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DIRECT NUMERICAL SIMULATION OF TRANSITIONAL FLOW IN A STENOSED CAROTID BIFURCATION

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Abstract

The blood flow dynamics of a stenosed, subject-specific, carotid bifurcation were numerically simulated using the spectral element method. Pulsatile inlet conditions were based on *in vivo* color Doppler ultrasound measurements of blood velocity. The results demonstrated the transitional or weakly turbulent state of the blood flow, which featured rapid velocity and pressure fluctuations in the post-stenotic region of the internal carotid artery during systole and laminar flow during diastole. High-frequency vortex shedding was greatest downstream of the stenosis during the deceleration phase of systole. Velocity fluctuations had a frequency within the audible range of 100–300 Hz. Instantaneous wall shear stress within the stenosis was relatively high during systole (~25–45 Pa) compared to that in a healthy carotid. In addition, high spatial gradients of wall shear stress were present due to flow separation on the inner wall. Oscillatory flow reversal and low pressure were observed distal to the stenosis in the internal carotid artery. This study predicts the complex flow field, the turbulence levels and the distribution of the biomechanical stresses present *in vivo* within a stenosed carotid artery.

Keywords

Carotid artery bifurcation; Stenosis; Spectral element method; Hemodynamics; Turbulence; Wall shear stress

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Introduction

Atherothrombotic carotid stenoses, followed by ischemic strokes, are one of the leading causes of mortality and morbidity in western countries (Murray and Lopez, 1997). The presence of a high-grade stenosis, manifested by plaque deposits, can result in transition to turbulence, which may produce audible sounds (*bruit*) discernible by a physician. (At typical hemodynamic Reynolds numbers (Re), blood flow does not exhibit the inertial range of scales associated with fully turbulent flow and might be more accurately described as highly unsteady or chaotic. For brevity, we refer to this state as turbulent or transitional.)

Many studies have been conducted to characterize local hemodynamics and their role on the early development of atherosclerosis. Atherosclerotic prone sites are often localized at bifurcations, junctions and regions of high-curvature, which are also regions of low wall shear stress (WSS) and flow disruption (Caro et al., 1969; Davies et al., 1988; Glagov et al., 1988; Ku et al., 1985; Zarins et al., 1983).

Regions downstream of severe constrictions, post-stenotic regions, can experience a significantly different biomechanical environment than healthy vessels due to turbulence. The WSS within the stenosis is usually high because of flow acceleration due to area reduction. High WSS and the presence of turbulence may damage the endothelial cells (Fry, 1968; Hellums, 1977) and play a role in platelet activation (Ramstack et al., 1979) or plaque rupture (Gertz and Roberts, 1990). In addition, the large spatial WSS gradient associated with turbulence is thought to enhance mass transport into the arterial wall, which may weaken the plaque and make it vulnerable to rupture (DePaola et al., 1992). High temporal shear gradients are shown to stimulate endothelial cell proliferation (White et al., 2001; Ojha, 1994). Moreover, Loree et al. (1991) demonstrated that turbulent pressure fluctuations are elevated by the severity of stenosis and may induce plaque injury.

Laser Doppler anemometry (LDA) measurements on *in vitro* stenosis models have provided insight into the turbulent characteristics in post-stenotic regions (Ahmed and Giddens, 1984; Cassanova and Giddens, 1978; Deshpande and Giddens, 1980; Lieber and Giddens, 1990). Several authors have conducted numerical studies of stenotic flows using turbulence models (Birchall et al., 2006; Ghalichi et al., 1998; Ryval et al., 2004; Stroud et al., 2002; Varghese and Frankel, 2003; Younis and Berger, 2004). Birchall et al. (2006) reported simulations with turbulence models in nine carotid arteries and emphasized the importance of using subject-specific geometries.

