The relative effects of arterial curvature and lumen diameter on wall shear stress distributions in human right coronary arteries

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Abstract. This study looks at blood flow in four different human right coronary arteries (RCAs), which have been reconstructed from bi-plane angiograms. A Generalised Power Law model of blood viscosity is used to study the blood flow at a particular point in the cardiac cycle. Large differences are found in the wall shear stress magnitude (WSS) distributions in the four arteries, leading to the conclusion that it is not possible to make generalisations based on the study of a single artery. The pattern of WSS is found to be related to the geometry of a particular artery, that is, lumen diameter and arterial curvature as well as a combination of these two factors. There is a strong correlation between WSS and reciprocal radius and a weaker correlation between high curvature and extremes of WSS, with high WSS on the ‘inside’ of a bend and low WSS on the ‘outside’ of a bend. This is in contrast to the situation for a simple curved tube with constant radius where the inverse is observed. For each artery, a region proximal to the acute margin is identified where low WSS is found and where WSS is lower on the ‘inner’ wall of the RCA than on the ‘outer’ wall. This region is one where several studies have found that the human RCA preferentially exhibits atherogenesis.

1. Introduction

Although atherosclerosis, the leading cause of death in the Western world, is associated with systemic risk factors such as hypertension, smoking, hyperlipidemia and diabetes mellitus (Malek et al. 1999), it is known that these factors can explain no more than half the variability in the occurrence of atherosclerotic lesions or coronary heart disease (Zhu & Friedman 2003). This leaves the so-called geometric risk factors (Nerem 1992), which influence the haemodynamic environment within the coronary arteries and hence the initiation and development of atherosclerosis (Zeng et al. 2003).

In this paper we continue our modelling studies in four different right coronary arteries (RCAs) begun in (Johnston et al. 2004), with the aim of elucidating the role played by changes in lumen diameter and curvature on the particular pattern of plaque localisation in the RCA. Most previous studies have considered only one RCA in any one study. Comparisons are presented here in terms of wall shear stress magnitude (WSS) distributions, as WSS is thought to be a significant factor in the onset of coronary heart disease (Fry 1968, Caro et al. 1971, Giddens et al. 1990, Ku et al. 1985). An understanding of the effect of these changes on flow in the RCA will give better insight into the relationship between the haemodynamic environment and the initiation and development of atherosclerosis. In addition, in the future, shear stress profiling (Feldman et al. 2002), for example, may lead to individual assessment of the risk of coronary heart disease and future therapeutic strategies (Malek et al. 1999), including better management of systemic risk factors.

Histological studies (Ojha et al. 2001) have shown that the RCA displays localised atherosclerotic plaques throughout its length and that the proximal region appears to be a site of eccentric intimal thickening, with maximum thickness on the myocardial side of the artery. The study also found that, even though thickening does occur in the acute margin and distal regions, no distinct pattern or location is evident. This is investigated in the present study by comparing WSS on lines drawn down the ‘inner’ (on the epicardial surface) and the ‘outer’ (opposite the epicardial surface) walls of the artery.

Since the RCA has relatively few bifurcations, it has been suggested (Asakura & Karino 1990, Friedman 1990) that curvature of the RCA produces the haemodynamic stimuli for the initiation/promotion of atherosclerosis. Other studies (Berthier et al. 2002, Kirpalani et al. 1999) have suggested that the local vessel diameter appears to play a role in influencing WSS patterns. This study looks at WSS distributions in four different human RCAs under steady state flow conditions. The arteries have been reconstructed in three dimensions from pairs of bi-plane angiograms, using a recently developed technique based on centre-line extraction and radius determination (Corney et al. 2001, Corney et al. 2004). These images were chosen because they showed no sign of atherosclerosis. WSS distributions, which were produced in previous steady flow simulations (Johnston et al. 2004), are related to the radius and curvature of the artery. Then, in order to separate out the effects of arterial curvature and lumen diameter,

simulations are performed on variants of the original arteries: the first involves an artery of constant lumen diameter, but with the same curvature as the original artery and the second involves a straight artery, where the lumen diameter varies as in the original artery. The WSS distributions produced in these two arteries are then compared with that of the original artery.

