Analysis of blood flow in an out-of-plane CABG model

Meena Sankaranarayanan, Dhanjoo N. Ghista, Chua Leok Poh, Tan Yong Seng, and Ghassan S. Kassab

1School of Mechanical and Aerospace Engineering and 2Bioengineering Division, Nanyang Technological University; 3Department of Cardiothoracic Surgery, National Heart Centre, Singapore; and 4Department of Biomedical Engineering, University of California, Irvine, California

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Atherosclerosis is a major disease of arterial blood vessels. The epicardial coronary arteries are very susceptible to atherosclerosis and plaque formation. The inflammatory disease process causes blockage of blood flow to the heart muscle and is a major cause of mortality and morbidity today. The coronary artery bypass graft (CABG) is a common surgical procedure that reroutes blood flow around a blocked artery through a conduit. According to the American Heart Association, the number of CABG procedures in the United States was more than 600,000 (1.2 million worldwide) in the year 2000, which is twice that of 1986.

Despite CABG being an effective surgical procedure, it has been reported that >50% of the grafts fail because of restenosis caused by arterial disease (6, 25). Although the mechanism of this disease is unclear, there are several possible reasons, including compliance mismatch between the graft and the artery (1, 9, 31), local hemodynamic effects of disturbed flow patterns, magnitude and direction of wall shear stress (WSS) (4), and graft incompatibility (21). Because the prone disease sites are near bends and junctions of graft with the host vessel (14), it is likely that hemodynamic factors such as WSS magnitude and direction play a vital role in atherogenesis.

An accurate assessment of WSS requires a faithful simulation of the velocity field. Numerous studies have determined the flow streamlines in patient-specific models of CABG (11, 15, 24). Despite the insight gained from these studies, an integrative model that takes into account all three elements does not exist: 1) the complete bypass flow domain, 2) measured physiological inlet flow conditions, and 3) nonplanarity of the bypass graft vessel. The objective of the present study is to present such an integrated model.

The present study addresses and ameliorates that the three-dimensional (3D) out-of-plane CABG geometry is an important determinant of the flow velocity and hence WSS. Our results support the view that the nonplanarity of the graft vessel is an important determinant of the average level of WSS and its variability on the bed of the stenosed left anterior descending (LAD) artery. Hence, a properly contoured (out-of-plane) graft may contribute to improved graft patency.

METHODS

**Geometrical Model**

The model simulation of the flow field of the anastomosis in aorto/left CABG is illustrated in Fig. 1A. The ascending aorta (AB) is considered to have a length of 80 mm with a diameter of 25 mm. The right coronary artery (RCA) (C, depicted in Fig. 1A) has a circular cross section with a 3.2-mm diameter, and the left main coronary artery (LCA; D, appearing diametrically opposite to the RCA) has a circular cross section with a 4-mm diameter. The LAD is assumed to be a straight cylindrical tube with diameter and length of 3.2 and 45 mm, respectively. The proximal portion of the LAD (FG) is assumed to be fully occluded.

The venous graft is simulated as a nonplanar vessel of circular cross section with diameter of 4 mm. It originates from the aorta at E and anastomosis with the LAD at F (Fig. 1A). The intersection between the graft and the LAD artery has an elliptical shape that is caused by the deformation of the larger-diameter graft because of its sutured attachment to the smaller LAD vessel. The overall dimensions of the total aorto/left CABG model are based on surgical observations. Specifically, the mean diameter, length, and angle of several clinical

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The blood is assumed to be an incompressible, Newtonian fluid with a dynamic viscosity ($\mu$) of 0.00408 Pa·s and a density ($\rho$) of 1.050 kg/m$^3$. The blood vessel walls are assumed to be rigid and impermeable. For a 3D flow, the conservation of mass and linear momentum are expressed by the equations of continuity and Navier-Stokes, respectively, as

**Equation of continuity:**

$$\nabla \cdot \vec{q} = 0$$  \hspace{1cm} (1)

**Equation of momentum:**

$$\rho \left( \frac{\partial \vec{q}}{\partial t} + \vec{q} \cdot \nabla \vec{q} \right) = -\nabla p + \mu \nabla^2 \vec{q}$$  \hspace{1cm} (2)

where $p$ denotes pressure and $\vec{q}$ denotes velocity vector in three dimensions. The velocity distributions are obtained as a solution to the governing equations for the appropriate boundary conditions defined below. The flow field is automatically updated during each time interval by adopting measured time-varying input data of the aorta (Fig. 2A), the ascending aorta (Fig. 2B), the LAD artery (Fig. 2C), the left circumflex artery (LCX; Fig. 2D), and the RCA (Fig. 2E). In this study, we have assumed that the total flow in the LCA is the sum of flows in the LAD and LCX (Fig. 2F). Because the LAD has been bypassed, the flow entering the LCX must be equal to the flow in LCA. Hence, in this study, the measured LCX flow waveform (Fig. 2D) has been imposed at the LCA entrance. Once the velocity field is computed, the WSS is computed as the product of viscosity and the radial gradient of the velocity.