Recently, direct numerical simulation (DNS) has been employed in idealized stenosis geometries. The advantage of DNS is that it is particularly well-suited to the transitional regimes where turbulence models can have difficulty. Mittal et al. (2003) simulated pulsatile flow in an idealized stenosis geometry, a planar channel with one-sided semicircular constriction, by using finite difference/spectral-based DNS and large-eddy simulations, with resolution of ~655,000 points. They reported transition to turbulence for Reynolds numbers higher than 1000 with principal pressure and velocity fluctuations driven by the periodic formation of Kelvin-Helmholtz type rollers. Sherwin and Blackburn (2006) examined the instabilities and transition to turbulence of steady and pulsatile flow in an axisymmetric tube with an idealized 75% stenosis using both linear stability analysis and DNS. They used a spectral/spectral-element approach with polynomial order of up to $N = 9$, corresponding to ~2 million points, and observed transition to turbulence under pulsatile conditions at Reynolds number $Re = 535$ and under steady inlet conditions at $Re = 722$. While these idealized geometries differ from the subject-specific geometry presented here, all of the above studies report turbulence-associated complex flow characteristics and demonstrate the level of detail that can be obtained using DNS compared to turbulence models. Varghese et

al. (2007a, 2007b) have used spectral elements to simulate transition under steady and pulsatile conditions in a configuration similar to Sherwin and Blackburn (2006), save that their stenosis featured a 5% eccentricity.

In the present study, we investigate transitional flow characteristics in a subject-specific carotid bifurcation with a stenosis by using the spectral element method (SEM). All scales of fluid motion are resolved and no turbulence model is employed. Validation of the code against laser Doppler anemometry measurements of transitional flow in arteriovenous graft models have been reported in Lee et al. (2007) and Loth et al. (2003). The results of a convergence study for the current geometry are reported in Fischer et al. (2007).

Methods

Images of a stenosed carotid bifurcation in a patient were acquired using a computerized X-ray tomography scanner (high speed CTI, GE Medical Systems, UK) with an axial spacing of 0.5 mm (256×256 pixels). The calculated pixel size was 0.5×0.5 mm and a total of 160 axial images were acquired. As noted in a recent survey by Thomas et al. (2005), aged or diseased carotids can exhibit a variety of deformations and tortuous morphologies that exhibit significant departures from an idealized case. The geometry considered here (Fig. 1) is an example of such a departure. It features a sharp bend at the end of the stenosis and significant non-coplanarity. The vessel diameters are: common carotid artery (CCA), 0.77 cm; external carotid artery (ECA), 0.29 cm; internal carotid artery (ICA), 0.46 cm; and ICA stenosis, 0.34 cm. Based on the ICA outlet, the stenosis corresponds to a 45% area reduction. To generate the numerical model, the images were processed to manually remove small branching vessels and the lumen geometry was segmented in order to obtain cross-sectional contours. Image pixelation effects in the contours were removed with a non-shrinking smoothing algorithm. An automated meshing procedure (Lee et al., 2002; Verma et al., 2005) was used to generate a high-quality mesh with balanced resolution in each branch and minimal deformation for each of the $K = 2544$ hexahedral elements. Surface curvature was represented by isoparametric mappings derived from C^1 bicubic patches on the vessel surface. (The mesh is available on www.biofluids.net/BioMesh.)

Color Doppler ultrasound (Acuson 129XP/10, Siemens, Germany) was used to obtain the pulsatile velocity in the carotid bifurcation. A velocity time trace was measured in the CCA, ECA and ICA by positioning the Doppler sample volume on the center of each vessel. The mean centerline velocity during the cardiac cycle was measured to be ~25cm/s, which is slightly lower than values reported by Holdsworth et al. (1999). The blood density and dynamic viscosity were taken to be $\rho = 1.05 \text{ g/cm}^3$ and $\mu = 0.035 \text{ g/cm-s}$, respectively. The Reynolds numbers $Re_{CCA} = 4\rho Q/\pi\mu D$ based on the CCA inlet diameter D were approximately 266 and 911, for the mean and peak flow rates Q , respectively. The local Reynolds numbers in the ICA stenosis $Re_{stenosis} = 4\rho(0.59Q)/\pi\mu D_{stenosis}$ were 355 and 1217 for the mean and peak flow rates, respectively. The flow waveform used for the inlet flow boundary condition of the CCA is shown in Fig. 2. The heart rate was 80 beats/min, corresponding to a period of $T = 0.75$ seconds for one cardiac cycle. The corresponding Womersley number ($0.5D\sqrt{2\rho/\mu T}$) was 5.96.