2. Methods

The four different human RCAs used in this study are labelled A, B, C and D and their reconstructions are shown in figure 1. These arteries were reconstructed in 3D from pairs of angiographic images, which showed no sign of atherosclerosis, using recently developed techniques based on centre-line extraction and radius determination (Corney et al. 2001, Corney et al. 2004). A full description of all these arteries may be found in an earlier paper (Johnston et al. 2004), while the variation in arterial radius (calculated as a geometric mean of the radii for each view), as a function of distance along the centre-line of the artery, is shown in figure 2.

The steady flow of blood in the RCA is modelled by assuming that blood can be represented by an incompressible fluid which is governed by the conservation of momentum equations

$$\rho \mathbf{v} \cdot \nabla \mathbf{v} = -\nabla \cdot \boldsymbol{\tau} - \nabla P \quad (1)$$

and the continuity equation

$$\nabla \cdot \mathbf{v} = 0 \quad (2)$$

where $\mathbf{v}$ is the 3D velocity vector, P pressure, ρ density (1050 kg m^{-3}) and $\boldsymbol{\tau}$ the shear stress tensor. Writing the conservation of momentum equations in this form allows a non-Newtonian model for blood viscosity to be included. We have shown previously (Johnston et al. 2004, Johnston et al. 2006) that the Generalised Power Law model (Ballyk et al. 1994) for blood viscosity provides an accurate representation of the non-Newtonian effects of blood in regions of low shear and this model will be used here. See (Johnston et al. 2004) for a full discussion of this work.

The highly non-linear governing equations (1) and (2) are solved using the finite volume method as implemented in the package CFD-ACE (CFDRC, Huntsville, Alabama, USA). The finite volume mesh is created from the reconstructed coronary artery (Corney et al. 2001, Corney et al. 2004) by meshing an inlet plane in 2D using quadrilaterals and this mesh is extruded along the centre-line of the artery to create a 3D mesh consisting of brick elements. Mesh independence has been confirmed by performing additional simulations on a more detailed mesh, see (Johnston et al. 2004).

The following boundary conditions are assumed: a no-slip condition along the walls of the artery; stress free conditions with the gauge pressure set to zero at the outlet and an elliptic paraboloidal velocity profile at the inlet. The inlet (x-y plane) is represented by an ellipse with semi-major and semi-minor axes a and b respectively and the velocity profile is of the form $w(x, y) = v_0 \left(1 - \frac{x^2}{a^2} - \frac{y^2}{b^2}\right)$, with centre-line velocity, v_0 . The form

of the velocity profile is discussed in (Johnston et al. 2004), while the elliptical inlet is suggested by artery radius data.

Branch flows are not included, in line with several previous studies, (Myers et al. 2001, Berthier et al. 2002, Krams et al. 1997, Zeng et al. 2003), despite the fact that flow rate ratios have been seen to significantly affect WSS (Asakura & Karino 1990).

In order to separate out the various effects on flow in the artery, simulations were performed in two ‘new’ arteries, for comparison with the simulations in the original arteries. The meshes for the new arteries were based on the mesh for the original artery and were constructed as follows: the first artery (the so-called ‘varying curvature’ artery) has the same curvature as the original artery but has a circular cross-section, based on the geometric mean of the inlet ellipse; the second artery (the so-called ‘varying radius’ artery) is merely a straight tube with the length of the original artery and with the radius varying according to the original artery.

Simulations were carried out for centre-line velocities of 0.02, 0.2 and 1.0 ms⁻¹ under the assumptions mentioned above. For comparison purposes with other flow simulations a Reynolds number, Re , can be defined as

$$Re = \frac{\rho D v_0}{\mu_\infty}$$

where ρ is defined above, D is the inlet diameter of the artery, v_0 is the centre-line velocity and μ_∞ is the Newtonian limit viscosity in the Generalised Power Law model. As mentioned above $\rho = 1050$ kg/m³; from figure 2 a reasonable value of D is 5 mm and from (Ballyk et al. 1994) $\mu_\infty = 0.0035$ Pa s. The Reynolds numbers corresponding to the three velocities above are then 30, 300 and 1500, respectively.

From these simulations, plots of WSS distributions were produced. For example, figure 3 shows the WSS distribution in artery B for centre-line velocity 0.2 ms⁻¹ from two different perspectives. Also shown in part (a) of this figure is a line down the ‘outer’ wall of the artery. The line was drawn using the same point on each of the ellipses from which the mesh was constructed. A corresponding ‘inner’ line (180° rotation from the ‘outer’ line) is shown in figure 3(b).

