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Application of the lattice Boltzmann model to simulated stenosis growth in a two-dimensional carotid artery

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Abstract

The lattice Boltzmann model is used to observe changes in the velocity flow and shear stress in a carotid artery model during a simulated stenosis growth. Near wall shear stress in the unstenosed artery is found to agree with literature values. The model also shows regions of low velocity, rotational flow and low near wall shear stress along parts of the walls of the carotid artery that have been identified as being prone to atherosclerosis. These regions persist during the simulated stenosis growth, suggesting that atherosclerotic plaque build-up creates regions of flow with properties that favour atherosclerotic progression.

(Some figures in this article are in colour only in the electronic version)

1. Introduction

There is a body of evidence that suggests a correlation between atherosclerosis, regions of low blood-flow velocity, rotational flow and low shear stress near the walls of arteries (Malek *et al* 1999, Asakura and Karino 1990, Gnasso *et al* 1997). Therefore, the study of the haemodynamic properties of the blood flow in these regions of the artery can lead to a greater understanding of atherosclerosis and its dependence on flow parameters. However, accurate measurements of quantities of interest, such as shear stress, are difficult to make *in vivo*, thus numerical simulation becomes a valuable investigative tool.

The lattice Boltzmann model (LBM) uses a simplified kinetic equation to simulate fluid flow and has been applied to many general problems including turbulence (Cosgrove *et al* 2003), magnetohydrodynamics (Chen *et al* 1991) and multiphase flows (Shan and Chen 1993), as well as in areas relevant to blood-flow simulation such as in flows with elastic and moving boundaries (Fang *et al* 1998), steady and pulsating flow (Fang *et al* 2002), particle

suspensions (Ladd and Verberg 2001) and flows with complex boundaries (Manwart *et al* 2002, Guo *et al* 2002).

It has been applied to a limited number of blood-flow simulations. Krafczyk *et al* (1998, 2001) consider blood flow through an artificial aortic valve. They present details of transient flows at selected fixed openings and also consider a two-dimensional model with moving leaflets. Bellerman and Sloot (2001) propose using the LBM in an ambitious project to build a 'virtual laboratory' in which LBM simulations will be combined with visualization techniques. Tamagawa and Matsuo (2004) simulate blood flow in a simple model of a blood pump or a medical fluid machine. They further estimate thrombus formation from the shear rate and the effective distance of the wall. Artoli *et al* (2004) consider a two-dimensional model of a symmetric bifurcation and compare the LBM results to a Navier–Stokes solver. The implementation of compliant walls in a two-dimensional tube representing a blood vessel was considered by Fang *et al* (2002) and Hoekstra *et al* (2004). Migliorini *et al* (2002) consider the forces acting on leukocytes due to red blood cells in a two-dimensional simulation of a blood vessel. This work was continued by Sun *et al* (2003). Li *et al* (2004a, 2005) consider the transport of red blood cells through a two-dimensional symmetric model of an artery containing a semi-circular stenosis. Measurement of force on moving boundaries and suspended particles has also been investigated (Li *et al* 2004b).

The developments outlined above show that the LBM is suited to simulating a number of features which are important in arterial haemodynamics. For a given simulation, the features that are deemed to be important can be integrated into a realistic model. In some areas, such as the simulation of transport of red blood cells, the LBM offers advantages over alternative numerical approaches. In other areas, such as simulating compliant walls, the LBM approach provides an efficient, validated approach to implementing a feature which can be found in alternative CFD codes. In the study of haemodynamics, the LBM can play as important a role as it does in other fields of fluid mechanics. The technique can be applied in the same continuum limit as the more traditional Navier–Stokes solvers. Further it can also potentially be applied at the smaller length scales of capillaries (Agarwal *et al* 2001, Spaid and Phelan Jr 1997, Lim *et al* 2002, Nie *et al* 2002) once its application to larger scales has been fully implemented.

In this paper, a two-dimensional model of the human carotid artery with a simulated stenosis growth is presented. The simulations are performed using rigid walls. This is a good approximation in the region of the stenosis (Steinke *et al* 1994) which is the area of interest here in terms of measuring the wall shear stress. Healthy regions of the artery will exhibit a level of compliance which is not considered. Even in a large artery like the carotid this will have some influence on the calculated velocity and shear stress field; however, given the two-dimensional nature of the simulation and the fact the stenosis shape, size and position varies considerably between patients, the aim of this paper is to investigate how the wall shear varies in the presence of stenosis growth, rather than determining detailed flow fields. The blood is modelled as a Newtonian fluid. This is generally assumed to be an acceptable approximation for larger vessels, such as the carotid (Quarteroni *et al* 2000, McDonald 1960). The stenosis growth is implemented in a region of the internal carotid artery which exhibits low velocity flow and a low near wall shear stress. This region is also one which is commonly prone to atherosclerotic progression. Maximum near wall shear stresses at points in the artery are compared with literature results and changes in the velocity and shear fields due to stenosis growth within the artery are examined. It is found that for the simulated stenosis growth, persistent regions of low velocity and low near wall shear are observed, particularly near the wall upstream from the stenosis growth. This suggests that the plaque build-up maintains flow conditions favourable to its progression.