The three-dimensional, unsteady, incompressible Navier-Stokes equations were solved with the assumptions of rigid vessel walls and constant viscosity (Newtonian fluid). The boundary conditions imposed at the CCA inlet were Womersley profiles based on the flowrate obtained from the ultrasound measurement. Stress-free boundary conditions were imposed on both ECA and ICA outlets. The flow division was based on the time averaged flow rate through ICA and ECA. The ICA flow rate was obtained from the ECA and CCA flow rate and mass conservation since measurements within the ICA were difficult due to

turbulence-induced spectral broadening. Because the relative phase of the flow waveforms was not available, a constant flow division between the ICA and ECA (59:41) was assumed. The division was enforced by adjusting the flow rates at the outlets with auxiliary unsteady Stokes solutions at each time step. Further details of the boundary condition treatment are given in Fischer et al. (2007) and Lee et al. (2007).

The Navier-Stokes equations are discretized using the SEM, which was introduced by Patera (1984). Essentially, a high-order finite element method, the SEM derives from a decomposition of the computational domain into K hexahedral (curvilinear brick) elements coupled with local function representations based on stable tensor-product Lagrange polynomials of degree N , to yield $n \sim KN^3$ gridpoints. As with global spectral methods, the use of tensor-product forms in the SEM allows for matrix-free operator evaluations that require only $O(Nn)$ work and $O(n)$ storage. With typical expansion orders of $N = 5-11$, the SEM contrasts sharply with the p-type finite element method, which has $O(N^3n)$ work and storage complexities and is thus practically limited to $N < \sim 5$. The principal attraction of the SEM is that the numerical truncation error decays exponentially with N . The rapid convergence and ability to handle relatively complex domains makes the SEM well suited for the simulation of transitional and weakly turbulent flows where, because of the small physical dissipation, small scale structures persist over relatively long distances and times and can thus accumulate uncontrolled phase errors. As with any numerical method, one must resolve the structures and boundary layers in the flow, which sets a lower bound on n . Once the relevant dynamical structures are resolved, the high-order approximations rapidly reduce the numerical error and are manifestly effective in marginally resolved cases.

Our spectral element code, described in Fischer et al. (2007), is highly parallel, employs high-order stabilizing filters and state-of-the art multilevel solvers. It has been validated by detailed comparisons with laser Doppler anemometry measurements for transitional flow in arteriovenous graft geometries (Lee et al., 2007; Loth et al., 2003). For the current geometry, convergence tests were conducted at peak systolic flow rates with polynomial orders $N = 7, 9$, and 11 (respectively, $\sim 888,000$, $\sim 1,854,000$, and $\sim 3,386,000$ grid points). The results of the study (Fischer et al., 2007) indicated grid convergence in the ICA mean and r.m.s. velocity distributions at $N = 9$, which is the value used in the current study. Time integration was based on a third-order semi-implicit scheme with time step $dt = 10^{-5}$ seconds chosen to satisfy the Courant stability criterion. Once the relevant dynamical structures are resolved, the rapid error reduction attained with high-order methods makes them particularly effective in marginally resolved situations.

Ten cardiac cycles were computed, with each cycle requiring approximately 20 hours of CPU time on 256 processors of the TSC1 platform at Pittsburgh Supercomputing Center. The last six of the cycles were used to obtain the ensemble-averaged velocity field at 50 time points during the cardiac cycle:

$$\langle u(x, t^i) \rangle := \frac{1}{6} \frac{50}{T} \sum_{m=5}^{10} \int_{t^{i-1}}^{t^i} u(x, t+mT) dt,$$

where $t^i := i \times T/50$, $i = 0, \dots, 50$. In addition, we define the velocity fluctuation, $\mathbf{u}' := \mathbf{u} - \langle \mathbf{u} \rangle$,

and the square-root of turbulent kinetic energy (STKE), $k^{1/2} := \sqrt{\langle \mathbf{u}' \cdot \mathbf{u}' \rangle / 2}$.

