Hemodynamics of Arterial Stenoses at Elevated Flow Rates

DONALD F. YOUNG, NEAL R. CHOLVIN, RICHARD L. KIRKEEIDE, AND ALLAN C. ROTH

SUMMARY This study is concerned with the pressure drop that develops across an arterial stenosis, with particular emphasis on the effect of the stenosis at high blood flow rates induced by a locally administered vasodilator drug. Stenoses, ranging in severity from 55.7% to 91.0% reduction in lumen area were artificially induced in the femoral and carotid arteries of large mongrel dogs. Instantaneous flow rates and pressure drops were measured over a wide range of flow conditions. Mean velocities varied from 3.9 to 88.8 cm/sec. Experimental data support the applicability of a relatively simple equation for predicting the pressure drop over this wide range of velocities and stenosis geometries. Results show that blood flow through a particular artery can increase by a large factor, in the range of 4–5, under conditions of vasodilation with a corresponding large decrease in pressure distal to the stenosis. The pressure drop increases in a nonlinear manner with velocity and thereby accentuates the importance of the stenosis at elevated flow rates. We suggest that a critical stenosis be defined in terms of its effect on maximal flow rather than resting flow.

THE DEVELOPMENT of a stenosis in a major artery may significantly alter the blood supply to the peripheral vascular beds supplied by the artery. Since the early work of Mann et al., much attention has been given to this problem, with special consideration given to the concept of the "critical stenosis," which generally has been defined as one for which a small, further increase in the severity of the stenosis will cause a significant reduction in blood flow rates. We suggest that a critical stenosis be defined in terms of its effect on maximal flow rather than resting flow.
flow. This definition of a critical stenosis is not complete since the effect of a stenosis is influenced not only by the severity of the stenosis, but also by other geometric factors such as length, the peripheral resistance of the distal beds, and collateral flow.  

The interaction between the flow through a stenosed artery, collateral flow, and peripheral resistance can be illustrated through the consideration of the simplified, but useful, hydraulic model shown in Figure 1. For this model it is assumed that the resistance of all peripheral beds distal to the stenosis can be considered as a lumped resistance $R_p$. The time-averaged flow through the stenosis is $Q_s$, net collateral flow is $Q_c$, and the flow actually supplied to the distal beds is $Q = Q_s + Q_c$. The arterial pressure proximal to the stenosis is $p_a$, and the venous pressure, $p_v$, will be assumed to be zero. The bar over a symbol signifies its time-averaged value, i.e., the average value of the quantity taken over one cardiac cycle.

In a normal vessel the commonly used relationship between pressure, flow, and peripheral resistance is

$$Q = \frac{p_a}{R_p}, \quad (1)$$

which demonstrates that the flow is a function of the driving pressure $p_a$ and the resistance $R_p$. Furthermore, under normal flow conditions the collateral flow is negligible because the collateral resistance is large compared to the resistance of an unobstructed artery.

As a stenosis develops, a pressure drop $\Delta p = p_a - p_i$ is created across the stenosis, so that the driving pressure for flow to the peripheral beds is now $p_i$ rather than $p_a$, and it is clear that the flow to the peripheral beds will be reduced unless the peripheral resistance can be reduced to compensate for the reduction in driving pressure or adequate collateral flow develops. In equation form

$$Q = \frac{p_i}{R_p} = \left(\frac{p_a}{R_p}\right)\left(1 - \frac{\Delta p}{p_a}\right), \quad (2)$$

and this relationship shows how the pressure drop across the stenosis interacts with the peripheral resistance and arterial pressure to control the flow to the distal beds. A complicating feature of Equation 2 is the fact that the term $\Delta p$ is a function of the flow through the stenosis. Thus, the effective resistance of the stenosis, as evidenced by $\Delta p$, changes the flow and, in fact, $\Delta p$ increases in a nonlinear fashion with flow.  

This simplified analysis shows that under resting conditions it is possible to maintain normal flow to the peripheral beds in the presence of a stenosis by simply reducing the peripheral resistance. Thus, as is well known, the effect of a mild stenosis on resting flow is negligible. However, as the stenosis becomes more severe, $\Delta p$ for a given flow increases and at some critical value of percent stenosis (percent reduction in lumen area) a further increase in the severity of the stenosis will cause the flow to be reduced dramatically because the peripheral resistance cannot be reduced sufficiently to offset the large pressure drop across the stenosis.