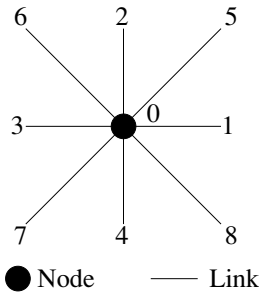

Figure 1. The D2Q9 lattice. The black circle is the node and the lines are the link directions numbered from 1 to 8.

2. Theoretical background

2.1. The lattice Boltzmann method

The lattice Boltzmann method (Chen and Doolen 1998) has recently been developed as an alternative method for simulating a range of fluid flows. In the LBM particle distribution functions, $f_i(\mathbf{x}, t)$ at point $\mathbf{x}$ at time t, are confined to move synchronously on a regular lattice. The distribution functions interact on the lattice in a way that conserves mass, momentum, isotropy and Galilean invariance. Here, i labels the lattice link the distribution function is on. The lattice used in this paper is the D2Q9, shown in figure 1.

The evolution of the distribution functions on the lattice is governed by the discrete Boltzmann equation (Chen and Doolen 1998)

$$f_i(\mathbf{x} + \mathbf{e}_i \Delta x, t + \Delta t) = f_i(\mathbf{x}, t) + \Omega_i(\mathbf{x}, t), \qquad (i = 0, 1, \ldots, M), \tag{1}$$

where for the D2Q9 lattice, see figure 1,

$$\begin{aligned} \mathbf{e}_0 &= (0, 0), && (i = 0), \\ \mathbf{e}_i &= \left(\cos\left(\frac{\pi}{2}(\mathrm{i} - 1)\right), \sin\left(\frac{\pi}{2}(\mathrm{i} - 1)\right)\right), && (i = 1, 2, 3, 4), \\ \mathbf{e}_i &= \sqrt{2}\left(\cos\left(\frac{\pi}{2}(\mathrm{i} - 1) + \frac{\pi}{4}\right), \sin\left(\frac{\pi}{2}(\mathrm{i} - 1) + \frac{\pi}{4}\right)\right), && (i = 5, 6, 7, 8), \end{aligned} \tag{2}$$

and Ω_i is the collision operator. The fluid density ρ and velocity $\mathbf{u}$ can be calculated directly from the distribution functions at each node by

$$\rho = \sum_i f_i \qquad \text{and} \qquad \rho\mathbf{u} = \sum_i f_i\, \mathbf{e}_i. \tag{3}$$

The collision operator Ω_i is given by the Bhatnagar–Gross–Krook approximation as (Bhatnagar *et al* 1954, Chen and Doolen 1998)

$$\Omega_i = \frac{-1}{\tau}\left[f_i(\mathbf{x}, t) - f_i^{\mathrm{eq}}(\mathbf{x}, t)\right], \tag{4}$$

where τ is the relaxation time and $f_i^{\mathrm{eq}}(\mathbf{x}, t)$ is the equilibrium value of the distribution function.

The equilibrium form of the distribution function in two dimensions for the D2Q9 lattice is given by (Quian *et al* 1992)

$$f_i^{\mathrm{eq}}(\mathbf{x}, t) = w_i \rho\left(1 + 3\,\mathbf{e}_i \cdot \mathbf{u} + \tfrac{9}{2}(\mathbf{e}_i \cdot \mathbf{u})^2 - \tfrac{3}{2}\mathbf{u}^2\right) \tag{5}$$

where $w_0 = 4/9$, $w_i = 1/9$ for $i = 1, 2, 3, 4$ and $w_i = 1/36$ for $i = 5, 6, 7, 8$. The relaxation time τ is related to the kinematic viscosity ν by

$$\nu = \frac{2\tau - 1}{6}. \tag{6}$$

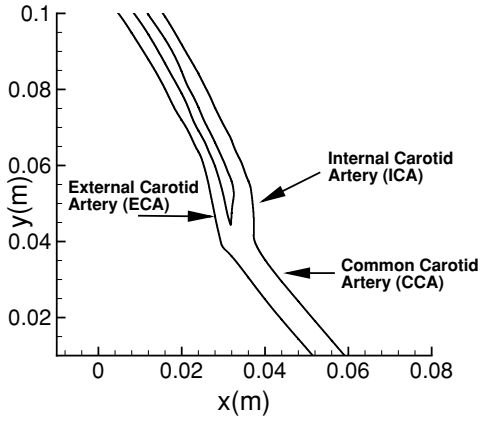

Figure 2. Carotid artery geometry used.

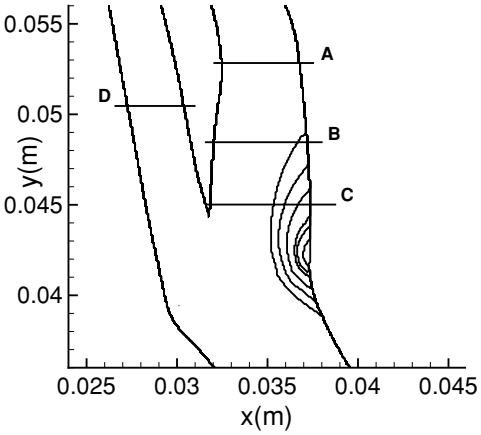

Figure 3. Stenosis implementation, every fifth increment shown. The intersections of lines A–D show the locations of near wall shear measurements taken for comparison with literature values.

