Pulsatile Flow in a Model Carotid Bifurcation

David N. Ku and Don P. Giddens

Pulsatile flow in an in vitro model of the human carotid bifurcation was studied by flow visualization using hydrogen bubble techniques. A glass model was constructed after determining an average geometry from 57 biplanar angiograms of 22 adult subjects ranging from 34 to 77 years of age. The flow pulse used was a half-sine wave superimposed upon a mean flow. Maximum and minimum values of the instantaneous Reynolds number were 1200 and 400, respectively, based upon conditions in the common carotid model artery; the frequency parameter was 6.0. The division of flow into the internal-external branches was 70:30. Visualization by hydrogen bubbles demonstrated significant deviations from steady flow behavior. Flow separated in the carotid sinus over the entire cycle, but the location and extent of separation varied strongly. The direction of flow near the walls of the model changed sharply during the cycle except for the region near the apex of the bifurcation where the orientation of streaklines was more nearly unidirectional at all times. Bubbles entering the separated flow region tended to remain entrapped there for several cycles. Rapid dispersion of bubbles occurred in the internal branch near the end of systole, suggesting the presence of flow disorder. The location of low wall shear stresses, directionally varying stresses, and longer residence times for fluid elements appears to coincide with the localization of early atheromatous plaques in human carotid specimens. (Arteriosclerosis 3:31–39, January/February, 1983)

The flow at the carotid artery bifurcation may have significant bearing on the development and management of atherosclerosis in this major blood vessel. For over a century, hypotheses have linked flow disturbances at this and other arterial branches with the formation of atheromas. Furthermore, advances in noninvasive ultrasound studies of blood velocity in the carotid arteries have pointed to the need for a more rigorous understanding of normal physiologic flow patterns at this branch. Fluid flow patterns at branches are highly dependent on geometry, Reynolds number, and flow division ratios. In the case of human physiology, the flow is additionally pulsatile, introducing a time-dependent behavior. Second order effects which influence the flow, but to a much lesser degree, are the compliance of the arterial wall and the non-Newtonian viscosity of blood.

Recent investigators have described an average geometry of the human carotid bifurcation based on biplanar angiograms and cadaver specimens. Steady flows at physiological Reynolds numbers and flow division ratios have been visualized and quantified. These results indicated circumferential secondary helices in the internal carotid branch, very low wall shear stresses at the outer wall of the sinus, and relatively large shear stresses at the apex and distal end of the sinus as the flow enters the internal carotid artery. No turbulence was observed in those studies. The secondary flow appeared to be influenced most strongly by a flow division between the daughter branches and, to a lesser degree, by the upstream Reynolds number.

This paper presents the results of visualization of pulsatile flow patterns in a model of the carotid bifurcation and compares our observations to the results obtained with steady flow experiments.

Methods

The model used in this study was constructed of blown glass after determining an average geometry from 57 biplanar angiograms of 22 adult subjects ranging from age 34 to 77 years. The human carotid pulse was simulated by a half-sine wave with DC offset to model the acceleration and deceleration of systole, followed by a relatively steady period of positive diastolic flow (figure 1 A). A servocontrolled shaker valve was connected in series with a constant pressure head to generate the pulsatile flow. The valve was placed approximately 100 diameters upstream from the test section. The downstream
branch resistances were fixed by two screw valves; and the fluid, a mixture of water and glycerine, emptied into a constant level recovery tank before recirculating. The frequency parameter \( (\alpha = R \sqrt{\omega/v}) \) used was 6.0, a value obtained by assuming a human common carotid artery radius of \( R = 4.0 \text{ mm} \), pulse rate of \( \omega = 8.4 \text{ sec}^{-1} \), corresponding to 80 bpm and kinematic viscosity \( (v) \) of \( 3.5 \times 10^{-6} \text{ cm}^2/\text{sec} \). The Reynolds number \( (Re = UD/v = 2Q/mR) \) based on diameter was 400 for the steady diastolic phase, corresponding to a common carotid flow of 8.8 ml/sec, and 1200 for the peak of systole.\(^6\) The percentage of flow division ratios of the internal to the external carotid were taken as 60:40, 70:30, and 80:20, with 70:30 being an average physiological value.

