A numerical study on the flow of blood and the transport of LDL in the human aorta: the physiological significance of the helical flow in the aortic arch

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Liu X, Pu F, Fan Y, Deng X, Li D, Li S. A numerical study on the flow of blood and the transport of LDL in the human aorta: the physiological significance of the helical flow in the aortic arch. Am J Physiol Heart Circ Physiol 297: H163–H170, 2009. First published May 8, 2009; doi:10.1152/ajpheart.00266.2009.—It has been proposed that a mass transfer phenomenon called concentration polarization of low-density lipoproteins (LDLs) may occur in the arterial system and is likely involved in the localization of atherogenesis. To test the hypothesis that concentration polarization of LDL may be suppressed by the helical flow pattern in the human aorta, hence sparing the ascending aorta from atherosclerosis, the effects of aortic torsion, branching, curvature, and taper on blood flow and LDL transport in the lumen were simulated numerically under steady-state flow conditions using four aorta models constructed based on in vivo MRI slices. The results showed that it was the aortic torsion that induced the helical flow in the aortic arch, stabilizing the flow of blood in the aorta, and compensated the adverse effects of the aortic curvature on blood flow and LDL transport. The helical flow reduced the luminal surface LDL concentration in the aortic arch and probably played a role in suppressing severe localization of LDL at the entrances of the three branches on the arch, hence, protecting them from atherogenesis. The taper of the aorta was another important feature of the aorta that further stabilized the flow of blood and delayed the attenuation of the helical flow, making it move beyond the arch and into the beginning part of the descending aorta. The results therefore may account for why the ascending aorta and the arch are relatively free of atherosclerosis.

low-density lipoprotein; helical flow; atherosclerosis; concentration polarization; shear stress

CLINICAL AND POSTMORTEM STUDIES revealed that atherosclerotic lesions in the arterial wall develop at certain sites in the human arterial system, such as the inner wall of curved segments and the outer walls of arterial bifurcations (1, 20, 32). This phenomenon is called the localization of atherosclerosis. It has been widely recognized that flow-induced shear stress is one of the most important hemodynamic factors in the localization of atherogenesis (26). Nevertheless, because of the fact that the early event leading to the genesis of atherosclerosis is the accumulation of cholesterol and other lipids within the arterial wall, in recent years researchers have been paying more and more attention to material transport in the circulation and the interactions of blood cells with the blood vessel walls (23). From the point of view of mass transport, the concepts of residence time for atherogenic agents (25) and deposition of atherogenic particles onto the blood vessel walls (18) were proposed to account for the localization of atherogenesis.

Deng et al. (9) has theoretically predicted a mass transport phenomenon of concentration polarization of atherogenic low-density lipoproteins (LDLs) with the LDLs increasing in concentration from bulk value toward interface within the arterial system and verified it experimentally in vitro (39). Apparently, the occurrence of concentration polarization of LDLs in the arterial system can affect the residence time and the deposition of atherogenic particles. They, therefore, have suggested that flow-dependent LDL concentration at the blood/wall interface may play an important role in the localization of atherogenesis.

