Flow Studies in a Model Carotid Bifurcation

Frank W. LoGerfo, Michael D. Nowak, William C. Quist, Howard M. Crawshaw, and Bala K. Bharadvaj

Boundary layer separation in a plexiglass model carotid bifurcation was investigated in relation to the origin of atherosclerotic plaque clinically found in this region. Our model was comparable to a human carotid in both dimensions and geometry. Water flowed through the model at Reynolds numbers from 200 to 1200 under steady and pulsatile flow conditions, with outflow through the external and internal branches varied. The near-wall flow was visualized by slow injection of dye through ports machined in the model. Under steady flow at a physiological Reynolds number of 500 and a flow split at the bifurcation similar to that of a human carotid at rest, boundary layer separation was found to occur in a carotid sinus across from the external carotid origin, forming a shell of slowly moving fluid around the bifurcation. The rapidly moving mainstream impinged directly on the flow divider. The location of atherosclerotic plaque correlates best with the low shear region of separation and not with the region of high shear at the flow divider. Preliminary studies with pulsatile flow demonstrated little change from the steady flow results. (Arteriosclerosis 1981; 1:235–241)

The predilection for atherosclerosis to occur at bifurcations in the arterial tree is well documented.1 2 This finding led to the hypothesis that hemodynamic factors are in some way involved in atherogenesis, although the precise nature of the flow or pressure changes favoring atherogenesis is not yet well understood. In humans, one of the most common sites for localization of atherosclerosis is at the carotid bifurcation, especially at the origin of the internal carotid artery where it may be the cause of transient ischemic attacks or stroke.3 4 There have been various investigations of flow through model carotid bifurcations.5–8 In previous work, Balasubramanian7 developed a model of the human carotid bifurcation based on averaged data from angiographic measurements. The flow field within this bifurcation model was studied with the laser Doppler anemometer under steady flow conditions, and velocity profiles across the bifurcation determined.9 This work demonstrated a large region of flow separation and low wall shear stress at the origin of the internal carotid. In previous work from this laboratory, we have demonstrated the three-dimensional nature of separation and secondary flow in model anastomoses and bifurcations.9 10

The Balasubramanian model does not incorporate the curvature of vessels that is seen on arteriography. We have taken the internal measurements from Balasubramanian’s model and have superimposed on that the curvature of vessels seen in the arteriogram of a carotid artery with atherosclerotic plaque present at the bifurcation (figure 1). This model is, therefore, a very close approximation of the carotid bifurcation seen in figure 1 prior to the formation of the atherosclerotic plaque. This allows us to demonstrate the flow field under steady and pulsatile flow conditions. The relevance of these studies to the formation of atherosclerotic plaque is also discussed.
Figure 1. Angiogram after which our carotid bifurcation was modeled. The model is a close approximation of the geometry of this carotid bifurcation before development of the atherosclerotic plaque. Note the location of the plaque in the carotid sinus opposite the origin of external carotid.

Methods

To produce the required model, internal dimensions taken from the Balasubramanian model were used to make a brass bifurcation with a common carotid diameter of 8 mm by turning on a lathe. Silicon rubber was poured around this brass model to obtain a mold which, in turn, was used to make a cast of the bifurcation from paraffin wax. This cast was matched with the carotid arteriogram of a patient with atherosclerosis (figure 1) for the purpose of adjusting the angles of branching and curvature of the vessels. Clear acrylic was poured around this wax casting to form an encasing transparent block. The wax was removed initially by warming the block of acrylic and later by dissolving the wax with xylene to obtain the final working model. The inner surface of the model was carefully polished to produce a smooth surface that had adequate optical clarity for visualization purposes.

The model was installed in a simple flow system designed to provide steady, as well as pulsatile, flow. A constant head tank, employed to generate the flow through the system, was connected by means of flexible tubing to a long plexiglass tube with an inner diameter of 8 mm. The straight section was chosen to be 80 cm in length (100 diameters) to ensure that fully developed laminar flow was established when the fluid entered the model bifurcation connected to the downstream end. The flow rate through each daughter vessel could be individually controlled by means of screw clamps located far downstream from the model. Provision was made to include a Harvard pulsatile pump in the flow system between the constant head tank and the straight plexiglass section to superimpose pulsations on the steady flow generated by the head tank.