The flow rates in both the internal and external branches were monitored continuously with a Carolina Medical electromagnetic flowmeter. Hydrogen bubbles were generated by applying a current of 0.5 mamp through a 0.0015 inch (0.038 mm) diameter stainless steel wire which was oriented in the plane of the bifurcation. Still photographs were taken sequentially using a Nikon F 35 mm camera with a 55 mm macro lens and motor drive on Kodak 2475 recording film. Movies were made with a Paillard/Bolex camera using Kodak 16 mm Four-X reversal film. The test section was illuminated by two 300 W spotlights from above and below against a black background.

Fluid dynamicists'\(^7\) define several quantities in describing flow fields: 1) streamline—a continuous line within the fluid whose tangent at any point is in the direction of the velocity vector at that point; 2) streak-
line—the locus of all fluid elements that have previously passed through a particular fixed point; and 3) particle pathline—the line formed by the trajectory of an individual fluid element. In steady flow these three "lines" are equivalent, but in unsteady flow they are all different. Consequently, the interpretation of hydrogen bubble patterns in pulsatile flow is not as straightforward as for steady flow. An additional complication is the fact that hydrogen bubbles do not precisely follow the trajectories of fluid elements due to buoyancy, although this effect is small in these studies. Figures 2–5 (see Results section) were obtained under conditions of continuous bubble generation and, consequently, the motion visualized represents streaklines of the flow. For figures 6 and 7, the bubbles were generated for one cycle, and then the wire current was abruptly stopped before the photographs were taken.

Results

The pulsatile waveform of flow in the system followed the input waveform very closely. Figure 1 B is a trace of the flow in the internal carotid branch as detected by the electromagnetic flowmeter and compares well with the input waveform shown in figure 1 A. The total flow remained positive throughout the cycle in both branches and the flow division ratio remained constant to within 5%.

The pulsatile flow in the carotid bifurcation changed rapidly and dramatically throughout the pulse cycle. Figures 2–5 show a sequence of such changes. The inset in the upper right-hand corner of each figure illustrates the time during the cycle at which the photograph was taken. The evolution of the separated flow region in the carotid sinus occurring within each beat is evident in this sequence of figures. During the increasing velocity phase (figure 2), the flow approaching the internal carotid branch separated along the outer wall shortly after entering the sinus. (The outer portion of the bulb was free of bubbles due to the fact that this photographic sequence was initiated immediately after the application of the current to the hydrogen bubble-generating wire so that no residual bubbles from the previous cycle existed.) Figure 3 was taken just after the peak velocity in the cycle was attained. The streakline defining the edge of the separation zone had moved toward the inner wall and the separation point had moved slightly proximally, both effects combining to yield a growing region of secondary flow. Helical flow patterns were now visualized within the separated region. Such secondary flow patterns, created by circumferential velocity components, existed throughout the cycle due to the curvature of fluid streamlines in such bifurcations. Figure 4 was taken near the end of systole, just before the constant velocity, diastolic portion of the waveform. The separated flow zone had expanded considerably and the bubbles forming the helical patterns had dispersed

Figure 3. Formation of helical secondary flows at the peak of systole. The conditions are \( \alpha = 6 \) with a 70:30 flow division.
Figure 4. Flow during the deceleration phase of systole. The conditions are $\alpha = 6$ with a 70:30 flow division.

Figure 5. Flow at the end of systole showing the vortex formation in the distal sinus and the breakup of secondary flows in the distal internal carotid. The white arrow points to the proximal point of separation along the outer wall.
somewhat within the sinus. The separation point had moved further proximally as shown by the arrow. Finally, the flow patterns during the constant velocity phase of the input waveform are shown in Figure 5. Again, the large separated flow region was well visualized and the deceleration of fluid entering the internal carotid artery resulted in a small vortex along the inner wall of the distal sinus. Additionally, the bubble patterns in the internal carotid branch were now rather diffuse, indicating that the predominantly axial flow, which entered this branch during the increasing velocity phase of the input waveform, became somewhat disrupted during diastole. This behavior is different from that observed in steady flow and may indicate a tendency toward turbulence. Quantitative measurements of velocity would be required, however, to determine whether turbulence actually existed.

