Transient Flow and Shear Stress Measurements in an anatomical Model of the Human Carotid Artery

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Abstract It is well known that the initiation, development and progression of arterial disease such as atherosclerosis is directly linked to the prevailing haemodynamic environment and wall shear stress (WSS) distribution within the arteries. This paper presents velocity and WSS measurements in a physiologically realistic carotid artery model under pulsatile flow conditions. The physiological flow phantom is developed from in vivo MRI angiogram data and the physiological in vivo inlet waveform is measured with phase contrast MRI. The flow field within the carotid artery model is investigated by means of phase locked planar PIV measurements. Spatial and transient WSS is measured using our recently developed interfacial PIV (iPIV) technique (Buchmann et al., 2008, 14th Int Symp. on Applications of Laser Techniques to Fluid Mechanics). The investigated flow geometry exhibits interesting geometric complexity that potentially provides more insight into the mechanisms of atherosclerosis within the human carotid artery. The analysis of time averaged and steady state WSS provides for an interesting comparison and conclusions regarding the applicability of the steady flow assumption are drawn. Furthermore, the pulsatile flow results illustrate the time-dependent variation flow recirculation, secondary flows and WSS distribution.

1. Introduction

Atherosclerosis is a systemic proliferation and inflammatory disease of the human vascular system, which leads to arterial remodeling and narrowing (i.e., stenosis) due to thrombosis. In its most sever form, atherosclerosis can lead to complete vessel occlusion (embolism) and is a major cause for stroke and ischemic infarction. The causative factors that contribute to the development of atherosclerosis are complex (Chatzizisis et al., 2007) and haemodynamic factors are an important determinant in the localised development of the disease (Zarins et al., 1983; Ku et al., 1985). The pathology of atherosclerosis primarily involves the response of the arterial wall to a disturbed haemodynamic environment in form of low and oscillating wall shear stress (WSS), flow separation and departure from unidirectional flow (Traub and Berk, 1998).

On of the predominant sites for atherosclerosis formation is the common carotid artery (CCA) and its bifurcation into the internal carotid artery (ICA) and external carotid artery (ECA) as shown in Figure 1. At the bifurcation, the vessel wall exhibits strong curvatures that lead to the development of a transverse pressure gradient (due to centripetal forces), which in turn promotes flow separation, secondary flows and spatially and temporally varying WSS. Studies by (Zarins et al., 1983) have demonstrated a clear correspondence between the formation of atherosclerosis and the prevailing haemodynamic environment in the human carotid artery.

This study will focus on WSS characteristics under pulsatile flow, as there are very few published studies concurring the measurement of these quantities in a realistic arterial geometries. The geometry of an individual carotid artery differs markedly from the population average (Goubergrits...
et al., 2002; Thomas et al., 2005) and the underlying haemodynamics and plaque growth are likely to be influenced by the individual vessel geometry. This causality is referred to as "geometric risk factor" in (Friedman et al., 1983) and a number of research groups have requested a greater focus on physiologically realistic geometries to further understand the individual blood flow characteristics (Perktold et al., 1998). While this demand is well met with numerical studies (Karner et al., 1999; Lee et al., 2009) such detailed in-vitro studies in physiological accurate models are still sparse.

Detailed velocity measurements in such anatomically realistic geometries (healthy and diseased) include LDV measurements by Liepsch (2002) and Liepsch et al. (1998), but who did not assess WSS and more recently, planar PIV measurements by Bale-Glickman et al. (2003) and Buchmann et al., (2009) as well as stereo-PIV measurements by Vetel et al. (2009). Consistent in all studies is the observation of complex three-dimensional flow structures and flow instabilities for higher physiological Reynolds numbers and steady flow conditions.

While the significance of WSS as an important haemodynamic indicator is undisputed, it is a notoriously difficult quantity to determine experimentally (Poelma et al., 2008). Estimating WSS from PIV data is difficult, particularly in complex geometries such as in the carotid artery. In principle, the field of velocity gradients can be obtained by discrete or analytical differentiation of the measured velocity field (Foucaut and Stanislas, 2002). However, imaging errors (optical distortions, light reflections and low tracer density near the wall) and systematic errors (peak fitting algorithm, image interpolation, etc.) can cause signal truncation and greatly affect the performance of conventional PIV near interfaces (Theunissen et al., 2008). These errors are further compounded when computing velocity derivatives and even for well-executed PIV experiments this can lead to substantial uncertainties in the velocity derivatives (Luff et al., 1999).

