Capillary Perfusion and Wall Shear Stress Are Restored in the Coronary Circulation of Hypertrophic Right Ventricle

Yunlong Huo, Carlos O. Linares, Ghassan S. Kassab

Abstract—It has been shown that right ventricle (RV) hypertrophy involves significant compensatory vascular growth and remodeling. The objective of the present study was to determine the functional implications of the vascular growth and remodeling through a full flow analysis of arterial tree down to first capillary segments. A computer reconstruction of RV branches including the proximal right coronary artery to the posterior descending artery was established based on measured morphometric data in arrested, vasodilated porcine heart. The flows were computed throughout the reconstructed trees based on conservation of mass and momentum and appropriate pressure boundary conditions. It was found that the flow rate was significantly increased in large epicardial coronary arteries in hypertrophic as compared with control hearts but normalized in the intramyocardial coronary arteries and smaller vessels in RV hypertrophy primarily because of the significant increase in number of arterioles. Furthermore, the wall shear stress was restored to nearly homeostatic levels throughout most of the vasculature after 5 weeks of RV hypertrophy. The compensatory remodeling in RV hypertrophy functionally restores the perfusion at the arteriolar and capillary level and wall shear stress in most of larger vessels. This is the first full analysis of coronary arterial tree, with millions of vessels, in cardiac hypertrophy that reveals the compensatory adaptation of structure to function. (Circ Res. 2007;100:273-283.)

Key Words: right coronary artery ■ intravascular pressure ■ coronary flow ■ right ventricular hypertrophy

It is widely recognized that cardiac hypertrophy is accompanied by vascular growth and remodeling. The degree of vascular compensation varies, however, in the left and right ventricles and depends on the degree of cardiac hypertrophy. In right ventricle (RV) hypertrophy, it has been documented that vascular angiogenesis and remodeling are nearly compensatory for flow. Kassab et al have previously shown significant expansive remodeling in the large epicardial vessels and new flow channels at the arteriolar level in a porcine model of RV hypertrophy.

With the availability of detailed morphometric data on vascular geometry and branching pattern of RV branches in control and RV hypertrophy, a systematic hemodynamic analysis can be performed to investigate the adaptation of structure to function (perfusion). To facilitate the structure/function analysis, our group has recently developed a computer algorithm that allows full reconstruction of the entire coronary arterial tree down to the arterial capillaries (first capillary segments) from morphological cast and histological data. The computer model facilitates the analysis of the coronary arterial circulation based on detailed anatomical data.

Here, a computer reconstruction of the RV branches was implemented down to the capillary level in the right coronary artery (RCA) proximal to the posterior descending artery of control and RV hypertrophy. The detailed morphometric data (ie, vessel diameters, lengths, and numbers) were used to determine the flow throughout the vascular network as well as the wall shear stress. The blood flow was calculated under steady state in arrested, vasodilated pig hearts. The distribution of wall shear stress was studied in the entire coronary arterial tree. One major finding is that RCA flow is significantly increased because of a decrease in vascular resistance primarily attributable to the increase in number of small vessels. Although the flow is increased in large vessels (eg, epicardial), it is normalized in smaller vessels (eg, arterial capillaries and arterioles). The wall shear stress in the majority of vasculature is nearly restored to the normal levels after 5 weeks of RV hypertrophy. This study demonstrates, for the first time, the hemodynamic implications of vascular growth and remodeling in RV hypertrophy.

Materials and Methods

Anatomical Model

Previously, Kassab et al have measured the morphometric data of the RCA posterior descending arterial tree. Briefly, the morphometric data on the coronary arterial vessels of diameters <40 μm were obtained from histological specimens. Plugs of myocardial tissues were removed from RV of hypertrophic and control pigs. Each plug was completely sectioned transmurally into 60- to 80-μm thickness. Each section was dehydrated with alcohol and cleared with methyl...
salicylate to render the myocardium transparent and the elastomer-filled microvasculature visible under a light microscope.

The morphometric data on the coronary arterial vessels of diameters >40 μm were obtained from cast studies. The same hearts used for the histological studies of the microvasculature were corroded after removal of tissue plugs for histological sections. The RCA posterior descending arterial casts were dissected and viewed with a stereo-dissection microscope and displayed on a video monitor through a television camera as described by Kassab et al.11 The images were retrieved by a digitizing system and analyzed using computer software. Several lumen diameter measurements were made along each vessel segment to obtain a mean diameter, and the vessel segmental length was obtained by measuring the distance between bifurcation points along the centerline of the vessel. The trunks of the coronary arteries were sketched and their segments measured. The subtrees arising from the trunk were labeled, excised, placed in separate jars, further sketched, and measured. This process was continued until the entire RCA posterior descending arterial tree was reconstructed down to the arterial capillaries.