To better visualize the flow patterns in the sinus, photographs were taken after the bubbles were produced for a cycle and then production was terminated. Figure 6 was taken during the subsequent cycle, at the time indicated in the inset. In this photograph the flow division ratio was 60:40. The bubble patterns clearly illustrate the existence of complex helical structures. Although it cannot be demonstrated by a single figure, real-time observation shows that this helical organization was actually moving proximally at the time the photograph was taken, and some of the bubbles spilled from the sinus into the external carotid branch.

Figure 7 is a photograph of the bubbles in the separation zone which have remained for two cycles after clearance of the mainstream for a flow division ratio of 70:30. Although these bubbles were soon purged from this region, indicating that it was not a true recirculation zone, Figure 7 illustrates that the residence time of fluid elements in the neighborhood of the outer sinus wall was considerably greater than elsewhere in the bifurcation.

It may be inferred, particularly from real-time observation, that portions of the outer wall of the sinus experienced rather low wall shear rates. This is further substantiated by figure 8, which is a photograph of a machined plexiglass model now used for laser Doppler anemometer measurements of velocity under pulsatile flow conditions. This figure shows the location of deposits of small debris which accumulated in this model after a long period of pulsatile flow operation at a 70:30 flow division. These deposits were denser than the fluid, and the model was oriented so that the arrow indicates the gravitational direction. Deposits occurred most heavily along the outer wall of the sinus, implying that the wall shear rate in

**Figure 6.** Flow during the deceleration phase demonstrating the reverse flow and the helical formation of the internal branch, which spills fluid into the external branch. The flow division ratio is 60:40. The arrow points to the extent of reverse flow which matches that shown in figure 5.
Figure 7. Prolonged residence time of the bubbles in the sinus region persisting after a clearing of mainstream bubbles.

Figure 8. Accumulation of fluid impurities in the sinus region after an extended pulsatile flow operation.
this region was insufficient to convect the debris away from the surface. A small number of deposits accumulated in the common carotid segment just proximal to the bifurcation. This was undoubtedly due to a low shear rate region which preceded flow separation at the entrance to the sinus. The region between these deposits was free of debris. It appeared from the flow visualization that this was an area where the flow separation point was alternately moving distally and then proximally during the cycle and was therefore experiencing forward and reverse wall shear stresses. Direct measurements would be required, however, to substantiate this hypothesis.

**Discussion**

It must be stressed that while visualization aids considerably in understanding flow phenomena under complex conditions, such studies are not an end in themselves. Rather, it is necessary to obtain quantitative data to describe the flow field adequately. Nonetheless, the visualizations in these experiments demonstrate that pulsatility presents a different character of flow at the carotid bifurcation from that of steady flow conditions. Steady flow at the branch shows a fixed separation region, no evidence of turbulence, a well-defined unchanging secondary flow structure, and wall shear stress vectors which do not change in time.

In contrast, pulsatile flow creates a continuously changing region of separation; possible turbulent disturbances at the end of systole; vortices which dramatically vary in size and energy level; wall shear stress vectors which change in both magnitude and direction, especially at the point of outer wall divergence, and the frequency-dependent phenomena of an increased residence time of the bubbles in the sinus, the extent of the separation region, and the size of the secondary vortices. Thus, although flow at a single instant of time in the pulse cycle may be similar to that of steady flow at a comparable Reynolds number, the continuously changing structure of pulsatile flow may be important to the physiological environment of the arterial wall.

Although a review of the numerous hemodynamic theories of atherogenesis is beyond the intent of this work, the flow patterns demonstrated in the present study and in previous steady flow experiments may aid in understanding the problem of localization of early atheroma. There are at least three prominent postulates relating wall shear stress to atherogenesis. In an early work, Fry suggested that high values of wall shear stress might lead to a disruption of the endothelial cells and subsequently to atherosclerosis. Indeed, numerous studies in experimental animals indicated that Evans blue dye uptake is greater in areas suspected of experiencing higher wall shear stresses. It is anticipated that this should be associated with increased lipid ingress into the arterial wall. At the other extreme of the wall shear stress controversy is the hypothesis of Caro et al. that atherogenesis occurs in regions of low wall shear stress as a consequence of the inhibition of lipid egress from the arterial wall. Finally, more recent work has suggested that a wall shear stress vector that varies with time in direction and/or magnitude may not allow endothelial cells and their nuclei to align uniformly. Fry observes that lipid deposition preferentially occurs in the less ordered, more cellular intimal thickenings around orifices and tends to spare regions with highly oriented collagen fibrils that exist in unidirectional flow areas. Intimal regions exposed to an unstable stress pattern tend to manifest a more exaggerated intimal thickening characterized by an increased population of smooth muscle and connective tissue cells, poorly oriented collagenous fibers, and a predilection for lipid deposition. The similarity of this description and that of atheromatous plaques is striking.