The aorta is the major blood vessel of complex geometry with such characteristics as branching, twisting, taper, and curvature (14). Moreover, it has been suggested that the asymmetric development of the aortic arch was attributed to the hemodynamics determined by a genetic program (40). Since blood flow in the aorta is intricate, plenty of velocity measurements have been performed in the past forty years using hot film, Doppler ultrasound, phase-contrast MRI, and other tools (22, 33, 34). The studies demonstrated that flow pattern in the ascending aorta was skewed and helical flows predominated in areas from the ascending aorta to the aortic arch, and this form of blood flow was a basic pattern for almost all the subjects no matter what age and sex (2, 17). This kind of flow pattern was believed to have certain beneficial effects (2, 22, 28) with a possible tendency of sparing the arterial wall from atherosclerotic plaques. It was also found that carotid atheromatous disease was associated with a reduction in the prevalence of systolic spiral flow pattern in the aortic arch (17). Therefore, a number of flow studies on the human aorta have been carried out to better understand its fluid dynamics (2, 17, 22, 34). Because of the difficulties in the measurement of blood flow in vivo, numerical flow simulation with its high resolution and straightforward presentation of flow parameters such as velocity distribution, pressure, and wall shear stress (WSS) is still the main tool to investigate blood flow patterns in the aorta (19, 35). Nevertheless, to our knowledge, no one has studied the hemodynamics of the aorta from the viewpoint of mass transport, particularly the transport or spatial distribution of atherogenic lipids such as LDLs. We believe that the helical flow observed in the aorta may have a great influence on the distribution of luminal surface LDL concentration, hence, on the spatial distribution of atheromatous plaques in the aorta, because it may suppress/eliminate areas of flow stagnation so as to prevent the accumulation of atherogenic lipids on the luminal surfaces of the ascending aorta and the aortic arch. To verify this hypothesis, in this study models of the human aorta with different features were constructed based on in vivo MRI.
slices and LDL transport in the models was investigated numerically to evaluate the distributions of LDL concentration on the luminal surface of the aortic models. The effects of aortic torsion, branching, taper, and curvature on the flow pattern and the luminal surface LDL concentration were also discussed.

**METHODS**

**MRI and Three-Dimensional Geometrical Reconstruction**

Images from 40 transverse slices of the thorax were acquired from a healthy male volunteer of 38 yr old using a 1.5 Tesla imager (Signa, GE Medical System). These slices included the images of the ascending aorta, the aortic arch, and a majority portion of the descending aorta. Volunteers gave written, informed consent to this study that approved by the Ethical Committee of Peking University First Hospital and carried out in accordance with the regulation of the hospital.

The manually segmented aortic lumen boundaries by Mimics (Materialise N.V.) were used to reconstruct the primitive three-dimensional model of the aorta while the centerline of the aorta was exacted directly. The constructed model was resliced in such a way to ensure that the new slices were perpendicular to the centerline. The resliced cross sections were simplified as circles with the same cross-sectional area, centroid, and vertical direction. Four models as shown in Fig. 1 were created from the simplified circles.

**Model 1.** Model 1 was created to mimic the human aorta with all its geometrical features intact.

**Model 2.** To investigate the influence of branching on the distribution of luminal surface LDL concentration, model 2 was made the same as model 1 but without the branches.

**Model 3.** To investigate the torsion of the aortic arch on the distribution of luminal surface LDL concentration, model 3 was constructed with the torsion of the aorta removed. The centerline curvature of model 3 was basically the same as that of model 2.

**Model 4.** Model 4 was created to investigate the effects of arterial taper on blood flow and LDL transport. This model had the same feature as that of model 3 but with the tapering removed along the model.

**Numerical Approaches**

**Assumptions.** Blood was treated as a homogeneous, incompressible Newtonian fluid (24, 30). Arterial wall was assumed to be no-slip rigid (13, 27). The simulations were carried out under steady-state flow conditions.

**Governing equations.** The flow simulation was based on the three-dimensional incompressible Navier-Stokes equations:

\[ \rho(\dot{u} \nabla)\dot{u} + \nabla p - \mu \Delta \dot{u} = 0 \]  
\[ \nabla \cdot \dot{u} = 0 \]

where \( \dot{u} \) and \( p \) represent, respectively, the fluid velocity vector and the pressure. \( \rho \) and \( \mu \) are the density and viscosity of blood (\( \rho = 1,050 \text{ kg/m}^3 \), and \( \mu = 3.5 \times 10^{-3} \text{ kg/m} \cdot \text{s} \)).

Mass transport of LDLs in flowing blood can be described by the following:

\[ \dot{u} \cdot \nabla c - D \Delta c = 0 \]

where \( c \) is the concentration of LDLs and \( D \) is the diffusion coefficient of LDL in blood, assumed to be 4.8 \times 10^{-12} \text{ m}^2/\text{s} (6).

