Effect of Stenosis on Wall Motion
A Possible Mechanism of Stroke and Transient Ischemic Attack

Richard L. Binns and David N. Ku

The mechanism by which atherosclerotic plaque causes stroke and transient ischemic attack is not fully understood. One possibility is that the plaque stenosis may set up hemodynamic conditions causing local arterial wall collapse. Arterial wall collapse may, in turn, affect the integrity of the plaque. This study was designed to define the effects of stenosis on the production of arterial wall collapse using a latex tube model. Stenoses ranging up to 81% by diameter were tested in a Starling resistor chamber under pulsatile pressure conditions upstream of the tube. Increasing the degree of stenosis progressively decreased the external pressure necessary to produce collapse, from 37 mm Hg with the 0% stenosis to 24 mm Hg for the 81% stenosis. The stenoses greater than 70% produced a new phenomenon of “systolic wall collapse” just distal to the stenosis. The maximum diameter decrease was 2.83 mm from the baseline diameter of 6.41 mm. Cyclic wall motion just downstream of the stenosis increased with the increased degree of stenosis from 0.34 mm at 0% stenosis to -1.28 mm at 75% stenosis. The phenomena are discussed in terms of simplified Bernoulli pressure drops. We conclude that local arterial stenosis can produce conditions favorable for wall collapse and increased wall motion at physiologic pressure and flow. This collapse may be important in the development of atherosclerotic plaque fracture and subsequent thrombosis or distal embolization.


Stroke is the third leading cause of morbidity and mortality in the United States and accounts for over 200,000 deaths a year. Extracranial atherosclerotic cerebrovascular disease localized within the carotid bifurcation is the cause of stroke and transient ischemic attack (TIA) in the majority of these patients. The exact mechanism by which atherosclerotic plaque causes hemispheric stroke and TIA remains controversial and unresolved. However, recent evidence strongly favors a plaque fracture-embolization hypothesis. One possible etiology is that the local stenosis produced by an atherosclerotic lesion may cause mechanical conditions favorable for plaque fracture and distal embolization.

This study was designed to investigate the effect of stenosis on wall motion and collapse in a latex tube model. Much work has been reported on the collapse of veins and other thin-walled tubes within Starling resistor chambers. These articles identify the critical controlling factors such as transmural pressure, upstream and downstream pressure, and longitudinal tension. Additionally, they report choking, the waterfall phenomenon, and dynamic behavior such as flutter. Most of these articles address problems of flow limitation and instability in veins, airways, or ureters. The experimental setup or boundary conditions test an elastic tube of uniform diameter. None of these articles specifically address the problem of wall excursion and collapse distal to a high grade stenosis. However, Elad et al. report on the increased likelihood of transition to supercritical flow with a local area constriction in a theoretical model of the pulmonary system, suggesting that stenoses may cause collapse.

Santamore and Walinsky have presented radiographic evidence of a decrease in arterial diameter distal to a snare-type stenosis of approximately 97% of the diameter. In their in vitro tests, they created a decrease in the distal peripheral resistance, but they did not vary the external pressure. Developing a theoretical model of the compliant arterial stenosis, Santamore and Bove described diameter and circumferential decreases, which may result from decreases in intraluminal pressure. Their analysis did not address the cases when the transmural pressure becomes negative with collapse. Schwartz et al. have also discussed the possibility of passive narrowing from decreases in lateral pressure when the distal pressure is lowered but did not present experimental evidence of diameter changes. In this article, we report physiologic conditions under which arterial wall collapse may occur distal to an atherosclerotic stenosis.

Methods

A drawing of the general experimental system is shown in Figure 1. The pulsatile continuous flow system was comprised of three constant pressure head reservoirs and a recirculating pump. This arrangement allowed for the precise and independent setting of the inlet and outlet pressures. The reservoirs consisted of partitioned chambers, which allowed one half of the chamber to contain a
constant height of fluid. The height of each reservoir above the experimental chamber determined and maintained pressure on the inlet and outlet side of the tubing. An on/off solenoid valve attached to the highest inlet reservoir produced pulsatile flow by alternating inlet pressure between the higher and lower reservoirs, maintaining the inlet pressure at 100/60 mm Hg. A one-way flutter valve prevented reflux into the lower inlet reservoir during flow from the higher reservoir. The pulse cycle was one cycle per second, with systole occupying one third of the period.