The raw data on the RV branches excluding the posterior descending artery were reconstructed in control and RV hypertrophy by using the growth algorithm introduced by Mittal et al.10 Based on measured morphometric data. A 2-step approach was used in the reconstruction of the entire coronary arterial tree down to the arterial capillaries (≤8 μm in diameter).10 Briefly, portions of the right ventricular branches missing from the cast data were computationally reconstructed from anatomical data. The growth of a broken or terminal cast vessel ≥40 μm entailed the search of intact bifurcations (for which mother and the 2 daughter vessels were measured) with mother diameter closest (smallest difference) to the terminal vessel. Once such a bifurcation was found, it was “pasted” to the broken vessel. A search was then made for each of the 2 daughter vessels of the pasted bifurcation and so on. This process was repeated until the tree was reconstructed down to ~40 μm. Once the tree was reconstructed down to 40 μm, the growth strategy changed from reconstruction of the tree bifurcation-by-bifurcation to subtree-by-subtree. Basically, a broken vessel whose diameter was in the range of 8 to 40 μm was allocated to a subtree by finding a vessel in the input arteriolar file (saved histological data) closest (smallest difference) to the cut vessel. The vessel closest to the desired diameter could be selected from anywhere along the measured subtree. The selected subtree was then pasted to the cut vessel. A check was made at each bifurcation to ensure that the mother-to-daughter diameter ratio was ≥1. The above process was repeated until the pasted subtree had branches with diameters ≤8 μm. In the present reconstruction, only RV branches were reconstructed down to the capillary level between the RCA and posterior descending artery. Figure 1 shows the main trunk proximal to the posterior descending artery.

It should be noted that the present digital morphometric model of coronary arterial tree is different from that reported by Kassab et al.1 because the assignment of orders was performed automatically after the entire tree was reconstructed. In the previous study, the data existed in hard records and the ordering scheme was performed manually. In that approach, the assignment of orders was made iteratively from the microvessels (histological specimens) to the larger vessels (cast).1 The differences between the morphometric data for the 2 schemes have been previously outlined in detail for the normal coronary arterial trees. In summary, the present anatomical mathematical model has exact data (diameters, lengths, and connectivity) for the larger vessels and statistical reconstructions for the microvessels, which was different from previous models that were based on statistical data (ie, reconstructions from tables with means and SDs for diameters, lengths, connectivity) for the entire tree.11,12

Flow Simulation
After the branching pattern and vascular geometry of RV branches were generated, a steady-state flow analysis was performed that was similar to a previous study.13 Briefly, the blood flow is assumed to be impermeable, incompressible, laminar Poiseuille steady-state flow, and free from end effects. The blood vessel was considered as an axisymmetric cylinder. The blood flow in every vessel was treated as axisymmetric flow. The details of the flow analysis are presented in the Appendix.

Data Analysis
Because of the enormity of data points, we adopted several schemes to represent the data to facilitate interpretation. The flow and wall shear stress (eg, Figures 2, 3, 5, 7a, and 7b) are presented as a function of the cumulative length of the trunk and primary branch pathways to the arterial capillaries. As shown in Figure 1, the main trunk begins at the root (the most proximal vessel) and is defined by the path corresponding to the largest vessel at each bifurcation down to the capillary vessels. Similar pathways are considered for each primary branch down to the arterial capillaries.

The distribution of wall shear stress in every vessel of the RCA tree (eg, Figures 6a, 6b, 7c, and 7d) is presented as density plots.
showing the frequency of data. To obtain these figures, a grid was superimposed on the data of interest and the number of data points in each square was counted. Based on a normalized count (frequency), a shade was assigned proportional to the frequency, i.e., darkest shade reflects highest frequency or density and the lightest shade reflects the lowest frequency.

For some hemodynamic parameters, the data were divided into ten diameter ranges as follows: range 1 (≤ 8 μm, arterial capillaries); range 2 (8 to 20 μm); range 3 (20 to 30 μm); range 4 (30 to 40 μm); range 5 (40 to 50 μm); range 6 (50 to 100 μm); range 7 (100 to 400 μm); range 8 (400 to 900 μm); range 9 (900 μm to diameter of largest primary branch; 1205 μm and 1540 μm for control and RV hypertrophy, respectively); and range 10 (trunk diameter, >1205 μm for control and >1540 μm for porcine RV hypertrophy).