**Boundary Conditions**

**Systolic phase.** The systolic flow is obtained by prescribing a blunt velocity profile (along the cross section) at the inlet to the aorta, from the left ventricle (LV). The velocity magnitude is computed from the physiologically measured stroke volume over the ejection period based on the flow wave form (13) as shown in Fig. 2A. During this period, the extravascular myocardial compression of the coronary circulation reduces the blood flow through the coronary arteries significantly.

During systole, the inputs to the model consist of 1) a blunt velocity derived from the time-varying flow-rate waveform ($Q_A$) at the inlet $A$ (shown in Fig. 1A) of the aorta (depicted in Fig. 2A) as adopted from Ref. 13; 2) the calculated time-varying flow-rate waveform ($Q_B$) at the exit $B$ from the ascending aorta (Fig. 2B); 3) the time-varying flow-rate waveform ($Q_A$) at the distal end $H$ (shown in Fig. 1A) of the LAD artery (Fig. 2C), as obtained from the mean velocity waveform obtained from phase contrast MRI scanning (30); 4) the time-varying flow-rate waveform ($Q_D$) (Fig. 2D) at the LCA entrance $D$ (shown in Fig. 1A) is equal to the flow rate in LCX (Fig. 2D), obtained by phase contrast MRI scan (30); and 5) the time-varying flow-rate waveform ($Q_C$) imposed at the entrance $C$ (shown in Fig. 1A) of the RCA (Fig. 2E), as obtained by means of a Doppler flowmeter catheter (22).

**Diastolic phase.** At the start of diastole, there exists a small amount of back flow into the LV through the aortic inlet as depicted in Fig. 2A. During the diastolic phase, the aortic valve remains closed, and it is the back flow from the ascending aorta (Fig. 2B) that perfuses the coronary arteries. Figure 2, C–F, reveals that the majority of the flow enters the LCA, which is in agreement with earlier works (23, 32). It can be noted that the perfusion to the coronary arteries is much greater during diastolic phase as compared with the systolic phase as shown in Fig. 2, A–F. This implies that the coronary vascular resistance in the left coronary circulation is significantly lower than that of the right.

In summary, the input data to the model consists of 1) the calculated blunt velocity profile at the ascending aorta (Fig. 2B) and 2) the flow conditions at the LAD exit and LCA and RCA entrances obtained from time-varying input flow-rate waveforms (Fig. 2, C–E).

**Fluid Dynamics Simulation**

The fluid dynamics simulations are performed by using a control volume-based computational technique, implemented in the computational fluid dynamics (CFD) code Fluent (Fluent User’s Guide, Fluent, Lebanon, NH). The computation procedure of the commercial code consists of several steps: 1) construction of the geometry with the use of a preprocessor, Gambit (Gambit User’s Guide, Fluent); 2) meshing the computational domain; 3) assigning boundary conditions (at A, B, C, D, and H) in terms of velocities and flow-rate weightings, i.e., flow rate normalized with respect to the entrance flow rate; 4) assigning fluid properties (viscosity and density); and 5) prescribing the solution algorithm.
The geometry of the aorto/left CABG model was constructed in Gambit by using the mean dimensions obtained from clinical cases. The elements employed to mesh the computational domain consist primarily of regular structured hexahedral elements as well as wedge elements wherever necessary (such as at proximal and distal junctions of the graft).

To carry out the mesh sensitivity analysis, numerical simulations were carried out by varying the number of grid cells in the computational domain. In the computational domain, the mesh sensitivity on the flow variables (velocity and WSS) was tested by varying the number of grid cells (namely, 65,390; 92,330; 213,324; 388,900; and 509,350). The velocity profile results obtained from different grid cells were displayed along the vertical center line of the graft section (G5) and the LAD artery section (C1) in Fig. 3, A and B. It was found that the computational domain of 388,900 cells was sufficient.