Although the effect of a stenosis on resting blood flow is of importance, it is clear that the limiting influence of the stenosis will occur at elevated flow rates, because the pressure drop caused by the stenosis increases with increasing flow. Under conditions of exercise in which maximal blood flow to a particular vascular bed is required, the stenosis can easily become the limiting factor in the system. In effect, a stenosis reduces the vascular bed reserve, i.e., it reduces the ratio of the maximal flow to a peripheral bed to the resting flow. Thus, a more meaningful index of the effect of a stenosis should be based on its effect on the vascular bed reserve. The present study was undertaken to investigate (1) the pressure loss across arterial stenoses of varying severities at elevated flow rates, and (2) the corresponding effect of the stenosis on the vascular bed reserve.

**Methods**

Stenoses were induced in the superficial femoral and common carotid arteries of large dogs (23–35 kg) by inserting hollowed cylindrical plugs into the artery. With this procedure the geometry of the stenosis is well defined, and the severity of the stenosis can be controlled by simply selecting a plug having the appropriate lumen diameter ($d$) relative to its outside diameter ($D$). This technique is the same as that used by Young et al. For all tests the ratio of stenosis length ($L_s$) to diameter $D$ was 2.0. The dogs were anesthetized with sodium pentobarbital (30 mg/kg, iv), heparinized, and allowed to breathe room air spontaneously through an endotracheal tube. Twelve dogs were used in the study: six dogs for the femoral artery experiments and six for the carotid artery experiments.

The instantaneous flow rate was obtained with a Biotronex BL-610 electromagnetic flowmeter with noncannulating flow probes. The zero flow baseline was obtained by occluding the artery distal to the flow probe, and the baseline was checked several times during the course of the experiment. Flowmeter calibration was obtained in situ at the conclusion of each experiment by cannulating the artery and measuring the time required for various volumes of blood to flow into a graduated cylinder. The dynamic response of the flowmeter was checked electronically and found to be flat to approximately 30 Hz with a linear phase shift of 3.6°/Hz. To increase the flow through the femoral artery, a small side branch, distal to the stenosis, was cannulated so that a vasodilator drug (acetylcholine) could be infused at various rates.

For each experiment involving the left femoral artery the vessel was exposed and two side branches, one proxi-
nal and the other distal to the site of the stenosis, were cannulated for pressure measurements. The distance between the two side branches ranged from 22 to 68 mm, with an average value of 46 mm. Pressures were recorded with two Statham P23Db transducers, and the pressure drop was obtained by electronically subtracting the two pressure signals. The pressure transducers were connected to the artery with relatively stiff catheters, and the pressure transducer-catheter system had a natural frequency in excess of 35 Hz. Before each test the pressure-measuring system was calibrated statically, and, subsequently, the catheters were connected to a common dynamic pressure source to ensure that both pressure transducers were properly balanced. For this purpose the contralateral femoral artery was cannulated, and both pressure catheters were connected to this common pressure source. Both pressure-measuring systems were, therefore, exposed to the same dynamic pressure; if they were properly matched, no differential pressure was observed. Small air bubbles in either system caused an imbalance; thus, before use the system was flushed repeatedly until no significant differential pressure was detected.

Stenoses were induced in the right common carotid artery of six large dogs in the same manner used for the femoral artery. Because there are no side branches along the common carotid suitable for cannulation for pressure measurement, specially designed pressure taps were used. A puncture was made in the artery and a small hollow tube with a flared end was inserted into the artery. The flared surface was held in place against the inner wall of the artery so that an "artificial side branch" was created which gave minimal flow disturbance. Pressure drop measurements were then made using the same procedures as those used for the femoral artery experiments. The distance between the two pressure taps ranged from 41 to 57 mm with an average value of 48 mm. Increased blood flow was achieved by infusing acetylcholine through a distal side branch of the common carotid while occluding the contralateral artery. The vertebral arteries were not occluded.

For each experiment, the mean and instantaneous flow rate and the pressure drop were recorded before insertion of the plug. After the plug had been inserted, the measurements were repeated. Flow and pressure data were recorded on both a Grass strip-chart recorder and an Ampex model FR-1300 magnetic tape recorder. The limiting apparent viscosity of the blood, as determined for each experiment from a Wells-Brookfield cone and plate viscometer, varied between 0.034 and 0.048 dyn/sec per cm².