**Statistical Analysis**

Student's t test was used to detect differences between control and RV hypertrophy. A probability value of <0.05 was indicative of a significant difference between the 2 populations.

**Results**

Ten anatomic reconstructions of the trees were performed in control and RV hypertrophy. Because the results were statistically similar, 1 typical reconstruction was used to simulate the hemodynamic parameters. The reconstruction algorithms yielded RV branches with a mean of 0.36 million and 1.3 million vessels for control and RV hypertrophy, respectively. The number of vessels in RV hypertrophy is ~4 times larger than that in control. Because the posterior descending arterial tree was excluded, the total number of vessels is significantly smaller than that of the entire RCA tree. The number of terminal vessels (precapillary arterioles)/perfused RV weight (=13.6 and 39.1 g for control and RV hypertrophy, respectively) yields values of ~7×10^3 and 8×10^3 for hypertrophic and control hearts, respectively. The perfusion (inlet flow rate per unit mass) is equal to 1.1 and 0.8 mL/min per gram for control and hypertrophic hearts, respectively.

There is a total of 16 and 23 primary branches (branches that arise directly from the trunk) in control and RV hypertrophy, respectively. The cumulative length of the RCA to posterior descending artery in porcine RV hypertrophy is ~3 cm longer than the control heart. The cumulative length of the paths through the primary branches to the arterial capillaries is also significantly longer in RV hypertrophy (~12 cm as compared with 7 cm for the control).

**Structure/Flow Analysis**

The solution to Equation 5 (Appendix) was obtained in the form of a column vector of nodal pressures throughout the RV branches for control and hypertrophic hearts. With every node pressure known, the flow rate through each segment can be computed according to Equation 1 (Appendix). Figure 2 shows the variation of flow (volumetric flow rate) along the cumulative length of the trunk and primary branches in the RCA tree proximal to the posterior descending artery. An abrupt drop for both control and RV hypertrophy can be observed at the order 8 vessels (=300 to 500 μm in diameter),14 where proximal to the drop are epicardial coronary arteries and distal to the drop are intramyocardial coronary arteries.

To understand the structure/flow relationship of RV branches, we studied the variation of cross-sectional areas (CSAs) along the cumulative length of the various pathways to arterial capillaries in analogy with the flow variation. Figure 3 shows the variation of CSA along the cumulative length of the trunk and primary branches in the RCA proximal to the posterior descending artery for the 2 groups corresponding to Figure 2. The variations in CSA mirror the trends in the flow in control and RV hypertrophy.

To explore the detailed differences in the flow rates, we examined the flows in different diameter range of vessels. Figure 4A depicts the mean and SD of flow rate in vessels of different diameter ranges. The differences in the mean flow rates in the smaller vessels (<400 μm in diameter) between control and RV hypertrophy were very small (~15%). The flows were significantly higher in RV hypertrophy, however, for vessels >400 μm in diameter. The difference in the flow of the main trunk and primary branches was more than 100% in RV hypertrophy as shown in Figure 4B. It was also found that the difference in the relative dispersion or coefficient of variance (CV=SD/mean×100) was smaller at the arterial capillaries in RV hypertrophy (Figure 4B). Hence, the inlet capillary flow heterogeneity tends to decrease in RV hypertrophy under vasodilated conditions.

**Wall Shear Stress**

The wall shear stress was computed from equation 6 (Appendix) as shown in Figure 5A and 5B for the trunk and main branches of control and RV hypertrophy, respectively. The mean value in the main trunk of RV branches was ~7 dynes/cm^2 and was not significantly different in the 2 groups (P=0.187). A density plot of the wall shear stress throughout the vasculature of control and RV hypertrophy is shown in Figure 6A and 6B, respectively. Although the patterns are very similar, the variances are somewhat larger in RV hypertrophy. Figure 6c shows the difference of wall shear stress between hypertrophic and control hearts at each diameter range. Specifically, the wall shear stress was found to be normalized in all vessels except in the range of 8 and 9 (400 μm, to primary branch diameters).