**Boundary conditions.** The boundary conditions (BC) for the Navier-Stokes equations were as follows:

**BC-A.** For model 1, at each of the three aortic arch branches, 5% of flow volume was allowed to be ejected (35).

**BC-B.** A flat inlet flow velocity profile was used, assuming the time average \( Re = 790 \) (velocity is 0.1 m/s) based the measurements (16, 29).

The boundary conditions for the mass transport equation were as follows (38):

**BC-1 inlet:**
\[ c = c_0 \]  

**BC-2 outlet:**
\[ \frac{\partial c}{\partial n} = 0 \]

**BC-3 wall:**
\[ \nu_c \frac{\partial c}{\partial n} - D \frac{\partial c}{\partial n} = \bar{m} \]

where \( c_0 \) is the LDL concentration in the bulk flow, \( \nu_c \) is the filtration velocity of LDL across the vessel wall \( (\nu_c = 4 \times 10^{-8} \text{ m/s}) \) (38), and \( c_w \) is the concentration of LDLs at the luminal surface of the artery. Suffice \( n \) indicates the direction normal to the boundary, and \( \bar{m} \) was assumed to be 0 (9).

**Computation procedures.** The numerical calculations were carried out using a validated finite volume-based algorithm Fluent (Ansys, USA) with a user-defined C-like function, which was used to solve the mass transport equation and was validated by the results from Deng et al. (9). The results were mesh independent.

**RESULTS**

**Velocity Profiles**

To facilitate the presentation of the velocity field in the aorta models, six representative slices were selected along the longitudinal direction of the centerlines shown in Fig. 2A. Slice A is located at the front part of the ascending aorta, slice B at the middle way of the ascending aorta, slice C at the entrance of the aortic arch, slice D between the left common carotid artery and the left subclavian artery, slice E at the exit of the aortic arch, and slice F at the anterior part of the descending aorta. The view of the velocity field for all the slices is shown in Fig. 2B.

For model 1, at the entrance, the axial velocity profile was flat. However, as blood moved to the front part of the ascending aorta (slice A), the flow was skewed toward the inner aortic wall, forming a crescentic distribution of axial velocity (Fig. 3). Unlike the planar models (models 3 and 4, Fig. 4), the crescent in model 1 was asymmetric. The flow remained the same fashion till the entrance of the aortic arch (slice C), the skewness of the flow shifted toward between the inner wall and the posterior wall. The distribution of the axial velocity became relatively uniform there. With the flow moving farther down, its uniformity was enhanced at the middle part of the aortic arch (slice D) with the skewness of the flow shifted to the posterior wall. At the exit of the aortic arch (slice E), the axial velocity became uneven again with a clearer skewing of...
the velocity profile toward the anterior wall. The skewness then shifted to the outer wall at the descending aorta. In general, the position of the peak axial velocity rotated clockwise in the aortic arch.

**Effect of branches.** For model 2 without branches, the axial velocity profile was similar to that for model 1, except that beyond the corresponding location for the branches, blood velocity was greater when compared with that for model 1.

**Effect of torsion.** The result for model 3 showed that without the torsion, blood flow was highly skewed toward the inner wall, and this effect was moving along the median plane to the outer wall in the descending aorta.

**Effect of taper.** The axial velocity profile of model 4 resembled that of model 3. However, without the arterial taper, blood velocity became smaller and the flow skewness was clearer in the descending aorta. It is worthy to mention that only in model 4 was the reversed axial flow detected in the middle part of the aortic arch.

### Secondary Flows

For model 1, as shown in Fig. 3, there were detectable secondary flows at the inner and posterior walls of the ascending aorta (slice A,a). A small vortex was developed near the anterior wall (slice A,b). At the middle portion of the ascending aorta, the secondary flow was strengthened with two vortexes formed along the anterior and the posterior walls (slice B). When the flow moved into the aortic arch, the secondary flows became stronger; meanwhile, the anterior wall vortex grew bigger squeezing the posterior wall vortex to a narrow region, which exhibited apparent characteristics of swirling or helical flows (slices C, D, and E). However, the swirling or helical flow formed in the arch attenuated in the descending aorta (slice F).