Dye injection ports were machined into the model at various sites in the region of the bifurcation and 100 diameters upstream. Toluidine blue and red fuchsin dyes were adjusted to neutral buoyancy with denatured alcohol titration for use in flow visualization. The dyes were injected through the various ports using a Harvard constant infusion pump, the site and rate of infusion being adjusted to give the best delineation of the region of flow separation at the bifurcation (figure 2). Blue dye was slowly infused so as to remain within wall streamlines and demonstrate points of separation, reattachment, and secondary flow. Red dye was injected as a 1 to 2 cc bolus into the upstream injection port. This dye entered the central flow stream and assumed a laminar flow profile as it approached the bifurcation. For the purposes of this study, mainstream flow (as noted in figures 2–8) was the visualized flow near the center flow line. Therefore, the mainstream flow was defined by the red dye, while the separation was simultaneously defined by blue dye. The flow patterns were photographically recorded (figure 3) and perspective drawings were based on the photographs. The drawings incorporate observations from different angles after several dye injections to completely define the flow field and to provide a three-dimensional perspective.

Similarity requirements dictate that the Reynolds number of flow in the model parent vessel be the same as that in the common carotid artery for meaningful results. \( \text{Re} = \frac{VDp}{\mu} \), where \( \rho \) is the density, \( V \) a representative velocity through the vessel, \( D \) the diameter of the artery, and \( \mu \) the absolute viscosity of blood. An examination of the literature pertaining to flow rates through the human carotid bifurcation11,12 and vessel diameters13,14 indicates that the Reynolds number of flow in the common carotid artery is about 450, based on the mean flow velocity and assuming a blood viscosity of \( 4.0 \times 10^{-6} \text{g/cm.s} \). However, due to the pulsatile
Figure 2. Experimental model. Near-wall boundary layer flow visualized using a syringe pump and selected ports for dye infusion. Mainstream demonstrated by bolus injection upstream of the model.

Figure 3. Separated boundary layer flow as demonstrated in our carotid model. Note region of separated flow encircling the bifurcation and deviation of mainstream flow. This condition occurs just before that shown in figure 7 ($Q_{ex}/Q_{com} = 0.28$).
nature of blood flow, the instantaneous Reynolds number varied between approximately 200 and 1200. Balasubramanian has shown in an earlier study that the basic characteristics of the flow field at the carotid bifurcation are only weakly dependent on the upstream Reynolds number in the range of 200 to 1200 under steady flow conditions. As the main purpose of this investigation was to study the effect of flow split between the two branches in a more realistic model, the bulk of the experiments described in this study were performed at an upstream Reynolds number of 500. Investigations were also conducted at other values of the Reynolds number between 200 and 1200. The flow split was varied continuously from the condition of no flow in the external carotid to the case when all of the flow was through this branch. The Reynolds number and flow split were obtained from the flow rates through the branches determined by timed collection.

For observation of the flow field under pulsatile conditions, the Harvard pulsatile pump was operated at frequencies between 50 and 120 per minute. Stroke volume for the pump was set at 1.5 ml, a value scaled from a volume comparable to the volume change in the human carotid during one heart cycle. The Reynolds number was derived from the measured mean flow rate.

Results

At a steady inlet Reynolds number of 500, the ratio of flow exiting from the external carotid branch to the inlet flow \( \frac{Q_{\text{ext}}}{Q_{\text{com}}} \) was varied. When there was no flow through the external carotid, dye injected along the wall of the sinus and common carotid opposite the external carotid remained attached with no separation (figure 4). Dye injected into the proximal ports (A and B) of the external carotid showed a complex three-dimensional eddy pattern and a drainage of the fluid into the carotid sinus. Dye injected into the external branch beyond the flow divider simply pooled outside the injection ports, indicating virtual stagnation. When the fraction of flow through the external carotid was increased so that \( \frac{Q_{\text{ext}}}{Q_{\text{com}}} = 0.17 \), a large region of separated flow was observed in the sinus on the side opposite the external carotid, extending from a point proximal to the flow divider to the proximal end of the sinus neck. This was manifested by retrograde flow along the wall of the sinus (figure 5). Dye injected through the ports on the external carotid side of the common carotid flowed completely into the external carotid, with a thickened layer across the origin of the external carotid. A very small region of separation with retrograde near-wall flow was seen in the middle of this zone.

As \( \frac{Q_{\text{ext}}}{Q_{\text{com}}} \) increased further, the separation zone in the sinus began to extend in a circumferential manner so that at \( \frac{Q_{\text{ext}}}{Q_{\text{com}}} = 0.28 \), a shell of separated fluid enveloped the carotid sinus (figures 6 and 7). The separated zone extended from the origin of the internal carotid to a point along the sinus neck, with the fluid shell beginning to extend into the external carotid. The point from which retrograde near-wall flow occurred had moved further downstream. Under these conditions, a region of very slow moving, nearly stagnant fluid was present along the wall of the sinus opposite the origin of the external carotid (figure 7). Dye injected along the external carotid remained attached to the wall, and the previous small region of separation had disappeared.