Results

The flow remains laminar in the CCA and ECA, but transitions to turbulence within the ICA during the systolic phase. A typical flow structure near peak systole is indicated in Fig. 3, which shows coherent structures identified by the λ_2 criterion of Jeong and Hussain (1995). The CCA exhibits a strong central vortex that changes in strength throughout the cardiac cycle but does not change in topology. Flow in the ICA is more complex, with an axially-aligned vortex pair threading through the stenosis and significant vortex break-up in the post-stenotic region. The resultant small scale structures are rapidly advected during systole and give rise to rapid temporal as well as spatial variation in local hemodynamic stresses.

The ensemble-averaged velocity profiles at the mid-plane of the bifurcation are shown in Fig. 4 at four different time points corresponding to: near peak systole, mid-deceleration, near the second systolic peak, and diastole (see time points in Fig. 2). Many of the velocity profiles are skewed towards one wall within the ICA and ECA due to significant curvature. The flow in the CCA retains a Womersley profile until the split between the ICA and ECA. The ICA is relatively straight in the stenosis and then turns sharply inward causing velocity profiles to be skewed toward the outer wall. Throughout the cardiac cycle, the ICA flow separates just downstream of the apex. Farther downstream, the flow separates on both the inner and outer walls, just distal of the stenosis. Strong Dean flow patterns are observed near the peak systole and mid-deceleration phases, as seen in the ensemble-averaged streamwise velocity distributions and instantaneous in-plane distributions of Figs. 5 and 6, respectively. The patterns are, however, asymmetric and rather complex due to the lumen irregularity. The transverse velocity downstream of the stenosis (B-B and D-D) is significant—as high as one-third of the streamwise velocity.

The temporal variation of the flow is illustrated in Fig. 7 by contours of instantaneous transverse vorticity, ω_y , in the mid-plane surface (passing through the branch centerlines). The flow separates from the apex, forming a long shear layer that does not reattach to the inner wall. This shear layer roughly defines a boundary that separates a slow flow region (left of the shear layer) and high-speed jet associated with the Dean's flow (right of the shear layer). At near peak systole and mid-deceleration (Fig. 7a and 7b), the shear layer becomes unstable, rolls up and the jet eventually breaks down. The ICA flow becomes nearly laminar prior to the second systolic peak (not shown), and then disturbed to a smaller degree during the second systolic peak (Fig. 7c).

Fig. 8 shows the STKE distribution at the bifurcation mid-plane. The STKE is greatest in the post-stenotic region and peaks during systolic deceleration. The apex- and stenosis-induced shear layers combine to form a thicker shear layer during systolic acceleration (Fig. 8a). These two layers interact more strongly during mid-deceleration (Fig. 8b). The turbulence is greatly reduced after mid-deceleration but does persist until the diastolic phase begins. The distribution of the Reynolds shear stress (RSS) at various cross-sections is shown in Fig. 9. The RSS is relatively small, though nonzero, at the stenosis exit (A-A). Moving through the post-stenotic region, the RSS distribution progresses from the vessel interior outward towards the walls, clearly indicating that the turbulence observed in this flow is not wall generated but is instead generated by the separation/shear layer phenomena described above.

The ensemble-averaged pressure distribution on the wall (Fig. 10) shows generally low pressure downstream of the stenosis and the lowest pressure at the distal end of stenosis. Regions of locally low pressure were located on the inner wall of curvature similar to that for Dean-flow. The ensemble-averaged WSS distribution (Fig. 11) shows high shear stress within stenosis and outer wall of ICA, corresponding to high velocity-gradient with skewed velocity profile. During peak systole, the instantaneous WSS values within the stenosis and

at the outer wall where the jet impinges are 25–45 and 35–70 Pa, respectively. Recirculation zones are characterized by patches of low WSS in the ICA, where the largest regions are associated with the apex-induced separation and the stenosis-induced separation along the inner wall.

Detailed temporal characteristics are depicted in the time traces of the axial component of velocity (Fig. 12) for specific locations within the ICA (a–f, see Fig. 1 for locations), where the onset of transitional flow fluctuations can roughly be determined. Flow disturbances with random fluctuations are observed downstream of the stenosis at locations (b)–(f) with the largest fluctuations located at the most distal point (f). Fluctuations were only present during systolic phase. Note that the magnitude of the velocity in the post-stenotic time traces (b–f) is significantly smaller than that at (a) due to the skewed velocity profile, which shifts the peak away from the centerline where the trace data were collected. As one moves distal from the stenosis, there is a general increase in the centerline velocities deriving from the jet break-up and entrainment. The frequency content of the time traces is quantified in Fig. 13, which shows distinct peaks at 60, 120, 160, 200, and 300 Hz at various positions (c)–(f). The shift of the peaks in moving from (e) to (f) is primarily due to the increased centerline velocity at (f), as the alternative explanation of a broadening of the range spatial scales is not indicated (e.g., Figs. 3 and 7).