**Effect of branches.** A comparison of the secondary flow between models 1 and 2 showed that model 2 had a very similar secondary flow pattern as model 1, indicating that the branches on the aortic arch had little effect on the formation of secondary flows in the aorta.

**Effect of torsion.** Figure 4 shows the flow patterns in models 3 and 4. Very different from the flow patterns in models 1 and 2, the vortexes formed in models 3 and 4 were symmetric with a typical characteristic of Dean flow, indicating that without torsion, the helical flow observed in the aortic arch would not appear in a curved tube without torsion.

**Effect of taper.** The secondary flow in model 4 resembled that in model 3, except that at the location of slice E, there were four vortexes instead of two formed in model 4, two of them occupied most of the space and squeezed the two small ones in the corner along the inner wall (slice E). This indicates that the aortic taper has the function to stabilize the flow of blood in the aorta.

For all the models, there was no apparent difference in the contours of the velocity, indicating that the secondary flow was only a small portion of the primary flow.

### Luminal Surface LDL Concentration

The distributions of luminal surface LDL concentration in the four models are shown in Fig. 5.

**Model 1.** Generally speaking, the luminal surface LDL concentration ($c_w$) in the ascending aorta was relatively even, which was 10–15% higher than the bulk LDL concentration ($c_0$). However, $c_w$ distributed quite unevenly in the aortic arch where $c_w$ along the inner wall was much higher than along the outer wall, especially from the distal end of the aortic arch to its apex (region B) where $c_w$ was 25% higher than $c_0$ and was the highest in the whole aorta. The second highest $c_w$ was at region A in the entry area of the brachiocephalic arch, where the luminal surface LDL concentration $c_w$ was ~20% higher than $c_0$, whereas $c_w$, in the neighboring areas of the other two branches (region D) was relatively low. The third highest $c_w$ was located in region C of the descending aorta. In general, almost the whole inner wall of the descending aorta was affected by severe LDL concentration polarization, leading to high $c_w$ in the descending aorta when compared with the ascending aorta.

**Effect of branches.** Evidenced from Fig. 5, the distribution of the luminal surface LDL concentration in model 2 was similar to that in model 1. However, $c_w$ in the descending aorta was generally lower for model 2 when compared with model 1.

**Effect of torsion.** The results indicated that for model 3 (with the torsion of the aorta removed), the distribution of $c_w$ along the outer wall of the ascending aorta became uneven when compared with model 2, and in the descending aorta, LDL concentration polarization turned more severe.

**Effect of taper.** For model 4, the luminal surface LDL concentration $c_w$ was elevated entirely when compared with other models. Without the arterial taper, almost all the inner wall of the descending aorta was affected by severe LDL concentration polarization, leading $c_w$ there 50% higher than $c_0$.

### Correlation of $c_w$ Distribution with the Distribution of WSS

Figure 6 shows the distributions of WSS in the four models. The highest WSS in model 1 was ~5.5 Pa and located at the flow divider of the brachiocephalic left common carotid branch (not shown in Fig. 6). There were another two places where the WSS was relatively high. One was located at the entrance of the left subclavian artery (not shown in Fig. 6); the other was located in an area along the anterior wall of the aortic arch very close to region E (Fig. 6). The two lowest WSS areas were located at regions A and B, respectively. The WSS in most portions of the ascending aorta was about 0.5 Pa, and it was...
relatively lower along the outer wall when compared with the WSS along the inner wall. The comparison of $c_w$ distribution with the distribution of WSS showed an adverse correlation between the two, especially in areas with WSS below 0.25 Pa, where $c_w$ was elevated significantly. Nevertheless, the numerical results also revealed that although the WSS of the ascending aorta was relatively low, the luminal surface LDL concentration there was not particularly high. In addition, region D in the aortic arch was the area with the lowest luminal surface LDL concentration, the value of which was almost the same as that of the bulk concentration $c_0$, but the WSS at region D was not the highest in the aorta. Therefore, the present study indicated that WSS was not the only factor that determined the distribution of $c_w$. The luminal surface LDL concentration was probably affected by other factors, such as flow pattern itself as well.