Flow in a human carotid was simulated by using latex rubber tubing to model the compliant artery because the elastic characteristics of this tube are similar to normal arteries under physiologic pressures. The test section consisted of an 18 cm length of 6.6 mm diameter latex penrose tubing mounted on 6.5 mm diameter glass tubing within the experimental chamber. The thickness of the latex tubing measured 0.3 mm. The tubing was mounted within the experimental chamber, with just enough longitudinal tension to keep the tube from sagging. The latex tube used in these experiments had an elastic modulus (measured by pressure-diameter tests) of $11 \times 10^5$ Pa over the relevant range of pressures used and a Poisson’s ratio of 0.5.

The system fluid consisted of a glycerine-water solution adjusted to an absolute viscosity of 4 centipoise, similar to blood. This gave a kinematic viscosity of 0.034 cm$^2$/sec. The mean upstream Reynolds number ($UD/\nu$) was maintained between 130 and 490, where $U$ is the average velocity, $D$ is the nominal diameter, and $\nu$ is the kinematic viscosity.

The stenosis was produced by two different methods. The first method produced an external stenosis similar to that described by Lowenstein et al. This method employed a caliper-string arrangement, which acted as a snare around the elastic tube. By measuring the distance between the arms of the caliper, the external diameter of the stenosis was quantified. Stenoses of 50% and 68% diameter narrowing were produced with this method. The second method involved the intraluminal insertion of an axisymmetric, rigid, straight-edged orifice made of polyvinylchloride plastic. The stenosis was held in place by a ligature. The length of the orifice measured 0.76 mm. Stenoses of 69%, 75%, and 81% diameter narrowing were produced in this fashion. All stenoses were placed 5 cm downstream from the inlet end of the experimental tubing.

The flow was continuously measured with a Transonic (model T101) in-line ultrasonic flow probe accurate to within 1% of total flow. The peak systolic, end-diastolic, and mean flow rates were manually recorded for each experimental arrangement. The mean flow was determined over 10 seconds. The wall motion and collapse of the elastic tube were recorded on a JVC GR-555U videotape recorder.

Experiments were carried out at each of six different downstream outlet pressures ($P_{out}$) ranging from zero to 100/60 mm Hg.
Figure 2. Wall motion distal to a 75% rigid stenosis showed an inward collapse during systole at the collapse pressure while expansion occurred proximal to the stenosis. The point of maximum collapse occurred 4 diameters downstream of the stenosis. At lesser degrees of stenosis, wall expansion occurred during systole.

25 mm Hg in increments of 5 mm Hg. These outlet pressures were chosen to simulate conditions found in patients with low carotid stump pressure. Carotid stump pressure is the pressure measured in the artery distal to an arteriotomy during carotid endarterectomy and is used to determine the level of collateral blood supply to the distal internal carotid. This stump pressure would be close to the pressure downstream of the stenosis in our model. At each outlet pressure, the external pressure around the experimental tubing (Peout) was increased from 0 mm Hg in increments of 5 mm Hg. The external pressure at which collapse first occurred was designated the collapse pressure. This point was reached when the diameter of the vessel was clearly reduced from the initial resting diameter with no external pressure applied (i.e., a transmural pressure of 60 mm Hg).

Wall motion, defined as the systolic diameter minus the diastolic diameter, was measured 2 cm downstream from the stenosis. Measurements were taken from the video monitor with calipers and were corrected for magnification error. Flow rate, collapse pressure, and wall oscillation were initially recorded for baseline conditions of no stenosis. We have termed this phenomenon "systolic wall collapse." For external pressures greater than the collapse pressure, the tube was collapsed during both systole and diastole, but the degree of collapse was greater during systole. For external pressures less than the collapse pressure, normal systolic wall expansion occurred throughout the tube.