In the solution algorithm used by Fluent, the governing equations (conservation of mass and linear momentum) were solved sequentially. Because the governing equations are nonlinear (and coupled), several iterations of the solution loop were needed before a convergent solution was obtained. With the use of this approach, the resultant algebraic equations for the dependent variables (namely the velocities) in each control volume were solved sequentially by a point implicit (Gauss Seidel) linear equation solver in conjunction with an algebraic multigrid method. The governing equations were solved iteratively until convergence of all flow variables was achieved. The solutions for all the flow variables were deemed to converge once their residuals were below the set convergence criterion of $10^{-5}$. There were no differences in the simulation results when the convergence criterion was set at $10^{-6}$.

**RESULTS**

Simulations were carried out at various time instants of the cardiac cycle. Herein we depict the simulation results at 1) start of ejection ($t = 0.0$ s), 2) mid-ejection ($t = 0.15$ s), 3) early diastole ($t = 0.32$ s), and 4) mid-diastole ($t = 0.57$ s). At these time instants, the flow velocity, secondary flow motion, and WSS distributions were determined.

The axial velocity profiles in the vessels (aorta, graft, and LAD artery) were computed along the vertical and horizontal center lines at all cross sections depicted in Fig. 1B. The in-plane velocity vectors were computed at specific sections of I) the aorta, at A1 and A3 (as shown in Fig. 1B) located at distances of 10 and 40 mm from the aortic inlet, respectively;
2) the graft, at G \textsubscript{1} (close to the proximal anastomosis junction) and G \textsubscript{3} (close to the distal anastomosis junction); and 3) the LAD artery, at C \textsubscript{1} and C \textsubscript{4} located at 20 and 40 mm from the proximal end of the artery GH, respectively.

**Systole Phase**

During systole, blood flows in the forward direction from the LV into the ascending aorta and into the coronary arteries. This is termed as forward flow.

**Start of ejection (t = 0.0 s).** To capture the flow features, the computed axial velocity profiles (in the aorta, graft, and LAD artery) along both the horizontal and vertical center lines (at all cross sections of the flow domain) are illustrated in Fig. 4, A–F. In Fig. 4, A and B, it can be seen that the blunt velocity profile assigned at the aortic inlet slowly changes to a parabolic profile as blood flows downstream in the ascending aorta. In the aorta, the axial velocity distribution along the horizontal center line results in an M-shaped profile because of the influence of the flow entering the RCA. This double-peaked velocity profile gradually changes to a somewhat parabolic profile with increasing distance from the aortic entrance (section A).

It can be noted that as the flow progresses further downstream in the aorta, the effect of the proximal anastomotic junction does not significantly affect the velocity profile because there is only a very small amount of flow entering the graft (Fig. 4A). In Fig. 4B, the computed axial velocity profiles along the vertical line exhibit a slight skewing toward the top wall of the aorta, which is due to the junction effects. In the graft, it is noted (Fig. 4, C and D) that as blood flow progresses from the proximal junction of the graft to the distal end, the axial velocity exhibits a parabolic profile with slight skewing. Figure 4, E and F, depicts the velocity profiles at different sections in the LAD artery.

Figure 4, G–L, depicts the in-plane flow field in terms of the in-plane velocity vectors at specific sections of the vessels mentioned in Geometrical Model. Figure 4, G and H, shows the computed secondary-flow vector fields in the cross-sectional plane of the aorta at A\textsubscript{1} and A\textsubscript{3}. In Fig. 4G, symmetric vortices are observed close to the top wall, whereas the rest of the core flow is directed upward; this may be due to some flow entering into the RCA junction. On section A\textsubscript{3} (after the proximal anastomotic junction), a similar pattern is observed in Fig. 4H with a slightly decreased peak velocity magnitude. In the graft section G\textsubscript{1}, the in-plane velocity vectors (depicted in Fig. 4I) are all directed toward the inner wall. As flow progresses downstream, the flow direction at section G\textsubscript{5} (as depicted in Fig. 4J) changes from the inner to the outer wall of the graft because of the curvature of the vessel. The flow distribution at section C\textsubscript{1} of the LAD artery immediately after the distal anastomotic junction, in Fig. 4K, depicts a small recirculation region at the center of the cross section; herein, the flow is skewed toward the bed of the artery. On reaching the distal end of the LAD artery (section C\textsubscript{4}), the secondary flow weakens and the flow is purely in the axial direction, as seen in Fig. 4L.

The flow velocity distributions result in WSS distributions depicted in Figs. 5, A and B. The flow disturbances due to the junction effects result in steep velocity gradients around the RCA entrance C and the proximal anastomotic junction E. Hence, the maximum WSS (~0.58 Pa) occurs at the proximal anastomotic junction, as depicted in Fig. 5A. Large spatial variation in WSS is also seen in the body of the graft (close to the region where the curvature of graft changes), which may be attributed to the skewing of flow as a result of the curvature effect. The magnitude of WSS around the distal anastomotic junction ranges from 0.30 to 0.41 Pa, whereas the toe experiences a WSS of magnitude 0.27 Pa. As the proximal portion (FG) of the LAD vessel is completely occluded, the flow is stagnant, with negligible WSS. When compared with the distal junction, the floor of the artery is subjected to a low (and uniformly distributed) WSS of magnitude 0.24 Pa (Fig. 5B).

