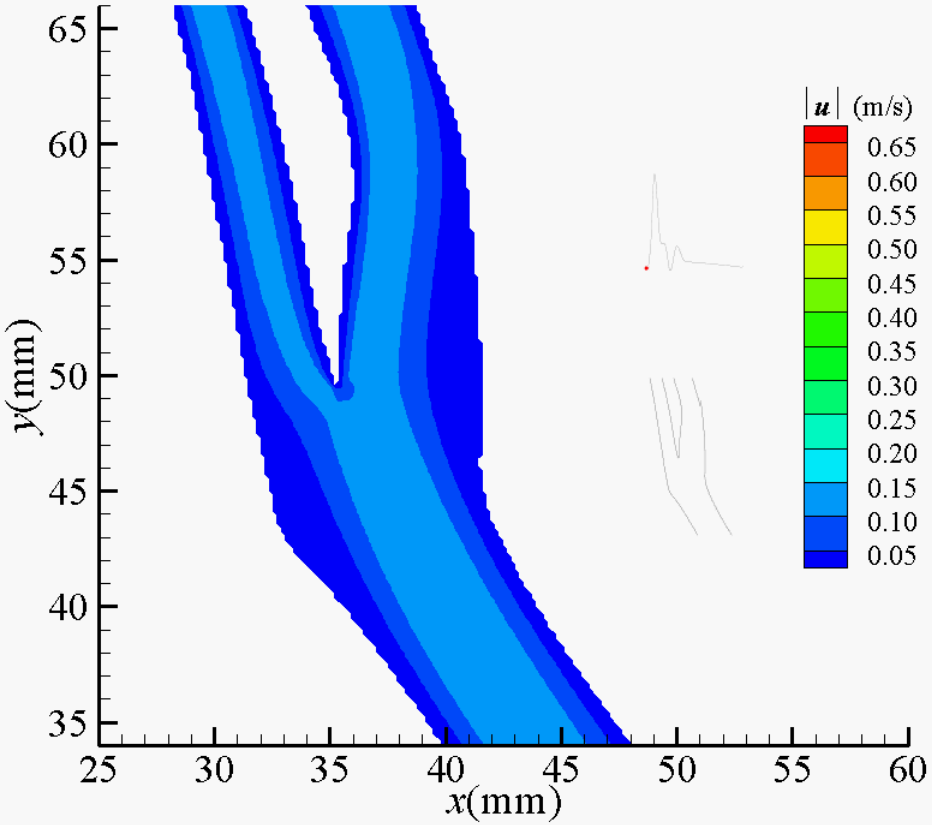


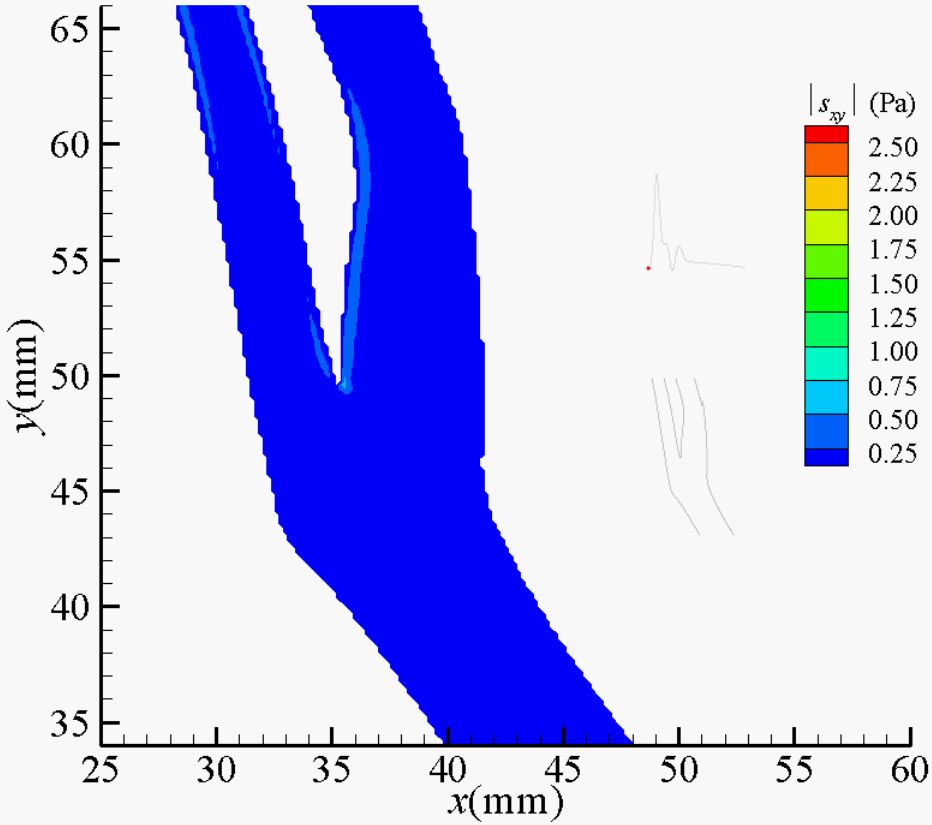
B) Velocity variation with the growth of the stenosis as a function of T_S