The LBM reproduces the Navier–Stokes equation in the nearly incompressible limit and is second-order accurate in the body of the fluid (Chen and Doolen 1998).

A sub-grid accurate extrapolation boundary scheme (Guo *et al* 2002) is used to implement the artery geometry in the model. This boundary scheme retains the second-order nature of the LBM and is well suited to modelling stenosis growth since it enables the shape of the artery wall and the stenosis to be modelled at a resolution greater than that provided by the underlying lattice (Boyd *et al* 2004).

At the entry of the artery, a predetermined profile was implemented, this will be described in the following section. This boundary condition was implemented by setting the distribution functions at the entry equal to their equilibrium values, calculated from equation (5), for the desired velocity and density. The profile was applied uniformly across the width of the entry except for a boundary layer region of width approximately 1 mm over which the velocity reduced to zero (Boyd *et al* 2004). It can be seen from figure 2, which shows the geometry of the carotid artery, and figure 3, which shows the region of the artery used to present the results, that the entry position of the computational artery is a significantly distance from the

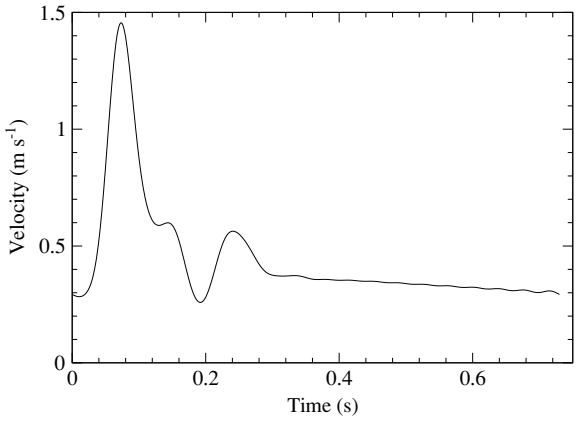

Figure 4. Pulse waveform implemented at the base of the common carotid artery geometry shown in figure 2.

measurement zone. Small variations in the entry parameters, such as the width of the boundary layer, were not found to make a significant difference to the presented results.

The unknown distribution functions at an exit site **x** were found from a linear extrapolation, based on (Neal 2002)

$$f_i(\mathbf{x}, t+1) = 2f_i(\mathbf{x}+\mathbf{e}_i, t+1) - f_i(\mathbf{x}+2\mathbf{e}_i, t+1). \tag{7}$$

3. Methods and results

Figure 2 shows the carotid artery geometry that was used for the simulation. Thirty incrementally larger stenosis growths were implemented, as shown in figure 3. The stenosis geometries were chosen to vary smoothly between increments. A pulsatile waveform, shown in figure 4 adapted from Holdsworth *et al* (1999), was implemented at the base of the artery. It was found that two pulse periods were sufficient for any transients in the flow to reduce to acceptable levels. A grid resolution of 12.5 grid points per millimetre was implemented and each pulse period corresponded to around 4 million simulation time steps.

Figure 5 shows the absolute velocity flow fields (VFF) and shear stress during the simulated stenosis growth in the carotid artery. The unstenosed artery and stenosis increments 10, 15, 20, 25 and 30 are shown. The images were all taken at the time of peak velocity in the unstenosed artery which corresponded to a time of $t = 0.066$ s into the pulse period (figure 4). In the healthy artery there is an inherent asymmetry in the wall shear stress (figure 5), due to the marked asymmetry of the artery. The stenosis was initially implemented in a region with low velocity and low near wall shear stresses. This is a region which is known to be susceptible to atherosclerosis (Gnasso *et al* 1997).

The primary region of interest is along the lower outer wall of the internal carotid artery (ICA). It can be seen that there is a large region of low velocity flow and corresponding low near wall shear indicating an area prone to atherosclerosis. It was in this region that the simulated stenosis was implemented. A similar region is also observed along the outer wall of the common carotid artery (CCA).

As can be observed from figure 5, the majority of the shear stress in the artery is concentrated along the walls. The largest changes during the simulated stenosis growth occurred near the boundary of the stenosis itself. In order to examine how the near wall shear