**Mid-ejection (t = 0.15 s).** During midacceleration, the blood enters the aorta with a very high velocity magnitude of ~0.85 m/s (compared to 0.04 m/s at the start of ejection). This instant corresponds to the maximum flow in the ascending aorta during the cardiac cycle. In Fig. 6, A and B, the computed axial velocity profiles at different sections of the aorta exhibit a blunt profile, with a slight variation in the magnitude of peak velocity. As expected, the magnitude of the peak velocity increases as the flow proceeds toward the ascending aorta. The flow in the core region has a blunt profile throughout the flow domain (Fig. 6, A and B), similar to an inviscid flow. In the graft,
Fig. 4. Axial velocity at start of ejection ($t = 0.0$ s). A: axial velocity profiles (m/s) extracted along horizontal center line of all cross sections of aorta. Blunt velocity profile changes to a parabolic profile with increasing distance from aortic inlet. M-shaped velocity profiles seen in $A_1$ and $A_3$ are due to effects of two junctions, namely, RCA and proximal anastomotic junction, respectively. B: axial velocity profiles computed along vertical center line of aorta sections exhibit a slight skewing toward top wall of aorta as a result of junction effects. C: axial velocity profiles in graft sections are parabolic with slight variations seen in velocity magnitude along different sections. D: vertical center line axial velocity profiles in the graft exhibit slight skewing due to effect of curvature of graft vessel. E: axial velocity profiles in different LAD sections are parabolic. F: vertical center line axial velocity plot indicates that blood flows in LAD vessel with a constant velocity except with a slight difference seen on $C_1$ due to effect of distal anastomotic junction. G: in-plane velocity vector components (m/s) in $A_1$ exhibit symmetric vortices close to top wall. H: similar behavior in the in-plane velocity vector pattern is observed in $A_3$. I: vector components are all directed toward inner (I) vessel wall of graft section $G_1$. J: in-plane velocity vector components shift from inner to outer (O) wall as seen in $G_5$ due to curvature effects of graft. K: small recirculation region observed in center of $C_1$ of LAD vessel. L: bicellular patterns of negligible velocity magnitude observed in section $C_4$ reflecting symmetric nature of flow.
significant skewing of the axial velocity profiles is seen at the different sections of the graft (Fig. 6, C and D). The curvature of the graft vessel results in skewing of the flow toward the outer wall. In Fig. 6C, it is seen that as blood flows toward the distal anastomotic junction, the peak velocity gets skewed toward the inner graft wall. The distal junction effects along with the nonplanarity of the graft are reflected in the axial velocity profiles. In the LAD artery, it is observed (Fig. 6, E and F) that, initially, the velocity profile is skewed toward the bed of the artery and becomes more uniform downstream.

Figure 6, G and H, depicts that the in-plane velocity vector components in the sections of the aorta are all directed toward the top wall (caused by the flow diversion into the RCA and the graft). The in-plane velocity vector components in the graft sections G1 and G3 are depicted in Fig. 6, I–J. Figure 6K reveals counterrotating vortices in the LAD artery section C1, with the peak velocity being on the floor of the artery. On reaching the LAD exit, the bicellular secondary flow pattern gradually changes to a single vortex pattern (as seen in section C4) of negligible magnitude as depicted in Fig. 6L; i.e., it tends toward developed flow.

The corresponding WSS distributions at the proximal and distal junctions of the graft are shown in Fig. 7, A and B. The blunt axial velocity profiles result in a uniform WSS magnitude of ~2.16 Pa in the aorta. Sites of peak WSS (6.185 Pa) are at the RCA and graft junctions (Fig. 7A) due to high velocity gradients at these sites. At the distal anastomotic region, the WSS at the toe is ~0.62 Pa. The WSS along the bed of the LAD artery is almost uniformly distributed (at 0.45 Pa), as seen in Fig. 7B.

### Diastole Phase

During diastole, the back flow enters the ascending aorta at B (Fig. 1B) because of the elastic recoil of the aortic wall. The flow profiles are hence depicted with a negative sign to indicate the reverse in the flow direction.