Therefore, a major focus of this study is the application of our previously developed iPIV technique (Buchmann et al., 2008) to an anatomically realistic geometry with physiological boundary conditions (i.e., inlet waveform). The geometry includes a non-uniform arterial wall and complex vessel curvature, and it will be interesting to observe how this will affect the WSS distribution and prevailing flow field. There is also a strong correlation between endothelial dysfunction and temporally oscillating shear stress (Ku et al., 1985). Therefore, it will be interesting to observe any differences between steady-state WSS and time-averaged WSS. Furthermore, WSS transients will be investigated to yield insight into the oscillatory nature of the prevailing haemodynamics.

2. Experimental Methodology

2.1 Flow Geometry and Boundary Conditions

The geometry of the carotid model is created from in-vivo MRI angiogram data of the left common (CCA,) internal (ICA) and external (ECA) carotid arteries of a healthy male volunteer and is shown in Figure 1. The geometry exhibits some interesting variations, such as asymmetry secondary curvature or non-planarity. The surface of the model exhibits true in-vivo characteristics. The wall includes topological variations, which lead to differences in cross-sectional area and asymmetry along the vessel axes.

The vessel has a nominal common carotid artery diameter of $D = 7.2\, mm$ and a bifurcation angle of approximately $40^\circ$. The geometry is scaled to 2.8 times life size and reproduced in a transparent silicone flow phantom. A more detailed description of the geometric reconstruction of the MRI data set and manufacturing of the silicone phantom can be found in Buchmann et al., 2009.
2.2. Flow Facility

A schematic of the flow facility is shown in Figure 2, which consists of a steady flow loop and a computer controlled piston pump to provide oscillatory flow. The computer controlled piston pump consists of a linear pneumatic actuator controlled by a proportional directional valve and connected to a hydraulic cylinder. To ensure accurate and repeatable motion of the hydraulic piston, a linear encoder and an electromagnetic flow meter at the test section entrance provides feedback control. Real time closed loop control of the piston pump is obtained with a LabView (National Instruments) motion controller. The piston stroke and the cycle frequency can be adjusted independently to obtain a wide range of physiologically realist waveforms with a maximum frequency up to 1Hz at 250mm stroke.

The cardiac waveform is illustrated in Figure 3 and is measured by Phase contrast MRI and the oscillatory flow component is obtained via Fourier decomposition. Once the unsteady component \( q(t) \) of the waveform is found, the inlet profile is obtained by superimposing the steady and oscillatory components to \( Q(t) = q_0 + q(t) \). The resulting waveform at the inlet of the bifurcation model is compared to the measure in-vivo waveform in Figure 3. Some differences occur during early flow deceleration and at late diastole. Overall however, the match between the two waveforms is satisfactory and the developed flow circuitry provides accurate and repeatable inflow conditions.

![Fig. 1. Reconstructed carotid artery geometry comprising the common (CCA), internal (ICA) and external (ECA) carotid artery: (a) side view; (b) front view.](image)

![Fig. 2. Schematic representation of the experimental facility showing the hydraulic flow circuit and optical setup.](image)
The dimensionless parameters describing the pulsatile flow are the Reynolds number $Re$ and the Womersley number $\alpha$ and are defined as follows:

$$Re = \frac{U_m D}{v}; \quad \alpha = \frac{D}{2} \sqrt{\frac{\omega}{v}}$$

(1)

where $U_m$ is the time-averaged mean velocity at the model inlet. For the present cardiac waveform, mean and peak Reynolds numbers are 540 and 920, and $\alpha = 4.54$, translating to an in-vitro time period of approximately 3s and a peak flow of 8.7l/min. The division of flow between the internal and external branch varies over the cardiac cycle, with a division of approximately 5:5 (ICA:ECA) during systole and 8:2 during early diastole. Overall, the cycle averaged flow division accounts to approximately 7:3 (ICA:ECA) and is consistent with previous studies (Ku et al., 1987).