Results

Typical flow rate and pressure drop recordings for the femoral artery experiments are shown in Figure 2. Before insertion of the plug creating the stenosis, recordings were made for the unobstructed artery and the corresponding rest flow waveforms, as shown in Figure 2a. After the stenosis was formed the tests were repeated, thereby obtaining data for flow through the stenotic vessel under resting flow conditions and various degrees of vasodilation (Fig. 2b, c, and d). To quantitatively characterize the flow recordings, the peak values of velocity, \( U_p \), and pressure drop, \( \Delta p_p \), were obtained from each test along with the mean velocity, \( \bar{U} \), and pressure drop \( \bar{\Delta}p \). The velocity, which represents the average velocity over the cross section, was obtained by dividing the instantaneous volume rate of flow by the unobstructed lumen area. The mean velocity is the time-averaged value of the velocity over a flow cycle, and the mean pressure drop is the corresponding time-averaged value of the pressure drop taken over a flow cycle.

UNOBSTRUCTED FLOW

Table 1 summarizes average values of velocity and pressure drop (± 1 sd), for unobstructed flow in both femoral and carotid arteries. An important dimensionless parameter frequently used to characterize fluid flow is the Reynolds number, Re, where \( Re = \rho UD/\mu \) with \( \rho \) the fluid density, \( U \) the velocity, \( D \) the lumen diameter, and \( \mu \) the fluid viscosity. Values for the Reynolds number are also tabulated in Table 1. The large increase in flow which can be obtained in a normal artery under conditions of vasodilation is clearly evident from these data (see last column in Table 1). For the femoral artery the ratio of the maximum (mean) velocity under conditions of vasodilation to the resting mean velocity was found to be approximately 5, and for the carotid artery the ratio was approximately 4. The large variation in the values for this ratio (as indicated by the standard deviation) is to be expected since the "resting state" can vary significantly from dog to dog depending on numerous factors, such as general health, the time required to prepare the subject for the trial, and the level of anesthesia at the time of measurement. Values
for the mean velocities, pressure drops, and Reynolds numbers were found to be similar for flow through the femoral and carotid arteries. However, some reversed flow during a typical flow cycle was commonly observed in the femoral artery (Fig. 2a) under resting conditions, whereas this was not the case for carotid flow. Also, as shown in Table 1, the ratio of peak velocity to mean velocity is considerably higher for flow in the femoral artery than for flow in the carotid.

FLOW WITH STENOSIS

Stenoses, ranging in severity from 55.7% to 91.0% reduction in lumen area, were induced in the femoral and carotid arteries. Tabulated values based on the data from these experiments are given in Tables 2 and 3. As expected, the presence of a stenosis generally reduced the flow through the vessel and increased the pressure loss. In Figure 3 the data are grouped according to percent stenosis and the pressure drop (both mean and peak values) is plotted vs. the corresponding mean and peak velocities. Although the pressure drop depends on several factors in addition to percent stenosis and velocity, these are the two principal variables. It can be seen from Figure 3 that the pressure drop increased dramatically with percent stenosis as the stenosis became severe, and increased in a nonlinear manner with increasing values of velocity.

For the femoral artery tests it was found that by precisely controlling the infusion rate of the vasodilator drug, the flow velocity could be increased gradually in a predictable manner. Three sets of data were obtained: (1) resting flow, (2) elevated flow at an intermediate value, and (3) maximum flow. Thus, in Table 2, three sets of data are given for each percent stenosis. Large increases in pressure drop occurred as the velocity was increased. Also, the shapes of both the flow and pressure drop waveforms changed as the peripheral beds were dilated. As shown in Figure 2 the waveforms became less pulsatile, backflow was eliminated, and, as observed from Tables 2 and 3, the