In this work, comparisons of ‘shapes’ of graphs will be quantified using the correlation coefficient, CC , as defined below.

$$CC = \frac{(\mathbf{s} - \bar{\mathbf{s}}) \cdot (\mathbf{t} - \bar{\mathbf{t}})}{\|\mathbf{s} - \bar{\mathbf{s}}\| \|\mathbf{t} - \bar{\mathbf{t}}\|} \quad (3)$$

where $\mathbf{s}$ is a vector representing the first set of data, $\mathbf{t}$ is a vector representing the second set of data and where $\bar{\mathbf{s}}$ and $\bar{\mathbf{t}}$ are the means of $\mathbf{s}$ and $\mathbf{t}$ respectively. Values of CC near unity indicate very good positive correlation.

The final quantity to be used as a point of comparison in this study is the curvature of the artery. Here the curvature k is defined as a function of distance s , along the centre-line of the artery, by

$$k = \sqrt{\left(\frac{d^2x}{ds^2}\right)^2 + \left(\frac{d^2y}{ds^2}\right)^2 + \left(\frac{d^2z}{ds^2}\right)^2} \quad (4)$$

for a curve with parametric equation $x = x(s)$, $y = y(s)$ and $z = z(s)$.

3. Results

3.1. Positions of Extrema

A consideration of plots of WSS, such as those presented in figure 3 for artery B with $v_0=0.2 \text{ ms}^{-1}$, reveals that there are many examples where WSS is high on the inside of a bend and low on the outside of the bend. Similar points can also be found in all four arteries presented previously (Johnston et al. 2004). This is the inverse of previous findings for a simple curved tube of constant radius (Perktold et al. 1991, Qiao et al. 2004, Jung et al. 2006), where WSS was found to be high on the outside and low on the inside of a bend.

To ensure that these findings are not confined to a particular inlet velocity, a study was undertaken to compare positions of extremes of WSS for various centre-line velocities. Plots of WSS in artery B (normalised using the maximum value of WSS for the various centre-line velocities considered) are shown in figure 4. Visual inspection shows that the positions of peaks and troughs of WSS are quite consistent between values of v_0 , especially for $v_0=0.2 \text{ ms}^{-1}$ and $v_0=1.0 \text{ ms}^{-1}$.

To further quantify the above observations, pointwise correlation coefficients were calculated for the three velocities on both ‘inner’ and ‘outer’ lines in all four arteries and are given in Table 1. The values in the table indicate that the pattern of WSS is almost independent of inlet velocity in a particular artery.

To eliminate the possibility of a radius effect in the positions of high and low WSS at a bend, plots of WSS in the ‘varying curvature’ artery (where radius is constant) were studied. In each case a number of bends with high WSS on the inside and low WSS on the outside were found; see, for example, the points labelled I–V in figure 5 for ‘varying curvature’ artery B with $v_0=0.2 \text{ ms}^{-1}$.

The variation of radius and curvature in real arteries is clearly more complicated than in a simple curved tube of constant radius and so later sections in this paper will consider the individual and then later combined effects of radius and curvature on overall WSS.

3.2. A comparison of WSS with radius in the ‘varying radius’ and original arteries

It was noted (Johnston et al. 2004) that there appeared to be a connection between local arterial radius and WSS, as would be expected from conservation of mass. This is illustrated in the left-hand panel of Table 2 labelled ‘Original artery’. This panel gives the correlation coefficient between the WSS down the ‘inner’ line (as shown in figure 3(b) for artery B) and the reciprocal of the local arterial radius for all four original arteries and the three inlet velocities. The correlations lie in the range 0.41 to 0.95 and while there are very good correlations ($CC > 0.8$) in many cases, particularly for low inlet velocities, it is obvious that factors other than radius affect the WSS distribution.

In order to eliminate the effect of curvature, now consider WSS in the ‘varying radius’ (but straight) artery. Correlation coefficients comparing reciprocal radius and WSS in this artery for various centre-line velocities were found to lie in the range 0.58 to 0.99 (right-hand panel of Table 2) and individually (except in one case) each is higher than those in the original artery. Such an observation would be expected as the effect of curvature has been removed. In addition, for both artery types the correlations decrease as centre-line velocity increases. This means that, while there is a strong inverse relationship between radius and WSS, this relationship does not account for the entire WSS distribution, especially in the case of higher centre-line velocities. Further, comparison of detailed plots of WSS and reciprocal radius (not shown) demonstrate that in the distal half of the artery there are effects that do not appear to be directly related to changes in radius.