Effect of branches. The WSS distribution of model 2 resembled that of model 1. Nevertheless, with the branches removed, WSS in the descending aorta for model 2 was generally higher than in model 1. The enhanced WSS in the descending aorta of model 2 led to the reduced $c_w$ there when compared with that in model 1 (Fig. 5).

Effect of torsion. When compared with model 2, the removal of aortic torsion resulted in a bigger area of low WSS in the ascending aorta, which led to the distribution of $c_w$ as uneven there. In addition, for model 3, the value of WSS in the descending aorta was generally lower than those for models 1 and 2, which caused the elevation of $c_w$ in the descending aorta of model 3 (Fig. 5).

Effect of taper. Without arterial taper, WSS in model 4 was generally lower when compared with the other three models, which led to an enhanced $c_w$ in the whole model with the outer wall of the ascending aorta and the inner wall of the descending aorta affected by a severe concentration polarization of LDLs (Fig. 5).
DISCUSSION

In vivo measurements revealed that blood flow in the aortic arch took a form with a corkscrew-like pattern, and there was no clear effect of age or sex on the presence of spiral flow (2, 17, 33, 34). The ascending aorta is not the only place in the circulation where spiral or swirling blood flow was observed. Stonebridge et al. (36) evidenced the existence of spiral blood flows in human infrainguinal blood vessels and found that out of 75 arteries examined, 51 had spiral folds on their endoluminal surfaces. With their observations combined with those of others (21, 34, 37), they argued that spiral blood flow was a normal physiological flow phenomenon, at least in parts of the circulation. Frazin et al. (11) even believed that spiral or helical flow may account for a significant amount of normal organ perfusion from branch vessels due to the centripetal spin induced in blood. Stonebridge et al. (36) hypothesized that the rifled endoluminal surfaces of the arteries might reflect the inherent structural features in the elastic wall of the vessels and that spiral flow could exert a beneficial effect on the mechanisms of endothelial damage repair. The study by Caro et al. (4) had supported this hypothesis and demonstrated that spiral flow may lead to a relative uniformity of wall shear and an inhibition of flow stagnation, separation, and instability.

We believe that the swirling or spiral motion of blood flow in the human aortic arch is a typical example of “form follows function” in the vascular system and hypothesize that it is the swirling blood flow that eliminates stagnation flow regions and provides guaranties for the inner surface of the ascending aortic wall to get smooth and even a flushing by the blood so that atherosclerotic plaques can hardly form in the area of the ascending aorta. To test this hypothesis, in the present study we simulated numerically the flow of blood in the aorta and its effect on LDL transport in the lumen of the aorta. We also verified the influence of aortic torsion, branching, and taper on...
blood flow and LDL transport by using four different models of the aorta.

Our simulation showed that because of the filtration flow across the aortic wall, the luminal surface LDL concentration \( c_w \) was always higher than the bulk concentration \( c_0 \), which is consistent with the findings by Deng et al. (9). The present study revealed that as the spiral flow originating from the middle of the ascending aorta was spinning over the ascending aorta and most of the portion of the aortic arch, the flow of blood became relatively stable, with flow separation as hardly occurring in these areas of the aorta (3, 22). As a result, the luminal surface LDL concentration \( c_w \) was low and distributed relatively evenly in the ascending aorta and in most of the aortic arch, which may account for why atherosclerotic plaques seldom form in the aortic arch. In contrast to this, severe LDL concentration polarization was predicted in region A of the brachiocephalic branch, in region B at the exit of the aortic arch, and along the whole inner wall of the descending aorta, which may explain why atherosclerotic plaques develop preferentially in these areas of the aorta (12, 15, 31).