The model inlets and outlets are connected to straight pipes of length $L = 75D$ to ensure fully developed flow at the model entrance and exit. The facility is operated with a refractive index matched working liquid composed of 39% water and 61% glycerin ($v = 10.2 \cdot 10^{-6} m^2 s^{-1}$, $\rho = 1.15 g/cm^3$ at 20°C). Depending on the temperature/viscosity of the working liquid, the maximum achievable Reynolds and Womersley numbers are approximately 2500 and 8.3, respectively.

![Fig. 3. Pulsatile in-vitro and in-vivo inlet waveform](image)

2.3. PIV measurements

The flow field and WSS are investigated with phase-locked PIV measurements. The planar PIV system consists of a digital CCD camera (Kodak Megaplus 1.0) equipped with a 55mm macro lens (Micro Nikkor Nikon) and a 120mJ dual-cavity Nd:YAG laser (New Wavee, Solo PIV 120) for illumination. Further details on the optical setup and image acquisition system are given in Buchmann and Jermy (2008). The PIV recording system is synchronized to the piston stroke via the real-time controller and consecutive image pairs are acquired at a rate of 14Hz or 43 image pairs per cycle. To calculate time and phase averages, a total of 100 waveforms are recorded.

The flow is seeded with hollow glass spheres of nominal diameter, $dp = 10\mu m$ and density $\rho = 1.1g/cm^3$. The ability of the tracer particles to follow the oscillating fluid motion is characterized by the particle response time (Raffel et al., 1998), which for the current conditions is approximately 0.5$\mu s$ and is significantly smaller than typical frequency of the mean flow (i.e., $> 1Hz$).

The velocity fields and WSS are measured in the median plane of the bifurcation and along the inner and outer walls. The recorded data are pre-processed to remove background intensities and image noise and are subsequently analysed with an adaptive multigrid cross-correlation routine.
(Buchmann and Jermy, 2008) to obtain the time and phase averaged velocity fields. The images are evaluated with an initial interrogation window of 64 pixel and two refinement steps with 50% window overlap. Wall shear stress is measured with the iPIV technique and explained in the next Section.

3. WSS measurements

Interfacial PIV (iPIV) is a modified form of the widely known Particle Image Distortion (PID) method to directly measure the velocity gradients at a fixed wall. Assuming the fluid-wall interface to be horizontal, (camera pixels are aligned with the wall) the basic method is to perform 1D cross-correlation on each line of pixels within an interrogation window. The result of this operation yields the wall-normal profile of horizontal velocity at each point from where the velocity gradient can be derived (Fig. 5). In the case of curved walls, conformal transformation (Nguyen et al., 2010) can be used to map the near wall region of the original image to a rectangular region in the transformed space ($\xi, \eta$) shown in Figure 5 (a-b). The particle image intensities are interpolated using the two-dimensional cardinal function interpolation formula (Scarano, 2002) and re-sampled onto a finer grid to provide sub-pixel accuracy.

The interpolated images are divided into interrogation windows of size $M \times N$ (32 x 80 pixel$^2$) and 1D cross-correlation is performed at each, horizontal line as shown in Figure 5 (c-d). The resulting correlation functions are stacked to produce a correlation map of horizontal displacement (wall parallel) and vertical distance (wall-normal). Peaks occur for strong tracer signals and lie at a horizontal position that corresponds to the tracer displacement (Fig. 5 (d)). The signal strength can be improved by ensemble averaging of the correlation stack, where ideally one peak exists at each vertical position, thus forming a ridge that lies along the velocity profile, (Fig.5 (e)).

Assuming no-slip, $U(0) = 0$, the velocity gradient at the wall is extracted by fitting a straight line through the correlation peaks as shown in Figure 5(d). The fit is obtained by ‘sweeping’ the line $R$ through the correlation map and computing the Gaussian weighted sum of correlation values to:

$$F(\text{grad}(y)) = \left[ \sum_{y=-N}^{N} R_{U,y} \cdot \Omega(y) \right] \left[ \sum_{y=-N}^{N} \Omega(y) \right]$$

where the correlation map is reflected around $(U = 0, y = 0)$ to impose the no-slip condition. The Gaussian weighting function $\Omega(y)$ is centred at the wall normal position, $n$ and given as:

$$\Omega(y) = \exp \left[ -\frac{(y-n)^2}{2\sigma^2} \right]$$

Weighting the correlation peaks as a function of wall normal distance reduces the measurement uncertainty and increase robustness by preventing detection of spurious or strong peaks further away. Subsequently, the velocity gradient at the wall $\partial u/\partial y$ is found as the slope of the line for which the functional $F(\text{grad}(y))$ is maximal. Hence, the velocity gradient at the wall is given as:

$$\left. \left( \frac{\partial u}{\partial y} \right) \right|_{y=0} = \max \{ F(\text{grad}(0)) \}$$