The growth of the stenosis influences mainly the flow at the stenosed regions.

FLOW PROFILES (UNSTENOSED ARTERY)

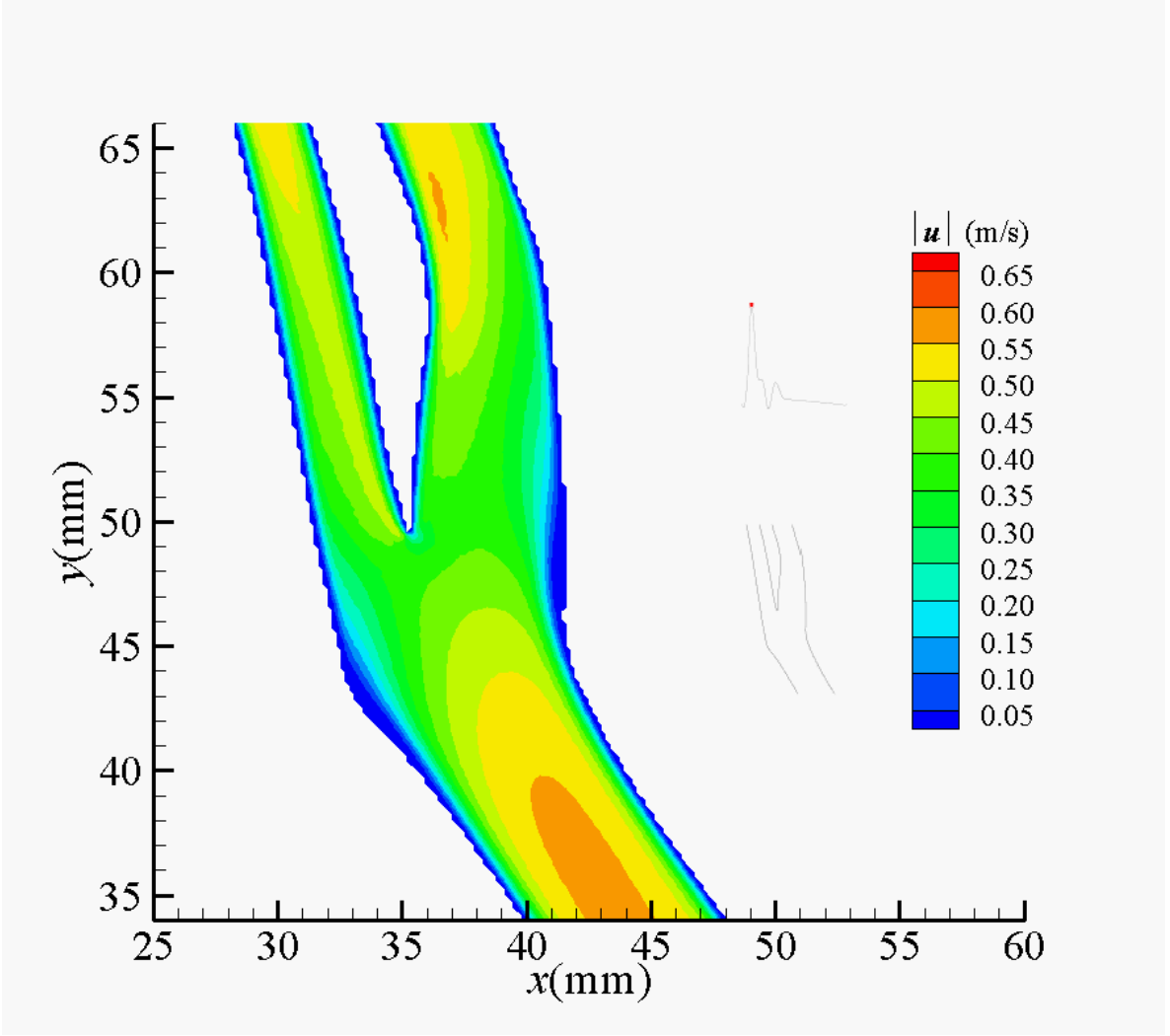
- Velocity Magnitude
- Shear Stress





Over ½ cardiac pulse

STENOSIS-GROWTH PROGRESSION



At the peak-pressure-time over the full cardiac pulse

FLOW PROFILES (FULLY STENOSED ARTERY)