Start of diastole ($t = 0.32$ s). At this point, there is only a small amount of back flow from the ascending aorta into the LV (through the aortic valve). Thereafter, the reverse pressure gradient on the aortic valve leaflets (protruding into the coronary sinus) makes the valve close quickly and allows only a small amount of blood flow back into the LV. A blunt velocity profile is prescribed at the ascending aorta exit B. As the flow progresses from B (shown in Fig. 1B) into the ascending aorta, the axial velocity profile gradually increases in magnitude, as depicted in Fig. 8, A and B.

The velocity profiles in the graft (Fig. 8, C and D) exhibit considerable skewing. The double curvature of the graft vessel results in change in the skewness of the velocity profile from the outer to inner wall as shown in Fig. 8, C and D. Figure 8E shows that the M-shaped velocity profiles at sections C1 and C2 changes to quasi-parabolic profiles at downstream sections C3 and C4. Prominent skewing of the axial velocity profiles toward the floor of the artery is observed along the vertical center line in Fig. 8F.

Because the flow advances in the ascending aorta from B toward the aortic inlet, the in-plane velocity vector components are first displayed at section A3, followed by A1 (between the RCA and the proximal anastomotic junction) in Fig. 8, G and H. On approaching the proximal anastomotic graft junction E, the suction effect is felt on the in-plane velocity vector components, thereby pulling the velocity vectors toward the top wall as seen in Fig. 8G. It is evident from Fig. 8H that the amount of flow entering the LCA is relatively higher than the flow entering the RCA, thereby directing more flow toward the bottom wall. The significant skewing of the axial velocity profiles (Fig. 8, C and D) is reflected in the in-plane velocity vectors shown in Fig. 8, I and J. In the graft, the velocity vectors (Fig. 8I) are directed toward the inner wall and the top portion of the graft vessel. Figure 8J depicts a small region of recirculation in the graft section G5. In the LAD artery sections (C1 and C4), the secondary flow patterns (Fig. 8, K and L) are qualitatively similar to those observed at the earlier time instant ($t = 0.15$ s).

The corresponding WSS distributions are shown in Fig. 9, A and B. Negligible WSS is seen in the aortic domain, with the aorta-graft junction depicting a high WSS of ~5.0 Pa (Fig. 9A). The WSS at the bed of the artery opposite to the distal junction (in Fig. 9B) is relatively high (6.0 Pa) as compared with that at the toe region (~1.5 Pa). This is expected due to the impingement of blood on the floor of the artery.
Fig. 6. Axial velocity at midjection ($t = 0.15$ s). A: axial velocity profiles at different sections of the aorta are all similar in shape to blunt velocity profile assigned at aortic inlet. Blood from LV ejects with a high velocity (0.83 m/s). B: vertical center line plots exhibit a similar profile except with a slight variation in velocity magnitude. Most of flow goes into ascending aorta with a small amount entering RCA, LCA, and graft. C: horizontal center line axial velocity profiles in graft sections exhibit skewing initially toward outer graft wall, whereas downstream, flow gets skewed toward inner graft wall because of the presence of curvature. D: similar pattern is observed in vertical center line profiles except with a change in magnitude of axial velocity in different vessel sections. E: axial velocity plots along horizontal center line of LAD sections depict a parabolic profile. Presence of distal anastomotic junction causes a shift in peak velocity in axial velocity profile in $C_1$. F: axial velocity profiles extracted along vertical center line of LAD sections. G: in-plane velocity vectors on $A_1$ are directed toward top wall. H: magnitude of in-plane velocity vectors on $A_2$ are similar to that seen on $A_1$. I: in-plane velocity vector components in $G_1$ are directed toward inner graft wall. J: there is a change in flow direction on reaching $G_5$. Although amount of flow entering graft is less during systole, curvature effects contribute to this swirling of blood. K: LAD artery section $C_1$ exhibits a bicellular flow pattern with one vortex of negligible strength as compared with the other reflecting skewing of velocity profiles. L: bicellular pattern changes to a unicellular pattern as we move from $C_1$ to $C_4$. 
MIDDISTOLE (t = 0.57 s). At this instant, the aortic valve remains closed and the majority of the back flow comes into the ascending aorta and enters into the graft vessel, with the remainder perfusing the LCA and RCA. Figure 10, A and B, shows the axial velocity distributions at different cross sections of the aorta along the horizontal and vertical center lines, respectively. With increasing distance from B, it is noted that the blunt profile (Fig. 10A) becomes parabolic. It is interesting to observe (Fig. 10B) that at section A2 (50 mm from B), the axial velocity distribution exhibits a “bidirectional behavior.” This bidirectional behavior may be due to the strong suction of the branching flow into the graft.