The present study showed that the flow pattern of model 2 with no branching was generally similar to that of the human aorta model (model 1), indicating that the spiral flow pattern in the aorta was not caused by the existence of the three branches, which, though, might affect the local blood flows in their neighborhood. The locally disturbed flows by the nonplanar branches rendered LDL polarization especially severe in region A of the brachiocephalic branch. Nevertheless, the results demonstrated that, in general, the helical flow induced in the aortic arch could stabilize the flow of blood in the three branches, which might account for why LDL polarization or \( c_w \) was relatively low and evenly distributed in most regions of the branches and at the aortic arch apex. Without the helical flow, this might not be possible, and both the flow disturbance and LDL polarization might have been severe in the three branches.

It has been suggested that the twisted figure of an artery is the most important factor causing spiral blood flow in the artery, and therefore in the arterial system, the curvature and branching of the large arteries are commonly nonplanar (5). With this realization, the idea of nonplanar geometrical features has been proposed in the design of end-to-side arterial anastomose (10) and arteriovenous shunt graft (4).

To our knowledge, there have been no studies that have analyzed the effect of aortic torsion on blood flow and LDL transport using a physiologically mimic model of the human aorta. The present numerical study has demonstrated that the human aorta definitely has advantages over planar-curved vessels in terms of hemodynamics and LDL transport. The comparison between models 1, 2, and 3 showed that the ascending aortas of models 1 and 2 had obviously reduced areas of low
WSS. In these areas of low WSS, $c_w$ was relatively low but distributed more evenly due to their nonplanar torsions when compared with model 3 that was a planar-curved vessel. Moreover, the concentration polarization of LDL in the descending aorta was relatively relieved as a result of the helical flow induced by the aortic torsion.

Previously, the study by Zabielski and Mestel (41) using a simplified model of the aorta arch without arterial taper showed that the nonplanar curvature of a vessel could limit the severity of flow separation along the inner side of the bend and reduce the spatial variation of WSS. The present study also predicted a similar result.

The aorta has a significant taper. It has been suggested that the cross-sectional area of the aorta changes in an exponential form (7, 14), with the lumen cross-sectional area reduced by about 50% from the root of the aorta to the beginning of the descending aorta (7). The comparison of the $c_w$ distribution between models 3 and 4 showed that without arterial taper, LDL polarization in model 4 was much severer than that in model 3. There might be two reasons for this. First, the aortic taper can stabilize the flow of blood. Unlike model 3 with the aortic taper that only has one pair of vortexes, the present study predicted two pairs of vortexes in model 4, which is consistent with the findings by Choi et al. (8) in their experimental study. The second reason is that with the taper, the flow velocity in the descending aorta of model 3 was generally much higher than that of model 4. This, in turn, would lead to a reduced LDL polarization in model 3 when compared with model 4 (9, 38).

The present numerical study revealed an adverse correlation between WSS and the luminal surface LDL concentration in the aorta. The high $c_w$ of LDL tends to be located in areas with very-low WSS. Nevertheless, this correlation between WSS and $c_w$ is not clear-cut for certain regions of the aorta. For instance, there are two locations along the outer wall of the ascending aorta that have lower WSS than other parts of the ascending aorta and even lower than most parts of the descending aorta. But $c_w$ in the ascending aorta distributes quite uniformly and is much lower than that in the descending aorta. The results, therefore, indicate that the luminal surface LDL concentration depends not only on the local WSS but also on both the global and local flow patterns. In this aspect, our results are in good agreement with those of Wada and Karino (38).

In conclusion, the helical flow induced by aortic torsion may stabilize the flow of blood in the aorta, reducing the flow disturbance and suppressing the separation of flow. This, in turn, may have a positive effect on the transport of LDLs in the lumen of the aorta, lessening the polarization of LDLs in the aortic arch. In addition, the helical flow may also play a role in suppressing severe polarization of LDLs at the entrances of the three arterial branches on the arch, therefore protecting them from atherogenesis. The taper of the aorta is another important feature of the aorta. It can further stabilize the flow of blood, making the helical flow induced in the ascending aorta move far beyond the arch and into the beginning part of the descending aorta. Without the taper, the helical flow might have been attenuated faster. Overall, we believe that the helical flow in the aorta has important physiological significance in the circulation.

REFERENCES