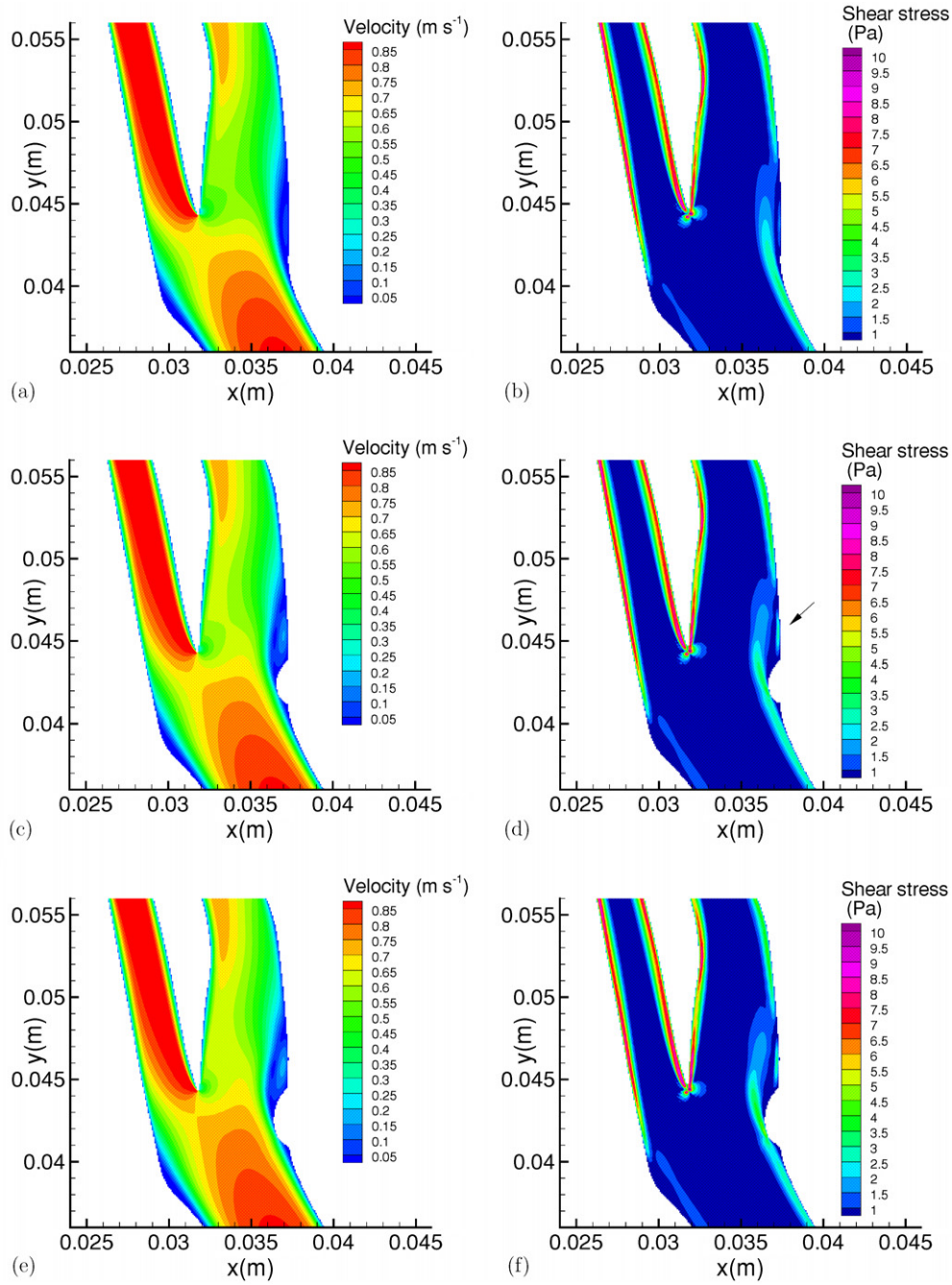

Figure 5. Velocity and shear stress fields in response to implemented stenosis growth. Unstenosed artery (a, b), stenosis increments 10 (c, d), 15 (e, f), 20 (g, h), 25 (i, j) and 30 (k, l) are shown.

stress along the boundary of the stenosis changes in response to the stenosis growth, the shear near a section of the wall extending from $y = 0.0384$ m to $y = 0.0496$ m, a total vertical distance of 11.2 mm, encompassing the stenosis boundary was examined.