Figure 7a and 7b show plots of the relationship between wall shear stress and pressure in the main trunk and primary branches of control and RV hypertrophy, respectively. A density plot is shown in Figure 7c and 7d for the entire arterial vasculature (including the arterial capillaries) of the 2 groups, which demonstrates that the wall shear stress first increases and then decreases with pressure. It can be noted that there is a decrease in wall shear stress in the arterial capillaries (Figure 7c and 7d), which is not observed in Figure 7a and 7b. The reason is that the main trunk and primary branches (in Figure 7a and 7b) descend to the arterial capillaries along the path of the larger diameter at each bifurcation, which does not reflect the trend of the whole RCA tree (Figure 7c and 7d). In Figure 7c and 7d, the lower wall shear stress corresponds to the larger and smaller vessel diameters that have correspondingly higher and lower pressures, respectively. The higher and more uniform wall shear stress occurs in the intermediate pressures.

**Discussion**

We report several novel findings including (1) the structural and functional hierarchy of coronary vasculature is preserved...
in porcine RV hypertrophy; (2) the increase in flow rate in larger branches along with the significant increase in number of vessels and vascular CSA in RV hypertrophy restores the inlet capillary flow to control values; (3) the relative dispersion of blood flow into the capillary bed is decreased in RV hypertrophy; (4) the wall shear stress in RV branches is nearly restored after 5 weeks of pulmonary banding except for vessels in the diameter range of 400 to 1540 μm; and (5) a novel relation is observed between pressure and wall shear stress throughout RV branches in control and RV hypertrophy. We elaborate below on each of these findings in turn.

**Structure/Function Adaptation**

The number of vessel segments in RV hypertrophy is \( \approx 4 \) times larger than that in control hearts. In particular, the number of small arterioles and capillaries increased signifi-

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**Figure 2.** Relationship between the flow in a vessel and the cumulative length of the vessel from the root to the first segment of capillaries through all primary branches in control (a) and RV hypertrophy (b), as shown in Figure 1. The pressure at the inlet of the RCA trees is set as 100 mm Hg, and the outlet of first segment of capillary pressure is 26 mm Hg. The rectangular bar indicates the transition (order 8 vessels, \( \approx 300 \) to \( 500 \) μm in diameter) from epicardial to intramyocardial vessels. PDA indicates posterior descending artery.
cantly after 5 weeks of pulmonary banding. The increase in the number of small arterioles and arterial capillaries plays an important role because they are the major site of vascular resistance. It is also found that number of terminal vessels/perfused RV weight and myocardial blood flow are similar in the 2 groups, which suggest a nearly metabolic adaptation in RV hypertrophy.

Kassab has previously noted a structural and functional hierarchy in the coronary circulation. An abrupt change in CSA and flow was observed at the junction of epicardial and intramyocardial vessels (order 8 vessels). The present data in the control heart is confirmatory (Figure 2a and 3a). An interesting finding is that the remodeling process in RV hypertrophy maintains the same structural and functional hierarchy as in control hearts (Figures 2b and 3b, respectively). The epicardial coronary arteries tend to maintain their CSA and flow fairly uniform to reach the entire surface area of the RVs. Because the surface area of RV is significantly increased in RV hypertrophy, the length of the main trunk of RCA and primary branches are significantly increased with
more primary branches arising from the RCA trunk as compared with the control heart. The growth of vascular length appears compensatory to the growth of myocardial surface.

To explore the demarcation of increased flow in the vasculature, we categorized the vessels in various diameter ranges. It was found that the increase in flow occurs for vessels >100 μm in diameter, whereas the flow in the microcirculation (<100 μm) was nearly restored to control levels (Figure 4). Hence, although the flow in RV hypertrophy was significantly increased in the proximal vessels, it was distributed through numerous more channels such that the flow in the inlet capillaries is similar to control.

**Flow Heterogeneity**

Kassab et al have shown previously that the degree of asymmetry of the resistance and flow at a bifurcation were reduced in RV hypertrophy, thus resulting in decreased flow heterogeneity. Growth of new vessels occurs primarily at the capillary and arteriolar level whose diameter asymmetry ratios are close to 1 (ie, fairly symmetric) and do not significantly change in RV hypertrophy. The primary changes in the diameter asymmetry ratios occur in the larger vessels; however, because of the remodeling of blood vessel lumen in response to flow overload. The relative dispersion of flow in the main trunk was decreased in RV hypertrophy, which is consistent with our previous findings of decreased asymmetry (Figure 4b).
Blood flow heterogeneity in normal myocardium has been investigated by many authors (see review19,20), but we cannot find any reference to the changes in flow heterogeneity in right ventricular hypertrophy. Einzig et al21 reported that there is a reduction in the degree of heterogeneity of left ventricular perfusion in the banded dogs as compared with those of controls. The results of the microsphere studies are consistent with the reduction of CV of flow into the capillary bed found in the present study (Figure 4B). The observed decrease of flow heterogeneity in hypertrophy may be attributable to a decrease in work heterogeneity in that state, as suggested by previous mathematical models22 and experimental measurements.23,24