A more detailed description of the current iPIV methodology under various experimental conditions is available in Nguyen et al. (2010) and Buchmann et al. (2008). Lastly, Figure 6 shows the iPIV
measurement uncertainty estimated from synthetic particle image simulations for different sized interrogation windows and weighting parameters $\sigma$. Similar to conventional 2D cross-correlation PIV, the measurement uncertainty increases for smaller interrogation regions and increasing shear rate. Overall, the measurements uncertainty is in the order of 0.01 pixel/pixel for shear rates less than unity. Compared to the conventional PIV method with subsequent differential gradient estimation, iPIV provides a direct estimate of the WSR, which is independent of the spatial resolution and measurement noise in the velocity field.

Fig. 5. Principle of interfacial PIV (iPIV): (a) re-sampled near-wall image in transformed space ($\xi$, $\eta$); (b-c) interrogation windows/lines for the 1st and 2nd exposure; (d) instantaneous correlation map composed from individual 1D cross-correlation functions. Note, peaks lie on the same position as the displacement profile (solid line); (e) ensemble averaged correlation map and gradient estimation.

Fig. 6. iPIV measurement uncertainty assessed by means of synthetic particle images of a parabolic velocity profile: (a) Bias error; (b) RMS error for different Gaussian weighting parameters $\sigma$ and linear interrogation size $M$. 
4. Results and Discussion

4.1. Time Average

The time averaged wall shear stress (TAWSS) over one cardiac cycle of period $T$ is defined as:

$$\text{TAWSS} = \frac{1}{T} \int_{0}^{T} \tau_w dt$$  \hspace{1cm} (3)$$

where $\tau_w$ is the instantaneous WSS vector calculated from (4) as $\tau_w = \mu \cdot \partial u / \partial y$. An important time dependent WSS index is the oscillatory shear index (OSI) originally proposed by Ku et al. (1985). This quantity provides fundamental information about the oscillatory nature of the prevailing haemodynamics and assesses the variation of the instantaneous WSS with the time averaged WSS as follows:

$$\text{OSI} = \frac{1}{2} \left(1 - \frac{\tau_{\text{mean}}}{\tau_{\text{mag}}}ight)$$  \hspace{1cm} (3)$$

where $\tau_{\text{mean}} = 1 / T \cdot \int_{0}^{T} \tau_w dt$ is the magnitude of TAWSS and $\tau_{\text{mag}} = 1 / T \cdot \int_{0}^{T} |\tau_w| dt$ the temporal mean of the WSS magnitude. From the above definition, the OSI can vary between 0 and 0.5, where 0 corresponds to unidirectional flow and 0.5 indicates purely oscillatory flow.

Time averaged WSS along the cut plane of the bifurcation outer walls is shown in Figure 7 and 8 and compared to steady flow WSS. The observed spatial variations in TAWSS along the outer walls of the two daughter branches are consistent with the observed flow patterns in this region (see Buchmann et al., 2009 for more details). The sharp decrease in WSS at the proximal location corresponds to flow separation and the small spatial variations seen in both distributions are due to local curvature variations. Overall, the shear environment in the ECA is considerably lower than in the ICA and is also dependent on Reynolds number and flow division.

A good match between TAWSS and steady WSS is obtained with some differences in the low WSS region. The lower WSS under steady flow conditions is due to a standing recirculation region, which in unsteady flow is reduced during the systolic flow acceleration. The variations in instantaneous wall shear is further analysed by the OSI, which spikes in regions of low TAWSS. In the ICA, the primary contribution to this OSI is a brief period of flow separation induced by the adverse pressure gradient during the systolic deceleration phase. However, OSI is very low, indicating almost purely uni-directional flow along the outer sinus wall. In the ECA, WSS exhibits strong temporal variations (see later) and is highly oscillatory with OSI $\approx$ 0.42. Maximum OSI occurs at flow separation and reattachment, while within the recirculation region, OSI and TAWSS are low.