<table>
<thead>
<tr>
<th>No. of dogs</th>
<th>Artery</th>
<th>L (mm)</th>
<th>D (mm)</th>
<th>Flow condition</th>
<th>Velocity (cm/sec)</th>
<th>Reynolds number</th>
<th>Pressure drop (mm Hg)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>U</td>
<td>U_p</td>
<td>U_m/U_r</td>
</tr>
<tr>
<td>6</td>
<td>Femoral</td>
<td>46</td>
<td>4.1</td>
<td>Resting</td>
<td>22.1</td>
<td>71.3</td>
<td>3.44</td>
</tr>
<tr>
<td></td>
<td></td>
<td>±17</td>
<td>±0.4</td>
<td></td>
<td>±9.1</td>
<td>±23.1</td>
<td>±0.76</td>
</tr>
<tr>
<td>13*</td>
<td>Femoral</td>
<td>43</td>
<td>3.7</td>
<td>Resting</td>
<td>19.4</td>
<td>67.1</td>
<td>3.97</td>
</tr>
<tr>
<td></td>
<td></td>
<td>±11</td>
<td>±0.5</td>
<td></td>
<td>±12.7</td>
<td>±27.2</td>
<td>±1.06</td>
</tr>
<tr>
<td>6</td>
<td>Carotid</td>
<td>48</td>
<td>4.2</td>
<td>Resting</td>
<td>19.0</td>
<td>40.6</td>
<td>2.19</td>
</tr>
<tr>
<td></td>
<td></td>
<td>±5</td>
<td>±0.5</td>
<td></td>
<td>±4.1</td>
<td>±6.3</td>
<td>±0.37</td>
</tr>
</tbody>
</table>

\[ L = \text{distance between pressure cannulas; } D = \text{internal lumen diameter proximal to stenosis; percent stenosis} = \frac{\text{percent reduction in lumen area}}{100}; \frac{U_m}{U_r} = \text{ratio of maximum (mean) velocity under conditions of vasodilation to resting (mean) velocity; } \frac{U_m}{U_r} = \text{ratio of maximum (mean) velocity with stenosis to maximum (mean) velocity without stenosis.} \]
HEMODYNAMICS OF ARTERIAL STENOSES/Young et al.

Table 3 Summary of Data from Artificially Constricted Carotid Arteries of Dogs

<table>
<thead>
<tr>
<th>Dog no.</th>
<th>L (mm)</th>
<th>D (mm)</th>
<th>% stenosis</th>
<th>Velocity (cm/sec)</th>
<th>Pressure drop (mm Hg)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Unobstructed</td>
<td>Stenotic</td>
</tr>
<tr>
<td>7</td>
<td>49</td>
<td>5.0</td>
<td>90.7</td>
<td>7.8</td>
<td>14.3</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>12.9</td>
<td>20.4</td>
</tr>
<tr>
<td>8</td>
<td>50</td>
<td>4.1</td>
<td>90.5</td>
<td>8.0</td>
<td>16.7</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>12.6</td>
<td>19.4</td>
</tr>
<tr>
<td>9</td>
<td>57</td>
<td>4.1</td>
<td>76.2</td>
<td>12.9</td>
<td>25.5</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>17.0</td>
<td>31.0</td>
</tr>
<tr>
<td>10</td>
<td>45</td>
<td>3.8</td>
<td>72.3</td>
<td>14.5</td>
<td>30.7</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>31.0</td>
<td>44.4</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>41.0</td>
<td>53.0</td>
</tr>
<tr>
<td>11</td>
<td>41</td>
<td>3.7</td>
<td>64.4</td>
<td>20.2</td>
<td>36.6</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>36.6</td>
<td>53.6</td>
</tr>
<tr>
<td>12</td>
<td>49</td>
<td>4.4</td>
<td>58.0</td>
<td>10.9</td>
<td>19.8</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>75.0</td>
<td>98.3</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>84.2</td>
<td>112.0</td>
</tr>
</tbody>
</table>

Abbreviations as in Table 2.

ratio of the peak to mean velocity systematically decreased with a decrease in peripheral bed resistance.

The ratio of the maximum velocity obtainable with a stenosis to the resting velocity with a stenosis (\(U_{m}/U_{0}\)) is generally reduced from that found in the unobstructed artery. However, no systematic variation in this ratio was observed. It is believed that this is due to the lack of control on the resting state which is used as a base for this ratio; i.e., due to the condition of the dog at the time the resting data are obtained (the peripheral beds may be constricted or dilated depending on the level of stimulation of cardiovascular and other naturally occurring control mechanisms). A more meaningful index, both from a clinical viewpoint and experimentally, is the ratio of the maximum (mean) velocity with a stenosis to the maximum (mean) velocity without the stenosis (\(U_{m}/U_{0m}\)). This ratio is an index of the loss in maximum flow due to a stenotic obstruction and is an indicator of the effect of the stenosis under conditions of exercise and stress. It can be seen from Table 2 that this ratio was drastically reduced for the stenoses with severities equal to or greater than 78%.