**Uniform Shear Hypothesis**

The remodeling of a vessel segment in response to flow overload (eg, in an arteriovenous fistulae) has been shown to obey the uniform wall shear-stress hypothesis.25,26 This hypothesis implies that the cube of diameter must increase in proportion to the increase in blood flow to restore a homeo-

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**Figure 5.** Relationship between wall shear stress and the cumulative length of the vessel from the root to the arterial capillary through all primary branches in control (a) and RV hypertrophy (b).
Figure 6. Relationship between wall shear stress and normalized vessel diameter in the RCA trees in control (a) and RV hypertrophy (RVH) (b). c. Difference in the wall shear stress ([RV hypertrophy − control]/control × 100) for various diameter ranges.
static level of wall shear stress (Equation 6 in the Appendix). Blood vessels can accommodate such a change in vessel diameter acutely through vasoactive mechanisms (flow-dependent constriction or dilation) and chronically by remodeling of vascular caliber. The present study is the first to examine the wall shear stress in flow-overload in an entire vascular system rather than a single segment. Our findings reflect a tendency for the restoration of wall shear stress for the majority of vessels. The wall shear stress remains elevated, however, in vessels of 400 to 1500 \( \mu \text{m} \) in diameter after the 5 weeks of duration (Figure 6). This is reasonable, as previous studies have shown that significantly longer periods (up to 6 months) are needed to more fully restore the wall shear stress in single vessels. It will be interesting to examine the wall shear stress in this intermediate size vessels after a longer period of adaptation.

**Figure 7.** Relationship between wall shear stress and pressure from the root to the first segment of capillaries through all primary branches in control (a) and RV hypertrophy (b). Relationship between wall shear stress and pressure in the entire RCA tree in control (c) and RV hypertrophy (d).

**Pressure/Shear Hypothesis**
Pries et al studied the relation between wall shear stress and intravascular pressure in arterioles, capillaries, and venules. They reported a monotonic transition from low to high wall shear stress as intravascular pressure increases from 15 to 50 mm Hg. The present study shows a parabolic profile with an extended plateau in the entire system of RV branches, as seen in Figure 7. It is noted that the wall shear stress is maintained and then decreases at the proximal and distal portions of the tree. The trend in RV hypertrophy is similar to that in control. Although the decrease of the wall shear stress in the distal tree (microcirculation) was previously noted by Pries et al, the decrease of wall shear stress in the proximal tree was not observed because of the limited range of vasculature (mesentery) investigated. The present results generalize the pressure/shear hypothesis in an entire system of coronary arterial tree including the arterial capillaries.
Critique of Study
Despite the extensive anatomical data used in the present simulations, a number of assumptions remain. First, the flow is assumed to be steady state in the diastolic state of the heart. Although we have recently extended the full vascular analysis to pulsatile flow,\(^\text{30}\) the steady-state mean flow is similar to that obtained here, and hence the conclusions will remain unchanged. Second, we assumed a fully developed Poiseuille-type flow in every vessel. This assumption is reasonable when the diameter-to-length ratio is small in the diastolic state heart, even if the flow is pulsatile.\(^\text{30}\) Third, the vessels are assumed to be in a vasodilated state in the absence of vasomotor tone where coronary flow reserve is substantially reduced. Although this will undoubtedly change the flow heterogeneity, the present analysis considers the minimal vascular resistance. Finally, future studies must consider the vessel/myocardial in systole as this will be different in control and RV hypertrophy. The present conclusions apply only to the diastolic state of myocardium.

Clearly, the flow reserve is different in normal and hypertrophic hearts.\(^\text{31}\) The present data and analysis only consider the minimal vascular resistance under vasodilated conditions. We opted for this approach because the vasodilated state is experimentally more reproducible. The degree of vasodilation is undoubtedly increased during the progression of cardiac hypertrophy as the workload is increased; ie, coronary flow reserve is decreased.\(^\text{31}\) Although the process of vascular remodeling is complex, the roles of local hemodynamic factors (shear stress, wall stresses, strains, etc), which depend on the tone of vessel, are eminent.\(^\text{32}\) Future studies are needed to elucidate the role of vascular tone on vessel growth and remodeling.