As \( \frac{Q_{\text{ext}}}{Q_{\text{com}}} \) increased further, the velocity of the retrograde and circumferential flow within the separated region increased, and the point from which retrograde near-wall flow occurred moved further downstream nearly to the end of the sinus. The circumferential pattern extended around the entire bifurcation to the far wall of the external carotid. Within the separation, in the center of the sinus (figure 8), a region of complex helical flow developed. The presence of this helical flow has been previously identified by Balasubramanian et al.

Once the separated region formed, the separation point moved only a short distance upstream as \( \frac{Q_{\text{ext}}}{Q_{\text{com}}} \) increased. Similarly, the reattachment point (the point from which retrograde near-wall flow occurred) moved downstream to the distal end of the sinus neck, but never beyond. Thus, this flow separation was limited to the sinus. The small region of separated flow present at the origin of the external carotid rapidly decreased in extent as \( \frac{Q_{\text{ext}}}{Q_{\text{com}}} \) increased and vanished when \( \frac{Q_{\text{ext}}}{Q_{\text{com}}} > 0.24 \) (figure 6). The rapidly moving central flow stream impinged directly on the flow divider at the bifurcation at \( \frac{Q_{\text{ext}}}{Q_{\text{com}}} = 0.28 \), with a portion flowing into the external branch. This central flow stream appeared to be deviated toward the flow divider as the separated region in the sinus increased in extent.

Under steady flow conditions, the \( Q_{\text{ext}}/Q_{\text{com}} \) at which separation in the sinus occurred decreased with increasing inlet Re. At an inlet Re = 1000, the respective transitions in figures 5–7 became more difficult to determine, and occurred approximately at \( Q_{\text{ext}}/Q_{\text{com}} = 0.1, 0.19 \), and 0.24 respectively. Otherwise, the separation characteristics changed little as the inlet Re was varied over the range 200 to 1200.

Preliminary studies with the pulsatile pump in operation were made at frequencies between 50 and 120 min". These studies indicated that the basic flow field characteristics are maintained from steady flow conditions. The region of sepa-
CAROTID FLOW STUDIES  LoGerfo et al.  239

Figure 4. Flow pattern at $Q_{ex}/Q_{com} = 0.00$. No outflow through the external carotid. Dye that is injected through ports a, b, c, and d drilled into the internal and common carotid on the side opposite the external carotid origin flows downstream without separation. The region of separated flow in the external carotid (ports A, B) extends to the flow divider, while the dye from ports C and D pools. Mainstream flow exhibits no deviation.

Figure 5. Flow pattern at $Q_{ex}/Q_{com} = 0.17$. Slow moving separated boundary layer flow in carotid sinus opposite external carotid, extending from a point opposite the external carotid origin to the proximal end of the sinus neck. Dye infused into the origin of the external carotid forms a small zone of separated flow. Mainstream flow deviates toward the flow divider.

Figure 6. Flow pattern at $Q_{ex}/Q_{com} = 0.24$. Dye injected into carotid sinus opposite external carotid origin separates from sidewall opposite external carotid origin, reattaching along the sinus neck. A “saddle-like” region of slow eddying flow within the separation forms across from the external carotid origin and begins to encircle the bifurcation. Dye infused along the external carotid origin remains attached to the wall forming a thickened boundary layer. Mainstream flow deviates to flow divider, but no flow as yet enters the external carotid.

Figure 7. Flow pattern at $Q_{ex}/Q_{com} = 0.28$. Separated boundary layer flow in the carotid sinus opposite the external carotid extends from a point across from the proximal end of the external carotid origin to a point along the sinus neck. A very slow, nearly stagnant region of flow is located near the proximal end of the separated region. A ring of separated flow encompasses the bifurcation and flows into the external carotid. Dye infused into near wall streamlines on the external carotid side of the model demonstrates no separation. Mainstream flow impinges on the flow divider with a portion entering the external carotid.

Figure 8. Flow pattern at $Q_{ex}/Q_{com} = 0.55$. Separated flow in the carotid sinus extends from a point across from the proximal end of the external carotid origin to the distal end of the sinus neck. This region of separated flow forms a fast moving helical pattern in the middle of the carotid sinus terminating through a shell about the bifurcation into the external carotid. The mainstream flow impinges upon the flow divider, splitting into the external carotid and along the carotid sinus wall adjacent to the external carotid origin.
rated flow previously described remained intact, and oscillated forward and backward during each cycle. Further comprehensive investigation of pulsatile flow through the model carotid is currently in progress.