The effect of collapse could be described as the change in diameter measured 2 cm downstream of the stenosis as compared with the usual systolic diameter with no external pressure (6.41±0.48 mm). These values are listed in Table 2. It can be seen that a collapsing external pressure caused an average 0.55 mm decrease for stenoses up to 70%. However, the 75% and 81% stenoses caused a much greater diameter change of up to 2.83 mm. This systolic diameter decrease is much greater than expected for transmural pressure merely reaching to zero. Pulsatile pressures created a large-scale wall motion, which could be quantified by the cyclic diameter changes. These values for wall motion are listed in Table 2. Wall motion increased from the baseline 0% stenosis value of 0.34 mm to a large value of -1.28 mm with a 75% rigid stenosis. The large amount of wall excursion was readily visible at the collapse pressure, and the negative sign indicates that maximum collapse occurred during systole. Note that the wall motion of the 68% snare stenosis was much larger than the 69% stenosis, a change that was probably due to the stenting effect of the rigid insert. Superimposed on the large-scale wall motion, there was a transient 50 to 100 msec, high-frequency wall oscillation at the beginning of each contraction and expa-
Table 1. External Collapse Pressure and ΔP for Each Percent Stenosis at Each Outlet Pressure

<table>
<thead>
<tr>
<th>P_{out} (mm Hg)</th>
<th>0%</th>
<th>50%</th>
<th>68%</th>
<th>69%</th>
<th>75%</th>
<th>81%</th>
</tr>
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<tbody>
<tr>
<td>25</td>
<td>45</td>
<td>45</td>
<td>38</td>
<td>42</td>
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<td>28</td>
<td>25</td>
<td>23</td>
<td>18</td>
<td>17</td>
</tr>
<tr>
<td>0</td>
<td>28</td>
<td>25</td>
<td>20</td>
<td>19</td>
<td>10</td>
<td>12</td>
</tr>
</tbody>
</table>

Mean ΔP: 24.5, 22.2, 18.0*, 17.8*, 12.7*, 12.0*

Standard deviation: 3.2, 1.9, 2.4, 0.8, 1.5, 0.6

Values are given in mm Hg.
ΔP = P_{stenosis} - P_{outlet}.
*Significantly less than 0% at p<0.0005.

Table 2. Diameter Change and Wall Motion Measured 2 cm Downstream of the Stenosis for Collapse Pressure at Different Stenoses

<table>
<thead>
<tr>
<th>% stenosis</th>
<th>Diameter change (mm)</th>
<th>Wall motion (mm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>0.71±0.60</td>
<td>0.34±0.12</td>
</tr>
<tr>
<td>50</td>
<td>0.46±0.76</td>
<td>0.79±0.15</td>
</tr>
<tr>
<td>68</td>
<td>0.80±0.68</td>
<td>1.08±0.54</td>
</tr>
<tr>
<td>69</td>
<td>0.22±0.08</td>
<td>0.56±0.16</td>
</tr>
<tr>
<td>75</td>
<td>2.83±0.42</td>
<td>-1.28±0.19</td>
</tr>
<tr>
<td>81</td>
<td>1.78±0.20</td>
<td>-0.62±0.19</td>
</tr>
</tbody>
</table>

Discussion

It is well known that arteries, being thin-walled elastic tubes, are collapsible. Santamore and Walinsky have demonstrated that arterial diameter changes can occur with a high-grade stenosis. The forces involved in the collapse of arteries distal to a stenosis may be understood from a simple model. By the Bernoulli principle, flow through a stenosis causes a drop in lateral pressure (static pressure) within the throat of a stenosis even though total pressure remains the same. As velocity increases through a stenosis, the static pressure falls proportional to velocity squared. Figure 4 is a graphic presentation of the theoretical systolic and diastolic pressures within the throat of a stenosis at two different steady flow rates. At a critical stenosis between 70% and 88% by diameter, which depends on flow rate, the systolic pressure in the throat can be less than the diastolic pressure. Both systolic and diastolic pressures quickly become negative with small increases in stenosis due to the velocity squared term in the Bernoulli equation. The higher the flow rate, the less the degree of stenosis necessary to produce this negative pressure. This theoretical pressure curve additionally explains systolic wall collapse where the direction of wall motion is exactly opposite of the usual systolic expansion. With high-grade stenoses, the negative pressures developed from the high velocities of systole tend to cause collapse of the vessel just distal to the stenosis with re-expansion during diastole.