3.3. A comparison of WSS with curvature in the ‘varying curvature’ and original arteries

Table 3 presents the correlation coefficients between WSS and curvature, k , in the original and ‘varying curvature’ arteries. Here the WSS is taken along the ‘inner’ line as demonstrated in figure 3(b). Unlike the comparison with the arterial radius in Table 2, there is a poor correlation in the case of curvature, even when the effect of radius variation is removed.

This can partly be rationalised by considering plots such as figure 5, which shows WSS in the ‘varying curvature’ artery B for $v_0=0.2 \text{ ms}^{-1}$, and other plots of curvature versus ‘inner’ and ‘outer’ WSS (not presented). Notice that point II in figure 5 corresponds to a peak in curvature and a peak in WSS on the ‘inner’ line and the same is true at the point V. However, at points I, III and IV, there is again a peak in centre-line curvature, but the peak in WSS is on the ‘outer’ line with a trough on the ‘inner’ line and this results in a lack of correlation between centre-line curvature and WSS taken on the ‘inner’ line. This can be summarised by the following: high WSS on one line (either ‘inner’ or ‘outer’) corresponds to high curvature on the same line, while low WSS on one line corresponds to high curvature on the other line.

3.4. A comparison of WSS in the original and ‘varying’ arteries

In this section the aim is to assess the individual contributions made by each of radius and curvature to the overall WSS. Even though there is no direct correlation between curvature and the WSS produced in the ‘varying curvature’ artery, it can be seen (for example in figure 6(a)) that the WSS in the original artery clearly follows the WSS in the ‘varying radius’ artery, but with the effect of WSS in the ‘varying curvature’ artery superimposed upon it. A comparison of the WSS produced by the sum of the WSS of the ‘varying radius’ and ‘varying curvature’ arteries with WSS in the original artery is presented in figure 6(b). It is clear that the pattern of the WSS is similar in each case, but that the magnitudes vary. This would be expected at the start of the artery,

since the sum of the ‘varying radius’ and ‘varying curvature’ arteries involves higher mass flow than the original artery. For the lower centre-line velocities $v_0=0.02$ and 0.2 ms^{-1} , the two WSS graphs remain largely parallel throughout the length of the artery, indicating that the WSS distribution is influenced independently by each of the radius and curvature of the artery. For the higher centre-line velocity $v_0=1.0 \text{ ms}^{-1}$, however, the two graphs do not always remain parallel. This shows that radius and curvature interact in the mid to distal region for $v_0=1.0 \text{ ms}^{-1}$, to produce higher values of WSS than would be expected simply by the sum of the two effects.

Another interesting observation is that certain combinations of radius and curvature act together to lower WSS. For example, in figure 6(a) and in similar figures for other arteries (not presented), it can be seen that there are points in the artery where WSS in the original artery is less than that produced by one of the ‘varying radius’ or ‘varying curvature’ arteries alone. Such points can be seen in artery B for normed arc length values of about 0.08, 0.18 and 0.28 across all centre-line velocities.

Correlation coefficients between WSS in the original artery and the sum of WSS in the two ‘varying’ arteries are given in Table 4, for graphs like those in figure 6(b), for all four arteries. Similar results (not presented) were found for correlations where the WSS on an ‘outer’ line is used. This table also includes correlation coefficients for the proximal half of the artery, where the correlations appear to be higher for some arteries. For example, in artery B, there are very good correlations ($CC > 0.9$) between each pair of graphs and the same is true in arteries A and D. The correlations in artery C are not as good, but this may be due to the fact that it is a more complex artery, with more variations in radius and curvature than the other three arteries, or it may be because it is an ‘ectatic’ artery with a much larger diameter than the others.

Another trend, which is consistent across all the arteries, is that the correlations for the whole artery decrease as v_0 increases. In addition, for the two higher v_0 values, correlations for the proximal half of the artery are higher than for the whole artery. These are interesting observations as they appear to indicate that, while at low values of v_0 the WSS distribution is almost solely dictated by the combination of curvature and radius, that is not entirely the case for the higher velocities. In that case, it would seem that secondary flows become significant in determining the WSS distribution, particularly in the distal half of the artery.