Significance of Study
RV hypertrophy is a compensatory response to pulmonary hypertension. It is physiologically and clinically important to understand the compensatory adaptations of the coronary circulation in RV hypertrophy to devise management and treatment. Here, we show that the capillary flow is essentially similar in RV hypertrophy as compared with control. Hence, vascular remodeling including changes in diameter, length, and number of vessels serve to maintain perfusion of the increased myocardial mass. Furthermore, the remodeling process restores the general pattern of wall shear stress through the majority of coronary vasculature. This is the first study to reflect this finding in a full network of this magnitude. Finally, a novel relation is illustrated between the wall shear stress and intravascular pressure in the entire coronary vascular system and generalizes the pressure/shear hypothesis. The detailed analysis provides a deeper understanding of vascular growth and remodeling and may provide insight into clinical management of ventricular hypertrophy.

Appendix

Network Analysis
The volumetric flow rate \(Q_i\) in a vessel between any 2 nodes, represented by \(i\) and \(j\), is given in terms of the pressure differential \(\Delta P_{ij}\) and vessel conductance \(G_{ij}\) as Poiseuille’s equation:

\[
Q_{ij} = \frac{\pi D_{ij}^4}{128} \Delta P_{ij}
\]

where \(\Delta P_{ij} = P_i - P_j\), \(G_{ij} = D_{ij}^4/\mu_j L_{ij}\), and \(D_{ij}\), \(L_{ij}\), and \(\mu_j\) are the diameter, length, and viscosity, respectively, between nodes \(i\) and \(j\). The viscosity is considered as a function of vessel diameter. Pries et al\(^\text{33}\) proposed a modified viscosity relationship based on a compilation of data, which reflect the Fahraeus–Lindqvist effect, as given by:

\[
\mu_j = \left[1 + 6 \cdot e^{-0.085D_j/2.44} + 3.2 - 2.44e^{-0.085D_j/3.2} - 1\right] \cdot \left(\frac{D_{ij}}{D_j - 1.1}\right)^2 \cdot \left(\frac{D_{ij}}{D_j - 1.1}\right)^2
\]

This relationship is used throughout the vasculature of a given vessel diameter \(D_j\).

By conservation of mass, we can have the following equation:

\[
\sum_{j=1}^{m_i} Q_{ij} = 0
\]

where \(m_i\) is the number of vessels converging at the \(j\)th node. The volumetric flow into a node is considered positive and flow out of a node is negative for any branch. From Equations 1 through 3, a set of linear algebraic equations in pressure for \(M\) nodes in the RCA tree may be written as follows:

\[
\sum_{j=1}^{m_i} (P_i - P_j)G_{ij} = 0
\]

Once conductance and suitable boundary conditions are specified, the final global matrix formulation may be written as:

\[
GP = G_0P_b
\]

where \(G\) is the matrix of conductance, \(P\) is the column vector of the unknown nodal pressures, and \(G_0P_b\) is the column vector of the conductance times the boundary pressure of their attached vessels. The pressure at the inlet and outlet was set as 100 and 26 mm Hg, respectively. The larger sparse matrix \(G\) is solved by using the LU decomposition with partial pivoting and triangular system solvers through forward and back substitution (SuperLU_dist, which is implemented in ANSI C, and MPI for communications). Once the pressure at every node is calculated, the flow in each segment can be determined from Equation 1. The wall shear stress may be calculated by the following equation:

\[
\text{wall shear stress} = \frac{32\mu_jQ_{ij}}{\pi D_{ij}^3} = \frac{\Delta P_{ij}D_j}{4L_{ij}}
\]

Equation 6 was used to compute the wall shear stress in each vessel of the trees.

Acknowledgments
We thank Henry Yu Chen for the coronary model in Figure 1.
Sources of Funding
This research is supported in part by National Heart, Lung, and Blood Institute/NIH grant 2-R01-HL05554-08.

Disclosures
None.

References
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Circ Res. 2007;100:273-283; originally published online January 11, 2007; doi: 10.1161/01.RES.0000257777.83431.13

Circulation Research is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7330. Online ISSN: 1524-4571

The online version of this article, along with updated information and services, is located on the World Wide Web at:
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