Collapse of the normally circular wall is a buckling phenomenon in which the external pressure exceeds the internal pressure. The transmural pressure required to buckle the artery has been analyzed by Fung as:

ΔP_{critical} = E h^2(1-ν^2)R^3

where E is the modulus of elasticity, h is the thickness, ν is Poisson's ratio, and R is the radius of the tube. The theoretical ΔP_{critical} would be approximately 3 mm Hg for the carotid artery or latex tube used in these experiments.

Shapiro developed the inviscid equations of fluid dynamics to provide a more comprehensive description of flow...
through thin-walled collapsible tubes. Elad et al. used these equations to describe local area changes in a model of bronchial flow. Our experimental findings support their results: that flow through a local stenosis should tend to critical flow at the throat of the stenosis with supercritical flow beyond. Cancolle and Pedley used a mathematical model, which included the effects of longitudinal tension and viscous losses from flow separation, to explore the mechanism of spontaneous collapse and wall oscillation. Each of these approaches may have a bearing on future analyses of the problem illustrated in this paper.

A number of investigators have shown that low distal pressures associated with low distal resistance can result in an increase in stenosis resistance. Although several factors may contribute, one possibility is that the arterial diameter may decrease passively. Certainly, the large-scale collapse of the artery distal to the stenosis, which is described here, could have a great effect on the in vivo resistance by choking as well as by viscous head loss. Our flow measurements indicate this trend, but the data presented here are insufficient to address this issue fully.

A question arises as to the magnitude of normal physiologic external pressure around the carotid artery. One might assume that the external pressure is equivalent to the interstitial tissue pressure. Tissue pressure has been measured at from 7 mm Hg to 13 mm Hg. Due to the anatomic arrangement of the internal jugular vein and carotid artery within a common fascial sheath, volume changes in the jugular vein may directly impinge on the external surface of the carotid artery. We have preliminary measurements in humans that show that Valsalva's maneuver and cough can cause sharp rises in jugular venous pressure to greater than 50 mm Hg. These transient pressure increases within the carotid sheath may augment the external pressure around the carotid artery and promote artery collapse under these circumstances.

Physiologically, the downstream pressure of the Starling resistor may be analogous to the carotid stump pressure as measured during carotid endarterectomy. Hahn and Hettiger reported a carotid stump pressure between zero and 25 mm Hg in 78 of 418 patients. Thus, the low downstream pressures reported here are within physiological range. If we assume an external pressure within the carotid sheath of 25 mm Hg, then our results would indicate no conditions favorable for collapse at 0% stenosis. However, collapse is possible for a 68% stenosis with a stump pressure of 7 mm Hg. The 81% stenosis could cause collapse with a stump pressure of 13 mm Hg. Higher grade stenoses should induce collapse with an even higher carotid stump pressure, provided viscous losses are not excessive.

Increased wall motion may be a function of the method used to produce the stenosis. With the external string stenosis, the tube wall within the stenosis is capable of collapsing. The rigid internal stenosis increased the wall motion at 75% stenosis, but decreased it at 81% stenosis. The rigid internal stenosis holds the tube open at the stenosis and longitudinal tension counteracts the inward force of collapse for a distance downstream of the stenosis, causing the downstream location of collapse. Due to the common decrease in compliance of atherosclerotic plaque, the rigid stenosis may be a better model of atherosclerotic narrowing than the string stenosis.