3.5. A comparison of WSS on ‘inner’ and ‘outer’ lines

As mentioned earlier in Section 3.1 and as can be seen in figure 3, peaks of WSS on the ‘inner’ wall of the artery often occur opposite to troughs of WSS on the ‘outer’ walls of the artery and vice versa. This is of interest since histological studies (Ojha et al. 2001) have shown that eccentric intimal thickening occurs predominantly along the inner wall of the RCA proximal region.

A detailed study of plots of WSS (an example for artery B with inlet velocity 1.0 ms^{-1} is shown in figure 7) for all four original arteries and three inlet velocities shows

that the WSS distribution along the ‘inner’ line is quite different from that along the ‘outer’ line. They do, however, display the same trend of increasing magnitude from the proximal to the distal ends of the artery. In particular, for higher values of centre-line velocity, many of the peaks of the ‘inner’ graphs occur at similar positions to the troughs of the ‘outer’ graphs and vice versa and this is also true in the ‘varying curvature’ artery.

Correlations between peaks and troughs in the ‘varying curvature’ artery are presented in Table 5. These show quite good negative correlations (CC around -0.6) in arteries B and D, poorer correlations (CC around -0.3) in artery A and in one case in artery C and no correlation for the other two cases in artery C. These results may be affected by the normalisation of arc length values, which results in the peaks and troughs being slightly displaced due to the distance down the ‘outer’ line being different to the distance down the ‘inner’ line. However, in general the results do indicate an inverse relationship, that is, peaks roughly opposite troughs.

3.6. WSS in the proximal region

It was also observed that for each artery a region can be found, between the inlet and the acute margin, where the WSS on the ‘inner’ wall is less than that on the ‘outer’ wall. Such a region can be seen in figure 7, for approximate normed wall arc length values of 0.04-0.12. Examination of the angiograms for each artery confirms that these are indeed proximal regions. When $v_0=0.02 \text{ ms}^{-1}$, these regions exist for two of the arteries only, whereas they can be found for all arteries for $v_0=0.2 \text{ ms}^{-1}$ and $v_0=1.0 \text{ ms}^{-1}$.

For all arteries and inlet velocities there is an initial inlet region of approximately 1-1.5 cm where the result is reversed and ‘outer’ WSS is lower than ‘inner’ WSS, a finding which is also consistent with the studies in (Kirpalani et al. 1999) and (Myers et al. 2001). The paper (Ojha et al. 2001) mentions previous studies on this inlet region and discusses reasons why the first centimetre of the RCA may be resistant to intimal thickening.

4. Discussion

This paper presents WSS distributions calculated in four different RCAs reconstructed from biplane angiograms. The WSS distributions are markedly different from one another, except for a general trend where WSS increases from the proximal to the distal ends of the artery (mainly due to tapering of the artery, as can be seen in figure 2). Differences in the geometry of the artery, both in curvature and radius, clearly affect flow and WSS patterns, as shown in previous single artery studies (Myers et al. 2001).

The present study correlates WSS with both curvature and radius in the four different RCAs. These correlations are made both in the original artery and also in two variants of it, the ‘varying radius’ and the ‘varying curvature’ artery, so that the effect of radius and curvature, respectively, could be removed to allow the study of each one to be made independently. A good correlation is found between reciprocal radius

and the WSS distribution in the original artery and an even stronger correlation with WSS in the ‘varying radius’ artery. Correlations between curvature and WSS were not good in either the original or the ‘varying curvature’ artery. This was rationalised as being a consequence both of the definition of curvature (a positive quantity with no direction attached) and the fact that because WSS is found on a particular line down the artery this line will not include all the bends where there is high curvature. Taking this into account, there does appear to be a connection between high curvature and high WSS on the ‘inside’ of a bend, with low WSS on the ‘outside’ of the same bend.

Generally, an alternating pattern of high and low WSS, relative to one another, can be seen in the proximal region of the artery. This has also been reported in several previous modelling studies (Myers et al. 2001). In the case of the arteries studied here, this appears to be predominantly related to curvature, as the radius is relatively large and only varies slowly at this point.

For each of the four arteries studied here, a region can be found, between the inlet and the acute margin, where the WSS on the ‘inner’ wall is less than that on the ‘outer’ wall. This region has previously been shown to be one where the RCA preferentially exhibits atherogenesis, as reported in (Kirpalani et al. 1999). In addition, histological studies (Ojha et al. 2001) have found eccentric intimal thickening on the myocardial side of this region.