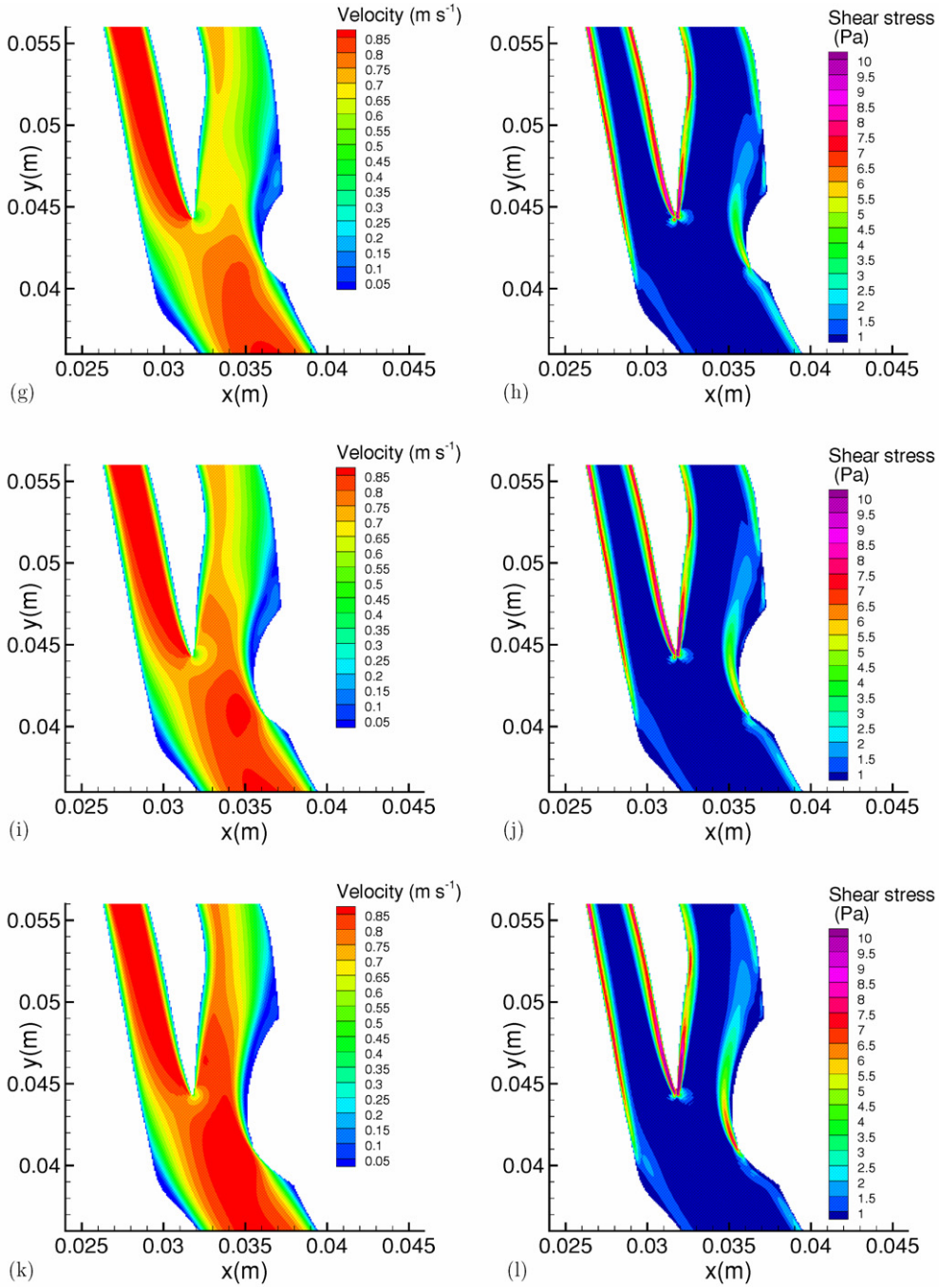

Figure 5. (Continued.)

Whilst the extrapolation scheme allows the LBM to model the carotid artery with a sub-grid boundary, the model only outputs velocity values at nodes within the fluid. Thus, to measure near wall shear stress at positions other than those lying on lattice nodes, a form

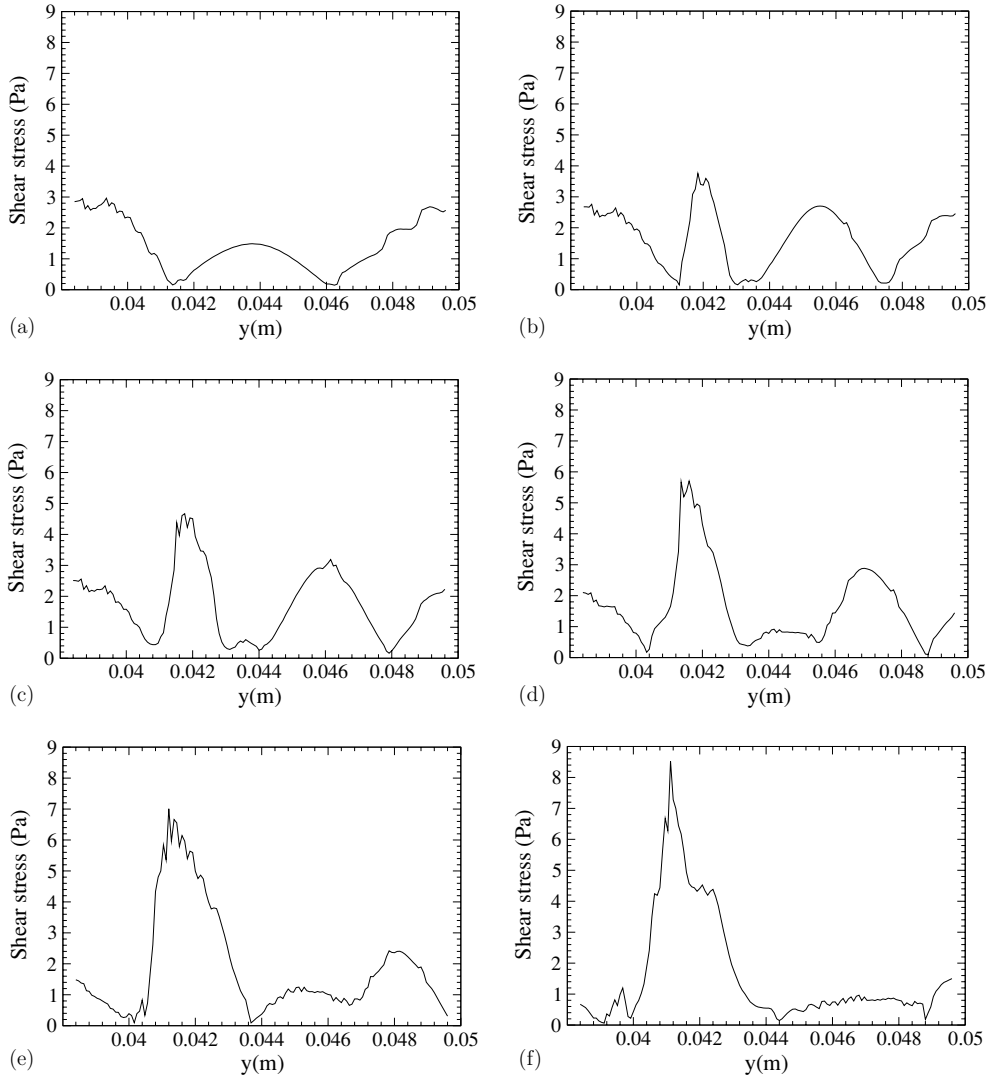

Figure 6. Response of near wall shear stress (0.16 mm from the wall) to stenosis growth, increments (a) 0, (b) 10, (c) 15, (d) 20, (e) 25 and (f) 30 are shown.