4.2. Transient

Figure 9 details phase averaged axial velocity profiles sampled at discrete time steps in the systolic and diastolic flow phase. The velocity profiles are skewed towards the flow divider wall and exhibit a low momentum and partially reversed flow region along the outer walls. Reduced axial flow exists during the diastolic phase with the profile skewing towards the inner wall. A completed description of the flow field is available in Buchmann et al. (2009).

Figure 10(a-c) presents transient WSS profiles at spatial locations indicated in Figure 10(c). Location A, B and C progress along the outer ECA wall in the separated flow region, whilst H is
located at the inner wall where OSI is high. In the ICA branch, location F and G correspond to high shear regions, whilst D and E progress through the low momentum region along the outer wall. Evidently, strong temporal variations in WSS exist, which are very location specific. For example, in the CCA and distal ICA (F,G), WSS follows the inlet waveform pattern closely with a sharp rise at systole and a decrease during diastole.

In the ECA at location A (bifurcation), WSS is low and close to zero throughout the cardiac cycle. At peak systole (t/T = 0.21), WSS drops rapidly due to the forward acceleration and the movement of the separation point upstream. Strong reversed axial flow is experienced throughout the cardiac cycle at the proximal location (B) and shear stress is lowest amongst the observed locations, reaching values of approximately -1.4 Pa during late systole. WSS in the separated flow region exhibits significant transients. Along the inner wall (location H), a short period of flow separation exists at late systole. This is caused by the adverse pressure gradient during flow deceleration and the interaction between the axial and secondary flows.

As opposed to the outer ECA wall, transient WSS and flow characteristics in the carotid sinus are less pronounced. WSS in the carotid sinus (location D and E) is low throughout the cardiac cycle with no observable flow separation. Flow in this region is predominantly uni-directional and variations in WSS follow the inlet waveform characteristics. The low momentum region near the outer wall is regularly 'washed out' due to strong secondary flows in the systolic flow phase.
Referring to Figure 10, WSS sampled at other locations exhibit similar characteristics. The results show that transient characteristics are significant in regions of flow separation such as in the ECA. These transients are caused by the growing, shrinking and directional change of the recirculation...
zone. Figure 11 depicts the distribution of WSS along the outer walls throughout the cardiac waveform. Regions of low WSS are smallest at peak systole and increase towards the end of diastole (white contour line). The most significant transients occur along the proximal and distal outer external artery and in the distal internal carotid branch.

![Graphs](image1.png)

**Fig. 10.** WSS transients: (a) external carotid artery; (b) internal carotid artery; (c) common and external carotid artery; (d) sampling locations on the bifurcation walls

5. Conclusion

The present study has highlighted that anatomically realistic models with physiological boundary conditions are important in the modelling of carotid artery haemodynamics. The branching of the artery leads to locations of spatial and temporal varying WSS, which is driven by secondary flows and vortex rotation due to secondary vessel curvature and variations in arterial caliber. Significant flow separation occurs in the external artery, while in the internal artery branch the flow remains mostly attached and fairly uni-directional under both, steady and pulsatile flow conditions. The coupling of the haemodynamic response to the arterial geometry and flow conditions support the idea of a geometric “risk factor” in atherosclerosis formation and consequently imply the necessity for more physiologically realistic experiments.

The novelty of this work is the application of the interfacial PIV technique to capture the instantaneous WSS distribution along the bifurcation sidewalls at a high spatial resolution (i.e., 1
pixel or 55µm). The iPIV technique has a measurement uncertainty of less than 0.1 pixel/pixel (or 0.014Pa) for gradients less than unity, and performs superiorly compared with other WSS measurements techniques as discussed by Buchmann et al. (2008).

![Figure 10](image1)

**Fig. 10.** Time variation in WSS plotted along the outer wall of (a) the internal carotid artery and (b) the external carotid artery. White line indicates contours of WSS ≤ 0.15Pa. Data recorded every 0.07sec (dt/T = 0.023)

**References**


Friedman, M. H. (1993). Arteriosclerosis research using vascular flow models: from 2-d branches to