The nonplanarity of the graft vessel breaks the flow symmetry, resulting in pronounced movement of the fluid from the outer wall toward the inner wall (depicted in Fig. 10, C and D) caused by the pressure gradient induced by the curvature of the graft vessel. The flow (in the LAD artery exiting from the graft at high speed) results in pronounced skewing of the axial velocity profiles toward the bed of LAD artery (Fig. 10, E and F). The in-plane velocity components in Fig. 10, G–L, illustrate that the flow features are found to be qualitatively similar to those observed at the earlier diastolic times, except with an increase in the velocity magnitude. This high velocity magnitude in turn results in high WSS distributions at the proximal and distal anastomotic junctions, depicted in Fig. 11, A and B. The WSS in the aorta is uniform and low (~1.0 Pa), except at the proximal anastomotic junction, exhibiting a WSS of magnitude ~17 Pa (Fig. 11A). The WSS at the toe (~3.0 Pa) is much lower as compared with that observed on the floor of the artery (14.0 Pa) opposite to the distal anastomotic junction (Fig. 11B).

**DISCUSSION**

Despite the significance impact of the previous studies on the understanding of CABG hemodynamics, the previous models have only focused on only some aspects of the problem. For example, much of the modeling work in graft flow is limited to only a part of the total bypass conduit geometry, namely, the anastomosis site. A fully integrated model that accounts for all the relevant factors such as realistic geometry, inlet flow conditions, and anastomosis angle is not available. Some models have focused on more realistic inlet flow conditions, others on the 3D geometry, while yet others on a local domain of the CABG anastomosis. The major objective of the present study was to integrate all these aspects to provide a novel, 3D nonplanar graft geometry with physiologically measured inlet flow conditions over the entire domain of the CABG. Quasi-steady flow simulations were carried out by using CFD. The present analysis focused on the blood flow pattern and WSS because they are intimately related to the development of arterial diseases and hence the patency or failure of bypass graft. The results indicate that a properly contoured (out-of-plane) graft may contribute to improved patency.

**Effect of Geometry on Flow Simulation**

Because of the difficulties in retrieving patient-specific geometry, researchers have considered idealized geometries (the diameters of the host and the graft vessels being assumed equal) to predict the flow field and WSS in bypass grafts. Most of the CFD studies of the end-to-side anastomosis are based on geometries of two cylindrical conduits of equal diameter intersecting at angles ranging between 30° and 45°. Bertolotti and Deplano (5) adopted a nonstenotic proximal vessel model having a flow rate of one-eighth the flow rate in the proximal artery. With regard to the anastomosis geometry, Song et al. (29) developed a Y-figure anastomotic model for proximal arterial stenosis (at angles ranging from 10° to 30°) to analyze the 3D simulation of CABG. In their end-to-side anastomosis model, all the vessels were simulated to have the same diameter. They found that the WSS was lowest (contributes to intimal hyperplasia) at the heel position in 10° anastomosis and much lower as compared with that observed on the floor of the graft. This behavior may be due to proper geometrical adjustments of the total bypass conduit geometry.

In contrast to previous studies that have only considered the distal anastomotic junction (with all vessels of equal dimensions), the present study takes into account the complete 3D CABG model, which includes the aorta, the RCA and LCA entrances, the proximal and distal anastomotic junctions, and the occluded LAD artery. Furthermore, we have used realistic graft artery dimensions as provided by our surgeon coauthor.
Fig. 8. Axial velocity at start of diastole ($t = 0.32$ s). A: blood flows from ascending aorta into coronary arteries and because aortic valve is not fully closed, there is a small amount entering LV. Flow profiles are depicted with a negative sign to indicate change in flow direction. Blunt profile gradually begins to develop as flow progresses toward aortic inlet. B: different sections of the aorta exhibit a similar trend in axial velocity profile when extracted along horizontal center line. Most of flow enters coronaries, especially LAD artery (through graft). C: blood enters graft with a high flow rate. This along with nonplanarity of graft vessel results in considerable skewing. D: pronounced movement of blood from outer vessel to inner graft vessel is observed. E: presence of M-shaped profiles in LAD artery sections in $C_1$ and $C_2$ are due to high flow coming out of distal anastomotic junction. F: predominant skewing of the axial velocity profiles is observed along the bed of LAD artery seen in $C_1$ and $C_2$. As flow progresses downstream toward LAD artery exit, maximum velocity shifts and flow pattern exhibits symmetry. G: in-plane velocity components in $A_3$ are directed toward top wall as a result of suction effect caused by proximal anastomotic junction. H: presence of RCA and LCA results in the in-plane velocity vector components skewed to either side. More blood goes into LCA than RCA, thus showing peak velocity directed toward bottom wall. I: high flow rate in graft along with curvature of vessel wall causes skewing of the in-plane velocity vector components in $G_1$. J: movement of flow from top to bottom is observed reflecting the presence of second bend in graft vessel. Skewing of the in-plane velocities toward bottom portion of outer wall results in a small region of recirculation near top wall. K: $C_1$ exhibits counterrotating vortices of comparable magnitude reflecting M-shaped profile of axial velocity. L: a small region of recirculation is seen in section $C_4$. This indicates that flow is almost parabolic in nature as observed in axial velocity plot.
on the host artery bed were larger for the femoral waveform patterns, it was seen that temporal and spatial gradients of WSS different flow waveforms (LAD artery waveform, a femoral equal diameter intersecting at 45°. With a study of the effect of tomosis geometry, consisting of two cylindrical conduits of inlet flow on the WSS distribution in an end-to-side anastomosis model for different flow conditions were studied for anastomotic angles of 45° and 60° by Inzoli et al. (15). To mimic the surgical geometry, the intersection between the graft and the coronary vessel was made elliptical based on dimensions of the saphenous vein and the LAD artery (3, 16). The results show that the WSS along the bottom wall of the artery is higher for a larger graft anastomosis angle. These results agree in principle with the numerical simulations of Fei et al. (11), who also analyzed the effect of angle and steady flow rate in distal vascular graft anastomosis. In the present simulation, the anastomotic angle is smaller and conforms better to the surgically sutured geometry. Hence, the predicted flow patterns and WSS close to the anastomotic junctions may be more representative of the in vivo condition.