of interpolation is needed. Figure 6 shows interpolated near wall shears measured 0.16 mm from the wall along the selected wall section. This distance is not a limitation of the LBM model, but was selected to enable comparison with literature results where wall shear stress is typically measured some distance from the wall. The small oscillations in the curve are mainly generated by the fact that the interpolation is taken along the links of the lattice, which may not be perpendicular to the boundary. These curves give a good indication of the behaviour of the shear near the wall, and thus a more complicated interpolation scheme is not required.

The shear values at the inner and outer walls indicated by the lines A–D in figure 3 were compared to literature values for maximum wall shear obtained from Ku *et al* (1985) and Perktold *et al* (1991), as shown in table 1. The distribution of the shears and the numerical values qualitatively agree with the literature values. Discrepancies in the values obtained may

Table 1. Wall shear stress comparison with data obtained from Ku *et al* and Perktold *et al*.

	Inner wall shear (Pa)			Outer wall shear (Pa)		
Axial position	Ku *et al*	Perktold *et al*	LBM model	Ku *et al*	Perktold *et al*	LBM model
A	10.9	6.74	7.4	4.9	6.48	2
B	4.1	4.18	3.7	1.3	1.92	1.7
C	5	12.58	8.4	0.4	0.88	1.2
D	5.6	–	4.0	3.5	–	6.4

arise from the 2D nature of the simulation, differences in the arterial geometry, the waveform implemented, the exact location of the measurements and the manner in which they were taken.

4. Discussion

4.1. Stenosis growth

A number of interesting features were observed during the stenosis growth. The region of low velocity flow observed in figure 5(a) is preserved behind the growing stenosis. The corresponding region of low near wall shear is also preserved, particularly along the upper edge of the stenosis.

A small region of slow rotational flow near the outer ICA wall is observed inside the larger region of low velocity flow, indicated by the small shear shown by the arrow in figure 5(d). This rotational flow also persists during the stenosis growth and remains in the area immediately after the upper edge of the stenosis.

The largest changes in carotid artery VFF occur once the stenosis edge penetrates into the central higher velocity flow observed in the unstenosed artery in figure 5(a). This occurs at around increment 15, see figures 5(e) and (g). Higher velocities are exhibited in the central region of the artery during maximal stenosis growth, particularly in the upper part of the CCA.

This increase in velocity corresponds to an increase in the near wall shear stress along the lower edge of the stenosis growth. The highest shear along the edge of the stenosis corresponds to the place where the stenosis edge penetrates from the initial low velocity region into the high velocity central flow corresponding to the change in the VFF noted above. A plume of higher shear extends from this point, with the shear increasing as the stenosis grows.

The velocities and shears outside the central region near the stenosis remain relatively unchanged. The shear along the walls of the external carotid artery, the bifurcation and the inner wall of the ICA remain uniformly high. A small region of low velocity and low near wall shear is also preserved along the upper right-hand wall of the CCA. Small rotational flow was also observed in that area, indicating it may also be a region of the artery susceptible to atherosclerosis. This is borne out by the literature (Gnasso *et al* 1997).

Along the wall of the stenosis, it can be seen that as the stenosis grows, two regions of higher shear stress develop (figure 6). The high shear stress that develops in the lower portion of the wall is centred around $y = 0.042$ m. This region of high shear stress corresponds to the portion of the stenosis wall which is closest to the interface between the central high velocity flow and the region of low velocity flow seen in figure 5. The wall shear stress in this portion of the stenosis boundary increases with the stenosis growth, the length of the boundary wall which is subject to the higher shear stresses also increases with stenosis growth.