Discussion

In these experiments, we have attempted to simulate the fluid dynamics of the human carotid bifurcation in an in vitro model. Arterial flows involve pulsatile flow of blood through complex elastic tubes. Although blood has a nonlinear viscosity-shear rate relationship, it can be treated with little error as a Newtonian Fluid when flowing through the major arteries. Although wall elasticity might be important with regard to wave propagation and reflections, it has only a small effect on velocity profiles. The complex geometry of the bifurcation and pulsatile nature of the flow are factors that influence the hemodynamics significantly. While the former of these has been adequately modeled in the study, investigation of steady flow is considered as a forerunner of the more realistic pulsatile flow studies currently in progress. The present results demonstrate that the basic features of the steady flow field are maintained during pulsatile flow.

The profound effect of altering \( \frac{Q_{\text{ex}}}{Q_{\text{com}}} \) on the flow field is evident in this study. Of particular interest is the flow split (\( \frac{Q_{\text{ex}}}{Q_{\text{com}}} = 0.28 \)) at which the extent of the sinus separation zone is greatest. Measurements of blood flow through the carotid arteries indicate that the flow split in humans is about 0.30, 11, 12 Thus, our present study indicates that under resting flow conditions there is separation and near stagnation of blood in the carotid sinus opposite the flow divider. In the angiogram corresponding to our model (figure 1), the site of plaque formation is in the sinus opposite the flow divider, while the apex and adjacent sinus wall are strikingly free from deposits.3, 4, 17

Our findings support the view that atherosclerotic plaque tends to form in regions of separation and low wall shear. Caro et al.18 have argued that low shear rates adversely affect near-wall biochemistry and initiate plaque formation. The low shear is thought to result in an accumulation of intimal metabolic products and a deprivation of nutrients. Flow separation has also been shown to be a site for accumulation of platelet aggregates in vitro.19 If endothelial injury occurred as a result of the metabolic effects of low shear, the platelet aggregates could adhere to the subendothelium and stimulate smooth muscle proliferation.20

Our findings and the clinical studies noted earlier also indicate that lesions are not produced due to endothelial injury caused by high shear stress. Balasubramanian et al.15 presented data indicating wall shear stress magnitudes at the flow divider of their carotid model on the order of 100 dynes/cm², and we would expect similar values for our model. This is not expected to be harmful, however, as a level of about 400 dynes/cm² is required to produce endothelial injury as reported by Fry.21

In our model, there are two regions of separation — one in the sinus and the other in the external carotid. At a clinical flow split of 0.30, however, the separation in the external carotid vanished, and the region of greatest stagnation was in the sinus across from the external carotid origin. If plaque formation begins in this region, it would then narrow the orifice of the internal carotid, which in turn could alter the flow split and increase \( \frac{Q_{\text{ex}}}{Q_{\text{com}}} \). As a consequence, even if the separation at the origin of the external carotid had existed, it would decrease, and the plaque would progress primarily in the sinus region with only a lesser deposit in the external carotid. This is at least a theoretical explanation for the observed localization of plaque in figure 1. Further studies have been planned to study the influence of plaque development on the flow field.

The marked dependence of the separation region on the flow split might explain why some individuals with atherosclerosis develop a carotid plaque and others do not. The precise flow split in a given individual probably varies with the individual anatomy of the circle of Willis and the vertebrobasilar system. In addition, carotid flow has been shown to be highly variable with position changes such as turning the head.12 The flow split at a given carotid bifurcations may therefore range above or below that of the straight bifurcation (below 0.17 vs above 0.19). Of particular note were the reattachment points of the separation zone in the sinus and the separation in the external carotid. The reattachment point of the straight model continued to move downstream until very high flow splits were reached. In the carotid model, the separation was confined to the sinus region and did not extend downstream.
CAROTID FLOW STUDIES  LoGerfo et al.  241

References

13. Burton AC. Physiology and biophysics of the circulation, 2nd ed. Chicago: Year Book Medical Pub, 1975

Index Terms: carotid bifurcation • fluid dynamics • atherosclerosis
Flow studies in a model carotid bifurcation.
F W LoGerfo, M D Nowak, W C Quist, H M Crawshaw and B K Bharadvaj

doi: 10.1161/01.ATV.1.4.235

Arteriosclerosis, Thrombosis, and Vascular Biology is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1981 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/1/4/235

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/