The finding of wall oscillation with moderate stenosis may or may not have clinical significance. The high-frequency transient wall oscillations were most pronounced during the beginning of systole and diastole and were probably induced by the sudden step changes in pressure similar to an undamped oscillator by our flow system. The changes in flow between systole and diastole would be expected to slightly alter the pressures as well. This quantity should be of the order \(-\frac{dQ}{dt}\) from the unsteady Euler equation. For this experiment, this quantity is less than 1 mm Hg. Additionally, wall tethering was not included in this model. While wall oscillation may produce additional stress, fatigue, and fracture at the plaque artery border, the strong negative pressure within the throat of a stenosis with the subsequent larger scale wall collapse should be emphasized.

The clinical motivation for this research stems from the clinical observation that atherosclerosis often becomes

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### Table 3. Peak Systolic, End-Diastolic, and Time-Averaged Mean Flow Rates for All Pressures

<table>
<thead>
<tr>
<th>Flow rate</th>
<th>0%</th>
<th>50%</th>
<th>68%</th>
<th>69%</th>
<th>75%</th>
<th>81%</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Systolic</strong></td>
<td>544±65</td>
<td>538±33</td>
<td>408±31*</td>
<td>442±40*</td>
<td>355±14†</td>
<td>267±68‡</td>
</tr>
<tr>
<td><strong>Diastolic</strong></td>
<td>387±66</td>
<td>381±54</td>
<td>248±32*</td>
<td>313±41†</td>
<td>196±241</td>
<td>143±14‡</td>
</tr>
<tr>
<td><strong>Mean</strong></td>
<td>434±76</td>
<td>428±62</td>
<td>275±36*</td>
<td>350±46*</td>
<td>243±21†</td>
<td>177±12‡</td>
</tr>
</tbody>
</table>

Values are given in cc/min.

*Significantly less than 0% at p<0.0005, †significantly less than 69% and 0% at p<0.0005, ‡significantly less than 75%, 69%, and 0% at p<0.0005.

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**Figure 4.** Theoretical graph of systolic and diastolic static pressure (mm Hg) within the throat of a stenosis versus percent stenosis at two different steady flow rates. At a critical stenosis, the systolic pressure in the stenosis becomes less than diastolic pressure. Higher flow rates produce negative pressure at lesser degrees of stenosis.
EFFECT OF STENOSIS ON WALL MOTION

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symptomatic when the lesion stenosis is greater than 75% by diameter. For the carotid artery, the likelihood of stroke is almost six times greater with high-grade stenoses than with lower-grade stenoses.\(^2\) Soft plaques are also more dangerous than calcific plaques.\(^3\) The terminal event is usually either total thrombotic occlusion or plaque fracture with distal embolization.\(^2,\)\(^3\) Arterial wall collapse may be partially responsible for plaque fracture with subsequent distal embolization. It is also possible that collapse of the artery may injure the endothelium, thereby promoting local thrombus formation. The resultant wall motion and oscillation during partial collapse may have a bearing on the etiology of intraplaque hemorrhage, plaque ulceration, angiographically observed “spasm,” and poststenotic dilatation. A mechanism for transient ischemia or stroke could be independent of absolute blood flow limitation.

In conclusion, we have found some physiologic conditions in which a local stenosis produces wall collapse in a latex tube model. Wall collapse can occur with high-grade stenosis combined with either low carotid stump pressure or elevated external pressure, as from Valsalva’s maneuver and coughing. High-grade stenosis can also produce a phenomenon of paradoxical systolic wall collapse. This cyclic wall collapse may contribute to atherosclerotic plaque fracture with subsequent embolization or thrombosis. The fact that collapse occurs only under the specific conditions of external and internal pressure may explain the intermittent nature of symptoms in patients with stroke or TIA. Further experimental work on the effect of stenosis on the in vivo collapse of human arteries needs to be done. The effect of stenosis contour and plaque stiffness on collapse pressure also requires further investigation.

References


Index Terms: stenosis • stroke • arterial wall • plaque • atherosclerosis • collapse • embolism • thrombosis
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