The present study in pulsatile flow, combined with previous steady flow investigations, have clearly identified four regions in the neighborhood of the carotid bifurcation that experience different wall shear stress environments. These are depicted schematically in figure 9. First, there is a region (I) near the apex of the flow divider that experiences relatively higher and nearly unidirectional wall shear stresses throughout the cycle. Next, there is a zone (II) along the outer wall of the carotid sinus that is always exposed to a relatively low wall shear stress; and, in fact, there appears to be a subset of this zone that is subjected to negative values of wall shear stress (i.e., directed proximally) throughout the entire cycle. This latter point was predicted by Bharadvaj et al. from steady flow experiments and has recently been demonstrated noninvasively by Wood in a human subject using multichannel pulsed Doppler ultrasound as reported by Check. Third, the lateral walls of the sinus (III) appear to be exposed to wall shear stresses that change direction and magnitude considerably over each heart cycle. Finally, there appears to be a small region (IV) between the common carotid artery and the zone of "permanent" separation within which the flow separation line migrates distally and proximally during each cycle. This region experiences a complete reversal of wall shear stress with each beat. Although the magnitude of wall shear stress at this location is not yet known and must await quantitative studies, it is likely to be relatively small in view of the low values found in the immediate vicinity.

An important question, of course, is that of where early lesions actually localize in the carotid arterial system. Preliminary studies of the topographic distribution of early atheromatous lesions in humans consistently indicate that the carotid sinus is affected first. The initial plaque is located radially at the outer wall of the sinus away from the flow divider, not at the flow divider itself. Thus, it appears that, at least in the human carotid artery, high wall shear stress is not associated with early atheroma. The outer wall of the internal carotid bifurcation is precisely the region which experiences low axial wall
shear stress during the cycle, variations in stress magnitude and direction over each cycle, and areas of prolonged residence time of fluid elements. Flow reversal at the wall extends upstream of the bifurcation during part of the cycle under the pulsatile conditions studied here. If these changing flow directions are related to the formation of atheromas, then this observation would predict that the first localization of early plaque would occur slightly upstream of just into the proximal segment of the sinus and/or along its lateral walls. However, if permanently low magnitudes of wall shear stress or increased residence time of blood particles are associated with atherogenesis, then early plaques should begin more distally into the sinus in the area of permanent flow separation. The exact topography of early lesions in the carotid bifurcation of humans must be determined for accurate correlation to the different hemodynamic conditions existing in close proximity. This information is currently being analyzed.

Insofar as the relation to noninvasive detection of localized plaques using flow disturbances is concerned, pulsatile flow through a model of the carotid artery bifurcation reveals time-varying secondary flows which disperse the bubble patterns at the end of systole. These disturbances, which maximize at the end of systole beyond the carotid sinus, are sufficiently dispersive to be considered as possibly turbulent. Turbulence was not present in steady flows at Reynolds numbers which are physiological in the carotid branch, nor has turbulence been observed in previous model studies of the aortic bifurcation. It is important to recognize the presence of velocity disturbances in physiologically normal pulsatile flows since turbulent behavior is being used to characterize stenotic disease in blood vessels. Such normal flow disturbances might be diagnosed as a false-positive indicator of carotid disease.

References
8. Dean WR. Note on the motion of fluid in a curved pipe. Phil Mag 1927;4:208–223

Index Terms: atherosclerosis • carotid bifurcation • pulsatile flow • shear stress
Pulsatile flow in a model carotid bifurcation.
D N Ku and D P Giddens

doi: 10.1161/01.ATV.3.1.31

Arteriosclerosis, Thrombosis, and Vascular Biology is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1983 American Heart Association, Inc. All rights reserved.
Print ISSN: 1079-5642. Online ISSN: 1524-4636

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://atvb.ahajournals.org/content/3/1/31

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Arteriosclerosis, Thrombosis, and Vascular Biology can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Arteriosclerosis, Thrombosis, and Vascular Biology is online at:
http://atvb.ahajournals.org//subscriptions/