Two previous studies in the one artery, by (Kirpalani et al. 1999) and (Myers et al. 2001), not only found that ‘inner’ WSS was less than ‘outer’ WSS in the proximal region, but that this was generally true throughout the artery, excluding the inlet. This is not the case for any of the four arteries studied here (see figure 7 for artery B), as in this work extremes of WSS alternate between the inner and outer walls. However, the pattern of alternating high and low WSS on a particular wall is a common finding in the three studies. In addition, the present study did not find, as did (Kirpalani et al. 1999) but not (Myers et al. 2001), that the large differences between ‘outer’ and ‘inner’ WSS appeared to be unique to the proximal region.

A number of studies (Perktold et al. 1991, Qiao et al. 2004, Jung et al. 2006) have reported that, in general, high WSS can be found on the outside of a bend and low WSS on the inside of a bend. This is consistent with the movement of the fluid away from the inside of the bend and towards the outside of the bend. However, it can be seen that in this work there are many instances where this is not the case (figures 3 and 5). The discrepancy is not related to the inlet velocity, nor is it related solely to radius changes, since the same effect can be seen in figure 5, which is a ‘varying curvature’ artery and hence is of constant radius. It would seem that fluid flow (and hence WSS) is more complex in arteries which contain multiple changes of curvature, including out-of-plane changes, than in simple in-plane curved tubes of constant radius, such as those in the studies cited above.

It has also been mentioned (Caro 2001) that several workers have reported a helical distribution of atherosclerotic lesions downstream of non-planar regions in arteries, consistent with a helical distribution of low wall shear. Examination of plots, such

as figures 3(a) and (b), shows that this may be the case in artery B, possibly after the first and second bends and, more likely, along the relatively straight section about one third of the way down the artery. Investigations were carried out in the ‘varying curvature’ artery, so that the effect of radius variation could be removed. It appears that this effect is more pronounced for the higher inlet velocities, but it is difficult to quantify and further work on a simpler version of the artery, perhaps containing just one non-planar bend, is needed.

5. Conclusions

Large differences are found in WSS distributions between the four arteries studied, just as there are significant variations in radius and curvature between arteries. This leads to the conclusion that it is not possible to make generalisations based on the study of a single artery. Various points are found in all four arteries studied, where there is high WSS on the inside of a bend and low WSS on the outside of the same bend. This is the inverse of findings for a simple curved tube of constant radius and yet the difference is not the result of changes in radius since it is still observed in the ‘varying curvature’ artery, which is of constant radius.

A high correlation is found between reciprocal radius and the WSS distribution produced in the RCA, regardless of the line chosen down the wall of the artery along which the WSS is presented, while there is a low correlation between curvature and WSS produced on any particular line down the artery.

The relationship between radius and curvature in the production of WSS is more complex than previously thought. In all of the arteries studied, the WSS distribution produced is clearly a combination of the effects of both radius and curvature. At low inlet velocities, radius and curvature appear to act independently to produce WSS. However, at mid to high inlet velocities, they act together to produce values of WSS which are higher than those which would be expected from the effects of radius and curvature alone. It seems that at higher velocities, secondary flows become significant in the production of WSS.

The study of WSS on the ‘inner’ and ‘outer’ walls of the artery shows that there is a relationship between extremes of WSS in these two places; namely, that a peak in WSS on one of the ‘inner’ or ‘outer’ lines of the artery corresponds to a peak in curvature on the **same** line, while a trough in WSS on one of the ‘inner’ or ‘outer’ lines of the artery corresponds to a peak in curvature on the **other** line.

In each of the four arteries studied, a region of low WSS has been found, where the minimum values of WSS on the ‘inner’ wall are less than those on the ‘outer’ wall. These regions are past the inlet and proximal to the acute margin in each case. This supports a connection between low WSS and atheroma production (Krams et al. 1997), when taken with the findings of several studies reported in (Myers et al. 2001), which have shown that the human RCA preferentially exhibits atherogenesis in regions proximal to the acute margin.