In Table 3 data obtained in the carotid artery experiments are summarized. For these experiments it was much more difficult to increase the flow in a predictable manner, and for three dogs intermediate values were not obtained. Maximum flow was achieved by infusing acetylcholine with the contralateral carotid occluded. The effect of a stenosis in a carotid artery is more complicated than one in a femoral artery because the peripheral beds in the head are supplied by four major vessels, the two common carotid arteries and the two vertebral arteries. In addition, the predominance of muscle (quite sensitive to acetylcholine) is lacking in the beds fed by these arteries. Thus, the reduction in flow through one partially occluded carotid artery depends to some extent on the flow that can be supplied by the other major parallel vessels. In general, it was found that at elevated flow rates the data from the carotid artery experiments were less predictable, and there were greater variations in data between experiments. However, the general trends described for the femoral artery tests also apply to the results for the carotid artery experiments.

**Prediction of Pressure Drop**

The pressure drop across a stenosis depends on numerous factors. On the basis of previous model studies and similar experiments in vivo in the femoral arteries under resting conditions, the following equation was proposed:

\[ \Delta p = \frac{K_u \mu}{D} U + \frac{K_l}{2} \left( \frac{A_0}{A_1} - 1 \right) \rho U^2 + K_o \rho L \frac{dU}{dt} \]  

(3)

where \(A_0\) = area of the unobstructed tube, \(A_1\) = minimum cross-sectional area of the stenosis, \(D\) = diameter of the unobstructed tube, \(K_u\), \(K_l\), and \(K_o\) are experimentally de-
termined coefficients, $L =$ length over which the pressure drop is measured, $t =$ time, $U =$ instantaneous velocity in the unobstructed tube (average over the cross section), $\rho =$ fluid density, and $\mu =$ fluid viscosity. The first term on the right of Equation 3 represents the pressure drop due to viscous effects, the second term is the pressure drop due to nonlinear effects associated with the convergence and divergence of the flow in the stenosis and with turbulence, and the last term accounts for the pressure differential required to accelerate the fluid. $K_v$ and $K_r$ are dependent on stenosis geometry ($K_v$ is strongly dependent on geometry) but can be approximated from steady flow tests.

To determine the validity of this equation for predicting the pressure drop at elevated flow rates, the predicted value of the pressure drop corresponding to the measured velocity, $U$, was calculated from Equation 3 for each experiment. Values of $K_v$ and $K_r$ were obtained from steady flow experiments in vitro in a rigid-walled system containing stenoses that were geometrically similar to those used for the experiments in vivo. Both mean and peak pressure drops were predicted and compared with the measured values. For each set of data two regression lines were obtained: one of the form

$$\Delta p_t = C_1 \Delta p_t + C_2$$

and one which is forced through the origin having the form

$$\Delta p_t = C_0 \Delta p_t.$$

Specific values for the coefficients for the regression equations for different groupings of the data are given in Table 4 along with the corresponding correlation coefficients, $r$.

For the combined data, including both mean and peak pressure drops, the regression lines are

$$\Delta p_t = 1.00 \Delta p_t - 1.1 \quad (r = 0.95)$$

and

$$\Delta p_t = 0.97 \Delta p_t \quad (r = 0.94).$$

A graphical presentation of these results is given in Figure 4, in which predicted values of pressure drop, $\Delta p_t$ (from Equation 3), are compared with the measured values, $\Delta p_t$. In this figure all data are plotted (both mean and peak pressure drops) including the previous results of Young et al. There is good correlation between the predicted and measured values, indicating that Equation 3 satisfactorily includes the major factors that influence the pressure drop.

**Discussion**

The results of the experiments show that blood flow through a particular artery can increase by a large factor, in the range of 4–5 for the canine carotid and femoral arteries, and Gould et al. have indicated this same order of magnitude for increases in flow in the canine left circumflex coronary artery. Since the pressure drop across a stenosis is strongly dependent on the flow rate through the vessel, it is clear that as the peripheral resistance is reduced to increase the blood supply to a particular vascular bed the pressure drop increases, thereby eventually limiting the blood flow to the peripheral bed. From Equation 2 it can be seen that when $R_p$ is at its minimum value, the flow through the artery is at its maximum and the effect of