Discussion

The present numerical study examines the detailed flow field within a subject-specific carotid bifurcation with a stenosis. The flow is weakly turbulent inside the ICA during the systolic phase and laminar during diastole while it remains laminar within the CCA and ECA throughout the cardiac cycle. Transition to turbulence with high velocity fluctuations is most prominent during the deceleration phase (Figs. 7–9), which is similar to the observation in transient pipe flow by He and Jackson (2000). This elevated disturbance in the decelerating phase was also observed by Long et al. (2001). The jet flow exiting the stenosis and impinging on the outer wall distal to the ICA (Fig. 4) with high WSS (Fig. 11) is characteristic of flow in severe stenosis and has been previously observed (Birchall et al., 2006; Long et al., 2001).

Flow downstream of the stenosis was not a pure periodic oscillation as reported by Plett et al. (2001), but more complex and chaotic (Figs. 7, 8, and 12). Significant turbulence was developed in the separation region about $0.5D$ downstream of the stenosis and the highest turbulence intensity was present between $1D$ and $2D$ downstream of stenosis. The fluctuations in this flow were not restricted to the post-stenotic region, but were also (weakly) present within the stenosis (Fig. 9) near the peak Reynolds number, $Re_{\text{stenosis}} = 1217$. The fluctuations in RSS correlated with the location of the vortex pair emanating from the apex of the bifurcation (Fig. 3). The peak RSS was associated with flow separation in the post-stenotic region, rather than resulting from shear at the vessel walls.

High temporal and spatial wall shear stress gradients were observed not only in post-stenotic region, but also within the stenosis. While high WSS is generally expected within the stenosis due to the marked rise in velocity, flow separation and low WSS were identified at the inner wall of stenosis, resulting in high spatial variability of WSS magnitude on the throat of stenosis. Such variability has been implicated in plaque weakening through enhanced mass transport (DePaola et al., 1992).

The smallest (Kolmogorov) scale of eddies for fully developed isotropic turbulence can be estimated using the relationship of $\eta = (\nu^3/\varepsilon)^{1/4}$. Here, ν and ε are kinematic viscosity and turbulent dissipation, respectively. This relationship, however, can still provide rough idea

of the smallest eddy scale of the post-stenotic flow. Turbulent dissipation can be further approximated by $\varepsilon \sim (2k/3)^{3/2}/l$, where l is the characteristic length of the largest turbulent eddy (the radius of distal ICA was considered). Papoutsakis (1991) found that the turbulent eddies may directly interact with blood elements through energy transfer if their scale is comparable to blood cells. The Kolmogorov length scale of the present simulation was $\sim 67\mu\text{m}$ during deceleration phase, which is an order of magnitude greater than the typical length scale of an erythrocyte ($\sim 7\mu\text{m}$). On the other hand, the maximum turbulent Reynolds shear stress was $\sim 29\text{ Pa}$ which is greater than the value ($10\text{--}16.5\text{ Pa}$) where platelet damage occurs (Hung et al., 1976; Williams, 1974).

Instantaneous peak-to-peak pressure fluctuation of $\sim 266\text{ Pa}$ (2 mmHg) was observed at approximately $2D$ downstream of the stenosis during systolic deceleration. The largest pressure drop, $\sim 1300\text{ Pa}$ (10 mmHg), between the proximal and distal ends of the stenosis was observed at peak systole. The variation of pressure drop in pulsatile flow produces oscillating compressive stress on the deposited plaque and may increase a risk of plaques rupture. Perktold and Rappitsch (1995) showed that the overall flow characteristics within a healthy carotid did not change significantly with a compliant wall model. Although the presence of the plaque may greatly reduce wall compliance (Cavalcanti, 1995), evidence show that the wall compliance can considerably alter the post-stenotic flow field and subject the stenotic region under significant compressive stresses (Bathe and Kamm, 1999; Tang et al., 2001, Li et al.). Further work will be needed to understand the effect of wall compliance on the flow field in subject-specific, severely diseased carotid as the irregular 3D shape may heighten the effects of wall compliance.