**Effect of Anastomotic Angle at Anastomosis**

For two different geometrical end-to-side coronary bypass models, steady-state simulations for different flow conditions were studied for anastomotic angles of 45° and 60° by Inzoli et al. (15). To mimic the surgical geometry, the intersection between the graft and the coronary vessel was made elliptical based on dimensions of the saphenous vein and the LAD artery (3, 16). The results show that the WSS along the bottom wall of the artery is higher for a larger graft anastomosis angle. These results agree in principle with the numerical simulations of Fei et al. (11), who also analyzed the effect of angle and steady flow rate in distal vascular graft anastomosis. In the present simulation, the anastomotic angle is smaller and conforms better to the surgically sutured geometry. Hence, the predicted flow patterns and WSS close to the anastomotic junctions may be more representative of the in vivo condition.

**Effect of Nonplanarity of Graft**

Recently, Sankaranarayanan et al. (27) introduced the first CFD model that includes the total bypass conduit in a planar geometry; i.e., the center lines of all the three vessels (aorta, bypass graft, and host vessel) lie in the same plane. Although the dimensions of the planar CABG geometry were somewhat different from those used in the present study, it was observed that the WSS was much higher in the planar geometry, particularly, at the toe and the bed of the distal anastomosis. Furthermore, the out-of-plane geometry breaks the symmetry of the flow and results in more uniform WSS on the bed of the distal anastomosis region as compared with that of the planar CABG model.

The in-plane bypass graft model is unattainable because of the geometry of the coronary system. Realistically, a bypass graft from the aorta to an occluded coronary vessel has an out-of-plane geometry. The present model indicates that the nonplanar graft vessel geometry results in low WSS at the toe and the bed of the LAD artery opposite to the distal anastomotic junction. Our results on WSS distribution are in agreement with Sherwin et al. (28), who carried out flow studies within a distal end-to-side anastomosis model (that was fully occluded proximal to the anastomotic junction) taking into account the nonplanarity of the bypass vessel. Their results showed that nonplanarity resulted in a 10% reduction in the peak WSS magnitude on the bed of the anastomosis. It was noted that the WSS was uniformly distributed along the bed of the artery, resulting in small spatial WSS gradients. This may be again due to the nonplanarity of the vessel geometry as seen in Caro et al. (8). They reconstructed the geometry of the aorta at sites of curvature and bends using casts and employed MRI techniques to measure the flow patterns in the arteries.
Fig. 10. Axial velocity at mid-diastole ($t = 0.57$ s). A: axial velocity profiles extracted along horizontal center line of different sections of aorta changes from blunt profile to an almost parabolic profile. B: junction effects are strongly reflected in axial velocity profiles extracted along vertical line of different sections of aorta. Profile in $A_2$ exhibit a bidirectional behavior, indicating that majority of flow enters graft during mid-diastole. C: pronounced movement of fluid from outer wall toward inner wall is seen due to pressure gradient caused by curvature of graft vessel. D: predominant skewing of velocity profile is observed with pronounced movement from outer to inner graft vessel caused by curvature of graft vessel. E: continuing effect of high flow rate from graft results in double-peaked and asymmetric velocity profiles. F: with increasing distance along LAD vessel, skewed velocity profile seen in $C_1$ and $C_2$ gradually shifts to center line of host vessel. G: similar trend as seen during early diastole is observed in the in-plane velocities in $A_3$. H: suction effect due to presence of LCA and RCA junctions is seen in $A_3$ of the in-plane velocity plot. I: a C-shaped profile is seen in $G_1$ of graft vessel. High flow in graft results in an increase in magnitude of the in-plane velocities as compared with early ejection. J: pattern of in-plane velocities is qualitatively similar to that seen at $t = 0.32$ s, except with an increase in magnitude. K: bicellular in-plane velocity vector pattern in $C_1$, with one vortex of higher strength (seen close to O) revealing effect of distal anastomotic junction. L: unicellular vortex pattern in $C_4$ reflects asymmetric distribution in axial velocity profile.
findings revealed that nonplanarity enhanced flow mixing and resulted in a more uniform WSS distribution.