<table>
<thead>
<tr>
<th>Table 4</th>
<th>Coefficients for Regression Equations 4 and 5</th>
</tr>
</thead>
<tbody>
<tr>
<td>Artery</td>
<td>Type of data*</td>
</tr>
<tr>
<td>Femoral</td>
<td>Mean</td>
</tr>
<tr>
<td></td>
<td>Peak</td>
</tr>
<tr>
<td>Femoral†</td>
<td>Mean</td>
</tr>
<tr>
<td></td>
<td>Peak</td>
</tr>
<tr>
<td>Carotid</td>
<td>Mean</td>
</tr>
<tr>
<td></td>
<td>Peak</td>
</tr>
<tr>
<td>Carotid and femoral‡</td>
<td>Mean</td>
</tr>
<tr>
<td></td>
<td>Peak</td>
</tr>
<tr>
<td></td>
<td>Combined</td>
</tr>
</tbody>
</table>

* Mean = calculation for mean pressure drop $\Delta p_t$; Peak = calculation for peak pressure drop $\Delta p_t$.
† Data from Young et al.
‡ Includes data from Young et al.
the stenosis (greatest $\Delta p$) is most significant under these conditions.

A useful index for characterizing the effect of a stenosis is the ratio of the maximum flow rate in the presence of the stenosis ($Q_{ms}$) to the maximum flow rate without the stenosis ($Q_{mo}$). This ratio represents the reduction in the vascular bed reserve due to a stenotic obstruction. For the simple model described by Equation 2 the maximum flow rate is given by the equation

$$Q_{ms} = \frac{\bar{p}_a}{(R_p)_{min}} \left(1 - \frac{\Delta p}{\bar{p}_a}\right). \quad (8)$$

If the stenosis were not present, the maximum flow rate would be

$$Q_{mo} = \frac{\bar{p}_a}{(R_p)_{min}} \quad (9)$$

so that

$$\frac{Q_{ms}}{Q_{mo}} = 1 - \frac{\Delta p}{\bar{p}_a}. \quad (10)$$

Since $Q = A_0 U$, Equation 10 can also be expressed in terms of the corresponding velocities, i.e.,

$$\frac{U_{ms}}{U_{mo}} = 1 - \frac{\Delta p}{\bar{p}_a}. \quad (11)$$

The results of the pressure drop measurements show that the pressure drop across the stenosis can be predicted satisfactorily from Equation 3. As discussed in the Appendix, the mean pressure drop can be written in the form

$$\Delta p = A \bar{U} + B \bar{U}^2, \quad (12)$$

where the coefficients $A$ and $B$ depend on the stenosis geometry, blood viscosity and density, vessel diameter, and the shape of the pulsatile wave form, and $\bar{U}$ is the mean velocity of flow through the stenosis. For the simplest case, in which collateral flow is negligible, the flow through the stenosis is equal to flow to the peripheral beds and Equation 12 can be substituted into Equation 11 to obtain an expression for the vascular bed reserve ratio ($U_{ms}/U_{mo}$). For a given vessel diameter, blood viscosity and density, mean arterial pressure, and stenosis shape, this ratio is a function of percent stenosis and $U_{mo}$, and its calculated variation with these two parameters is given in

![Figure 5](image)

**Figure 5** Variation of the vascular bed reserve ratio with percent stenosis and maximal normal velocity. For these calculations $\mu = 0.04$ dynsec per cm$^2$, $\rho = 1.05 g/cm^3$, $B = 1.0$, $D = 4 mm$, $p_a = 100 mm Hg$, $L/D = 2$, and $K_u$ and $K_i$ were determined from equations given in Appendix.

![Figure 6](image)

**Figure 6** Effect of maximal normal velocity on the value for a critical stenosis as predicted from results given in Figure 5.

Figure 5. These results show that the value of the maximal normal flow $U_{mo}$ plays an important role in determining the limiting size of a stenosis before it causes a serious reduction in the vascular bed reserve. If a critical stenosis is defined as one which reduces the normal vascular bed reserve ratio by some specified percentage (10% arbitrarily used for calculations), then the variation in the size of a critical stenosis with maximal normal flow can be estimated; this variation is shown in Figure 6. Thus, the effect of a stenosis depends on the normal, maximal blood flow to the particular organ system or tissue bed supplied by the stenosed artery, and, as noted by May et al., the critical stenosis for the various organ system or tissue beds will be different if their normal resting velocities and vascular bed reserves are significantly different. The percent stenosis corresponding to a critical stenosis, based on a criterion related to the reduction in vascular bed reserve, will al-
ways be less than the critical stenosis for resting flow. This highly idealized model is not applicable if there is significant collateral flow. However, as shown by Roth et al., collateral flow under conditions of vasodilation will not significantly alter the effect of the stenosis on the vascular bed reserve unless there are major collateral vessels. Thus, for example, it is expected that this model adequately describes the effect of a stenosis on the vascular bed reserve for the femoral artery, but not a stenosis in a common carotid artery, which has major collateral vessels (the contralateral common carotid and the two vertebrals). For this latter case a considerably more complex model is required in order to provide a detailed analytical prediction of flow characteristics.