The present simulation has been carried out on the Newtonian assumption. Gijssen et al. (1999) and Chen and Lu (2004) have reported significant difference when a non-Newtonian model was employed in steady bifurcation model. In contrast, Perktold et al. (1991) and Lee and Steinman (2007) showed only minor influence of shear thinning fluid compared to the Newtonian flow.

In conclusion, the present work describes transitional flow patterns within a patient-specific stenosed carotid artery and the resultant biomechanical environment to which the artery walls are exposed. The simulation demonstrated several complex flow features (e.g., vortices in the stenosis interior, strong Dean's flow, and shear-layer destabilization) associated with the vessel geometry. The transition to a weakly turbulent state was marked by rapid velocity and pressure fluctuations in the post-stenotic region of the internal carotid artery. Further research on patients may help elucidate the role of carotid hemodynamics on plaque vulnerability. Once such relationships are understood, hemodynamic simulations might provide clinically relevant information for risk assessment and surgical planning.

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Fig. 1. Spectral element mesh with $K = 2544$ elements based on CT scan images of *in vivo* stenosed carotid. Insets figure denote the plotting location for velocity time traces (a)-(f) (Fig. 12) and cross-section slices for spatial velocity variation A-A, B-B, D-D and F-F (Figs. 5, 6 and 9).

Fig. 2. Flow waveform used for inlet flow boundary condition (CCA) based on color Doppler ultrasound measurements *in vivo*.

Fig. 3. Coherent vortical structures within ICA at systolic peak identified by the λ_2 criterion of Jeong and Hussain (1995).

Fig. 4. Ensemble-averaged velocity vectors plots on the mid-plane of the bifurcation at various flow phases (a) near peak systole, (b) mid-deceleration phase, (c) near second systolic peak and (d) diastole. See Fig. 2 for time points.

Fig. 5. Ensemble-averaged streamwise velocity $\langle u_s \rangle$ distribution at four cross-sections (i) near peak systole and (ii) mid-deceleration phase. See Figs. 1 and 2 for cross-section locations and time points, respectively.

Fig. 6. Instantaneous in-plane velocity vectors at four cross-sections (i) near peak systole and (ii) mid-deceleration phase. See Figs. 1 and 2 for cross-section locations and time points, respectively.

Fig. 7.

Instantaneous transverse vorticity ω_y contour plots on the mid-plane of the bifurcation at various flow phases: (a) near peak systole, (b) mid-deceleration phase, (c) near second systolic peak and (d) diastole ($\omega_y = -2000, -1000, -500, 0, 250, 500, 1000, -500, 0, 250, 500, 2000 \text{ s}^{-1}$). See Fig. 2 for time points.

Fig. 8. Mid-plane distribution of $k^{1/2}$: (a) near peak systole, (b) mid-deceleration phase, (c) near second systolic peak and (d) diastole. See Fig. 2 for time points.

Fig. 9. Reynolds shear stress $\langle u_s' u_t' \rangle$ at various flow phases: (i) near peak systole, (ii) mid-deceleration phase, and (iii) near second systolic peak at four cross-section locations. See Figs. 1 and 2 for cross-section locations and time points, respectively.

Fig. 10. Ensemble-averaged pressure distribution at various flow phases: (a) near peak systole, (b) mid-deceleration phase, (c) near second systolic peak, and (d) diastole. See Fig. 2 for time points.

Fig. 11. Ensemble-averaged WSS distribution at various flow phases: (a) near peak systole, (b) mid-deceleration phase, (c) near second systolic peak, and (d) diastole. See Fig. 2 for time points.

Fig. 12. Time traces of z -component velocity at six streamwise locations (see Fig. 1) for four cardiac cycles. There is an offset in velocity at each location to view each trace more clearly.

Fig. 13.
Frequency spectra of z -component velocity corresponding to the time traces in Fig. 12.