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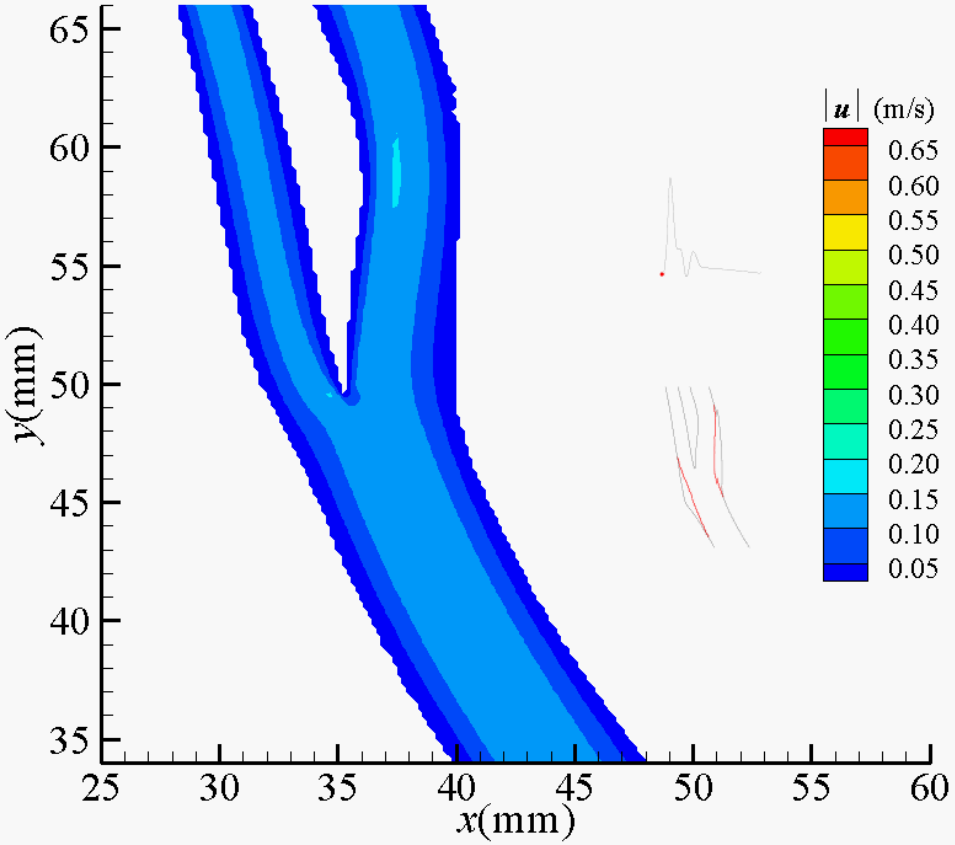
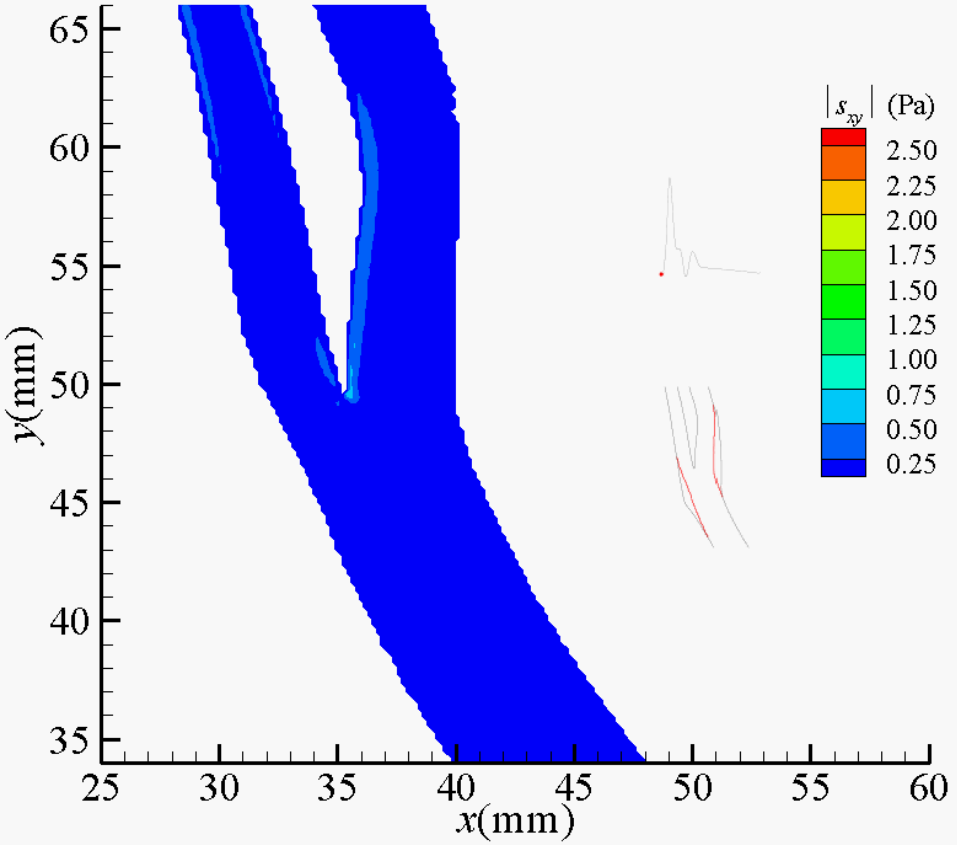
- Velocity Magnitude
- Shear Stress





Over ½ cardiac pulse

Conclusions

- The position of the stenosis has been simulated here based on regions of flow stagnation.
- The stenosis is formed on the outer wall of both the ICA and the ECA in the region where vortex motion develops in the healthy artery.
- The growth of the stenosis influences mainly the flow close to the stenosed wall.
- An increased velocity was observed in both the ICA and the ECA with the growth of the stenosis.

Future research

- Simulation of the progression of the stenosis in a number of other arteries which are similarly susceptible to this disease, in particular the human aorta
- Comparison of haemodynamics between stenosed and stented arteries
- Simulation of the non-Newtonian blood considering alternative models to the Carreau-Yasuda
- Development of a 3D model to enable the secondary flows in the artery to be assessed and their effect on stenosis development

References

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