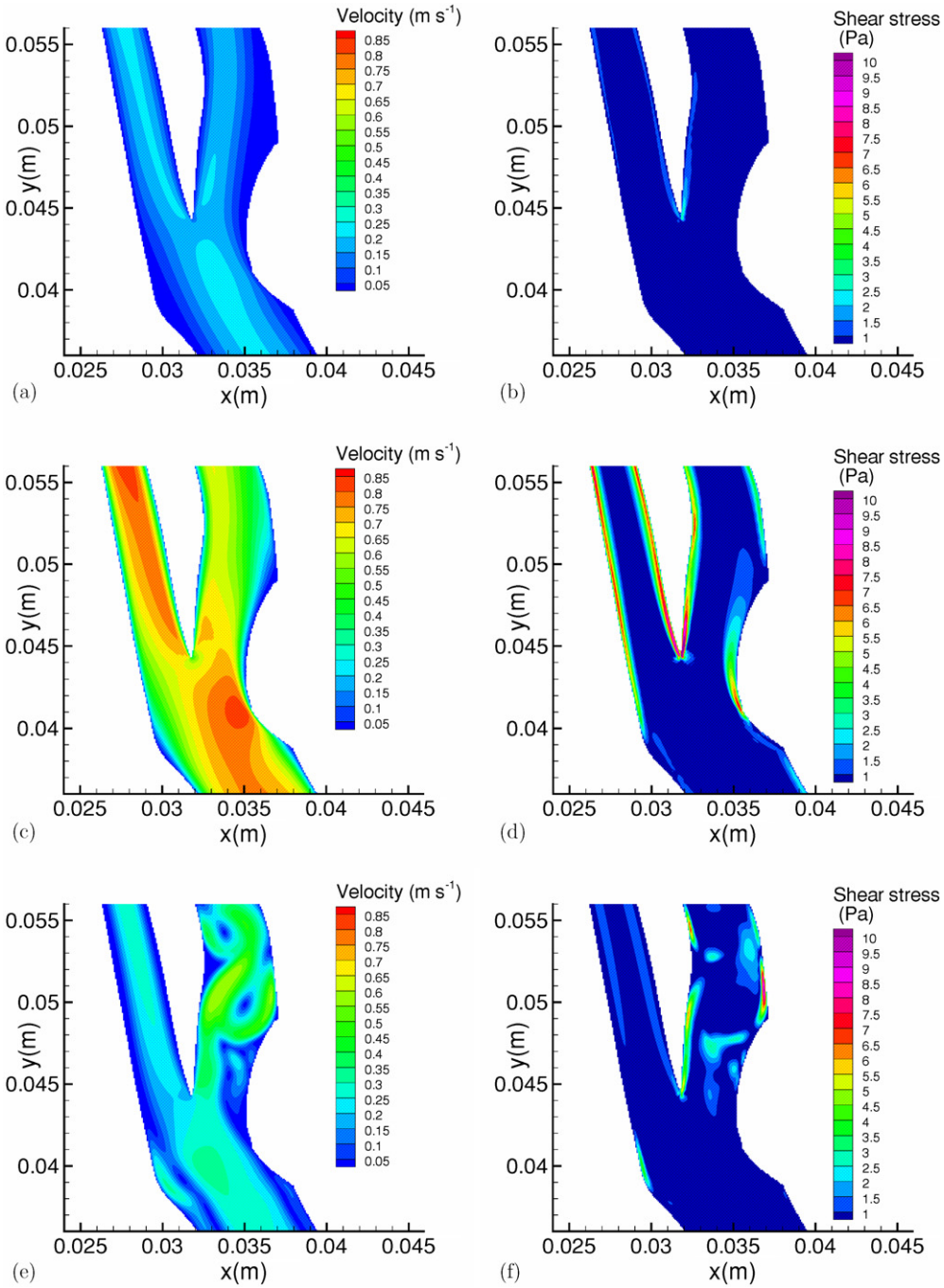

Figure 7. Velocity and shear stress during pulse cycle for maximally stenosed artery. (a, b) $t = 0.05$ s, (c, d) $t = 0.12$ s, (e, f) $t = 0.17$ s, (g, h) $t = 0.22$ s, (i, j) $t = 0.28$ s and (k, l) $t = 0.50$ s.

The second region of high wall shear stress is initially centred around $y = 0.0456$ m. As the stenosis grows, this region of high shear stress moves up the artery wall. During the initial