These observations underscore the role of 3D graft geometry on the hemodynamics. The value of WSS in the toe region observed in this study is close to the physiological range of WSS (1–2 Pa), with an exception at mid-diastole, where the toe experiences a higher WSS of about ~3.0 Pa. Hence, it may be that the in vivo nonplanar graft geometry enhances graft patency.

**Critique of Model**

Despite the sophistication of the present analysis, a number of limitations exist in the model. For example, we have reported only four time points in the cardiac cycle (two during systole and two during diastole), whereas information on the temporal gradients of WSS and oscillatory shear index (OSI) requires additional time points. The extension of the model to additional time points for the determination of temporal gradients of WSS and OSI will be a natural extension of the present model. Furthermore, the elasticity of the vessel wall and the non-Newtonian fluid property of blood must be considered in future studies.

**Clinical implications of study.** The role of WSS in arterial disease has been the subject of debate for over three decades. Fry (12) reported that if the endothelial cells are exposed to a WSS magnitude of over 37 N/m², the endothelial surface would be subject to denudation. Caro et al. (7), on the other hand, reported that early atherosclerotic lesions are present in low WSS areas because of shear-dependent mass transport for atherogenesis. Furthermore, regions of low WSS and flow recirculation have been shown to correlate with locations of atheroma in coronary arteries (2). In summary, the hemodynamic culprits for intimal hyperplasia and atherogenesis are disturbances to streamlined flow, including WSS magnitude (too low or too high relative to a homeostatic value) and direction (forward vs. reverse flow), WSS gradients (temporal and spatial), high OSI, flow separation and secondary flow, and long particle residence time (17). The present paradigm is that these hemodynamic features influence endothelial cell response by inducing platelet activation, cell migration and vascular smooth muscle cell proliferation, release of mitogenic factors and proteinases (see review in Ref. 17).

In the present study, we determined the WSS distribution from the velocity profiles based on realistic out-of-plane 3D geometry of CABG. Our results show that the WSS is uniformly distributed on the bed of the anastomosis (opposite to the distal anastomotic junction) and the magnitude of WSS at the toe region is close to the physiological range. The section of the host artery close to the heel is continually exposed to low WSS because of the proximity of relatively stagnant fluid in this vessel segment. Our nonplanar WSS model results are consistent with those obtained by Sherwin et al. (28) for a distal end-to-side anastomosis junction. The nonplanar graft vessel geometry has resulted in swirling of blood between the outer and inner walls of the graft. This may be deemed advantageous because it might wash out the deposits of the arteries that cause arterial disease (18).

It is well known that fluid shear stresses have a definite bearing on endothelial cell shape and function. It has been shown that, in lesion-prone regions, the endothelial cells are polygon shaped, whereas in nonatherosclerotic regions they are elongated and aligned in the flow direction (26, 33). Lei et al. (20) have shown that, in disturbed flow regions, the elongation and alignment of endothelial cells are difficult to achieve because of high WSS gradients. The realistic out-of-plane graft geometry leads to smaller WSS and WSS gradients that cause the endothelial cells to be more elongated and aligned in the flow direction. Along these lines, it may be best to provide more laxity at the ends of the anastomosis to enhance the out-of-plane geometry. This recommendation is now being adopted by our surgeon coauthor (T. Y. Seng).

In conclusion, the nonplanar geometry of the present aorto-left CABG is seen to reduce the spatial variation of WSS distribution in the distal anastomotic region. This geometry also reduces the overall level of WSS variation along the bed of the anastomosis, thereby reducing damage to the endothelium which may improve graft patency.

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