In presenting these conclusions, it must be noted that they are made under the model assumptions given in Section 2. For example, the artery is assumed to be rigid and fixed, whereas the RCA is distensible and moves with the beating heart. However, (Zeng et al. 2003) has shown that the haemodynamic effects of RCA motion can be ignored as a first approximation in modelling studies. Another assumption is the use of steady rather than pulsatile flow. Studies in the RCA, such as (Feldman et al. 2002, van de Vosse et al. 2001) have shown that the time-averaged WSS distribution of pulsatile flow is in close agreement with the WSS distribution produced from steady flow simulation.

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Captions

Table 1: Correlations between pairs of WSS distributions for various centre-line velocities

Table 2: Correlation coefficient for reciprocal radius versus WSS in (a) the original artery and (b) the ‘varying radius’ artery

Table 3: Correlation coefficients for WSS versus curvature in (a) the original artery and (b) the ‘varying curvature’ artery down an ‘inner’ line

Table 4: Correlation coefficients for WSS in the original artery versus the sum of WSS in the ‘varying curvature’ and ‘varying radius’ arteries, in (a) the whole artery and (b) the proximal half of the artery

Table 5: Correlations between WSS distributions for ‘inner’ and ‘outer’ lines for the ‘varying curvature’ artery.

Figure 1: Reconstructions of the four coronary arteries considered in the study

Figure 2: Arterial radius as a function of arc-length for each of the four arteries

Figure 3: Wall Shear Stress distribution (in Pa) for $v_0=0.2\text{ms}^{-1}$ in artery B on (a) an ‘outer’ line and (b) an ‘inner’ line

Figure 4: Normed Wall Shear Stress in artery B on an ‘inner’ line for various centre-line velocities

Figure 5: Wall Shear Stress distribution (in Pa) in the ‘varying curvature’ artery B for $v_0=0.2\text{ ms}^{-1}$

Figure 6: Wall Shear Stress (in Pa) along an ‘inner’ line for $v_0=0.2\text{ ms}^{-1}$ in (a) each of the arteries based on artery B and (b) in the original artery B, as well as WSS in the sum of the ‘varying radius’ and ‘varying curvature’ arteries based on artery B

Figure 7: Wall Shear Stress along ‘inner’ and ‘outer’ lines in artery B for $v_0=1.0\text{ ms}^{-1}$

Centre-line velocity v_0 (ms^{-1})	'Inner' line				'Outer' line			
	A	B	C	D	A	B	C	D
0.02/1.0	0.89	0.65	0.74	0.83	0.71	0.49	0.57	0.82
0.02/0.2	0.87	0.60	0.89	0.89	0.73	0.59	0.83	0.85
0.2/1.0	0.95	0.91	0.93	0.96	0.94	0.93	0.90	0.97

Table 1.

Centre-line velocity v_0 (ms^{-1})	Original artery				'Varying radius' artery			
	A	B	C	D	A	B	C	D
0.02	0.95	0.81	0.76	0.95	0.98	0.98	0.82	0.99
0.2	0.79	0.44	0.52	0.83	0.93	0.92	0.65	0.97
1.0	0.62	0.41	0.72	0.76	0.92	0.85	0.58	0.93

Table 2.

Centre-line velocity v_0 (ms^{-1})	Original artery				'Varying curvature' artery			
	A	B	C	D	A	B	C	D
0.02	-0.14	-0.20	0.01	0.14	0.30	0.43	0.17	0.45
0.2	-0.14	-0.08	-0.05	0.06	0.07	0.17	-0.02	0.21
1.0	-0.13	-0.05	0.02	0.11	-0.01	0.18	0.14	0.26

Table 3.

Centre-line velocity v_0 (ms^{-1})	Whole artery				Proximal half of artery			
	A	B	C	D	A	B	C	D
0.02	0.94	0.98	0.65	0.99	0.93	0.98	0.72	0.98
0.2	0.91	0.91	0.51	0.96	0.95	0.93	0.76	0.98
1.0	0.89	0.90	0.64	0.95	0.94	0.92	0.68	0.98

Table 4.

Centre-line velocity v_0 (ms^{-1})	Original artery			
	A	B	C	D
0.02	-0.18	-0.65	-0.08	-0.72
0.2	-0.36	-0.66	-0.39	-0.62
1.0	-0.23	-0.58	0.06	-0.55

Table 5.

Artery A

Artery B

Artery C

Artery D

Figure 1.

Figure 2.

Figure 3.

Figure 4.

Figure 5.

Figure 6.

Figure 7.