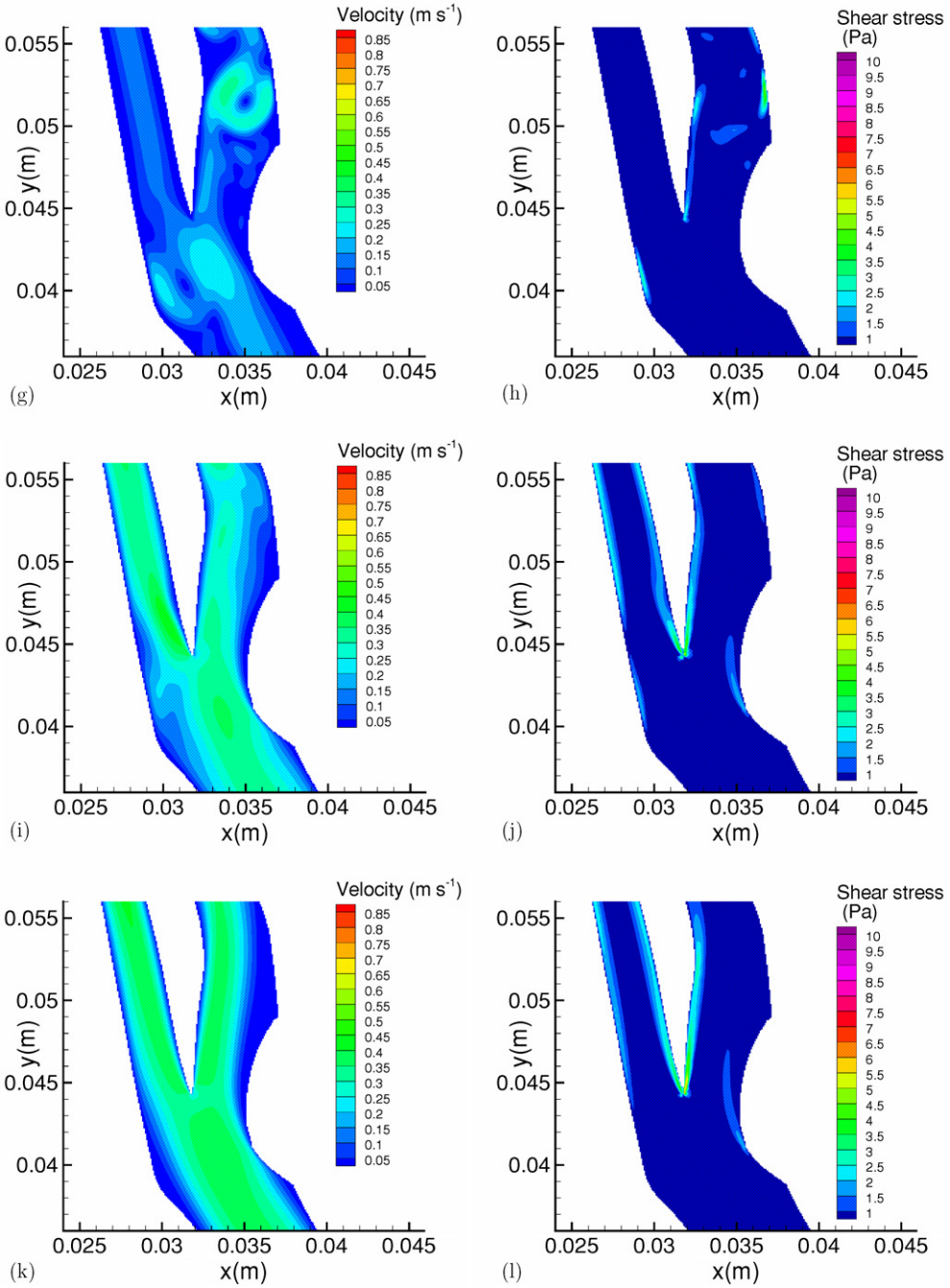

Figure 7. (Continued.)

stages of growth, this shear stress in this region increases (figures 6(a)–(c)), but then steadily decreases as it moves up the wall (figures 6(d) and (f)). This region of higher shear stress corresponds to the area of low velocity rotational flow observed in figure 5(d).

The wall shear stress between these peaks remains low for the duration of the stenosis growth. Thus, it can be seen that the high wall shear stresses created by the stenosis growth are mainly concentrated in the area of the stenosis wall that breaches the interface between the regions of higher velocity central flow and the lower velocity flow near the wall. This high shear stress may play a role in the rupturing of such a stenosis growth. This is an interesting result which merits further study, the model used in this study does not have the capability to study this process however.

4.2. Pulse cycle

It can be seen from figure 4 that the peak velocity flows presented in figure 5 only occur for a relatively brief period in the pulse cycle. For the rest of the pulse cycle the flow velocity remains relatively low. It is, however, interesting to consider how the velocity and shear stress changes over a pulse period. This is shown in figure 7 which shows the velocity and shear stress at selected times for the maximum level of stenosis.

At times $t = 0.17$ s and $t = 0.22$ s in figures 7(e, f) and (g, h), respectively, large regions of rotational flow are observed. These regions occur after the peak velocity flow and were also observed for the unstenosed case. The region of rotational flow occurs upstream of the stenosis and remains relatively fixed. It is completely dissipated before the next pulse cycle. The upper part of the large region seen in figure 7(e) does, however, break away and move further up the ICA.

The majority of the high shear stress is concentrated near the walls of the artery. It is observed that higher shear stresses are always present around the point of bifurcation in the artery. High shear stresses were also present along the walls of the ECA for a large part of the pulse cycle. The regions of rotational observed also created patches of higher shear stress in the central region of the ICA. Low wall shear stress was prevalent for most of the pulse cycle in the CCA. The right-hand wall of the ICA also had large regions of low wall shear stress during most of the pulse cycle. The largest areas high shear stress along the wall of the ICA occurred near the times of peak velocity flow.

5. Conclusion

Despite the two-dimensional nature of the model, results for the peak near wall shear compared well with literature values. Regions of low velocity, rotational flow and low near wall shear are observed in areas of the unstenosed artery known to be susceptible to atherosclerosis. These regions persist during the simulated stenosis growth as well as during the pulse cycle, suggesting that growing stenosis maintains conditions in the artery which can further promote their growth. In particular, a small region of rotational flow is observed just upstream of the upper edge of the implemented stenosis. This may increase the particle residency time for lipids and blood cells in this region and further promote stenosis progression.

High shear along the wall of the stenosis is observed when the wall of the stenosis penetrates into the higher velocity central flow, a factor that may be relevant when studying the events leading to plaque rupture and ischaemic stroke.

These results show the promise of the LBM for arterial blood-flow simulation and in particular its possible application to the further study of the haemodynamic influences on atherosclerotic progression and plaque rupture. Future directions for this work include a three-dimensional implementation of the LBM. The local nature of the LBM also makes it ideal for parallel implementation enabling three-dimensional simulations to be performed. More realistic artery conditions such as compliant walls (Fang *et al* 2002) and red blood cells

(Li *et al* 2004a) can also be added. These areas will be the focus of continuing work in this area.

Acknowledgments

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