Values for the vascular bed reserve ratios as obtained from the femoral and carotid artery experiments are tabulated in Tables 2 and 3, respectively. In Figure 7 the experimentally determined values for $U_{mo}/U_{ms}$ are plotted. For the purpose of this graph, the data were grouped into three groups corresponding to small, intermediate, and large values of the maximal normal velocity $U_{mo}$. The general trends shown in this figure compare favorably with the predicted behavior; i.e., the vascular bed reserve ratio decreases with increasing percent stenosis for a given $U_{mo}$ and decreases with maximal velocity for a given percent stenosis. For three experiments with relatively mild stenoses at moderate velocities, the vascular bed reserve ratio exceeded unity. It is believed that this behavior is simply due to an inability to obtain precisely maximal flow through the use of vasodilator drugs. Thus, in those cases in which the stenosis is mild and the maximal velocity small, there will be only a slight decrease in the vascular bed reserve, and small experimental errors and variability in response to the vasodilator drugs can cause the experimentally measured ratio to exceed unity.

The value of the maximum normal flow, $U_{mo}$, varied over a much wider range for the carotid artery experiments than for the femoral artery experiments. This is to be expected because, as noted previously, there are major collateral vessels which supply the same vascular beds as the stenosed common carotid artery. The potential increase in blood flow through one common carotid artery when the distal beds in the head are dilated is a function of the resistances of the collateral vessels relative to the stenosed artery and the degree of communication between the four major vessels. Although there is a highly developed communication network between the carotid-vertebral system, it is expected that the degree of development is variable between different animals or individuals, thereby leading to greater variability in the measured values of $U_{mo}$.

In summary, the present investigation has confirmed through an extensive series of experiments on flow through canine femoral and carotid arteries, that the pressure drop across a stenotic obstruction can be predicted from Equation 3. This equation indicates that the pressure drop depends on the blood viscosity and density, velocity (including waveform), and stenosis geometry (including shape, length, and percent stenosis). The experimental results show that large increases in velocity are experienced under conditions of vasodilation with a corresponding large decrease in distal pressure. The pressure drop increases in a nonlinear manner with velocity, thereby accentuating the importance of the stenosis at elevated flow rates. It is suggested that a critical stenosis be defined in terms of its effect on maximal flow rather than resting flow.

Appendix

An expression for the mean pressure drop across a stenosis can be found by integrating Equation 3 over a flow cycle, i.e.,

$$\Delta p = \frac{K_t \mu}{D} U^2 + \frac{K_t}{2} \frac{A_2}{A_1} \beta |U| U,$$  \hspace{1cm} (13)$$

where the bar over the symbol indicates the time-averaged value over a cycle. The acceleration term in Equation 3 drops out of the equation when integrated over a cycle if the flow pulse is periodic. The time-averaged value of the velocity squared $|U| U$ can be written as $\beta (U)^2$, where the factor $\beta$ depends on the specific flow waveform. For example, if the velocity waveform is approximated as a half-sine wave over part of the cycle with a constant velocity over the remainder (Fig. 8a), values for $\beta$ can be calculated by a simple integration. A typical curve calculated in this manner is shown in Figure 8. Values of $\beta$ as determined from the experiments (including data from Young et al.) are plotted on the same figure and indicate that values of $\beta$ estimated from the simple waveform of Figure 8a are reasonable, even though the oscillatory waveforms in the femoral and carotid arteries are only roughly approximated with the simplified waveform. For elevated flows for which the ratio $U_p/U$ is relatively small (about 1.5), $\beta = 1$.

Sceley and Young have shown from an extensive series of steady flow tests in vitro, using plugs that were geomet-
rically similar to those used in the present study, that (1)
the factor $K_v$ is essentially constant and equal to 1.52, and
(2) as a first approximation,

$$K_v = \frac{32}{D} \left( \frac{A_0}{A_1} \right)^3,$$

(14)
corresponding to Poiseuille flow through the lumen of the
stenosis. With these approximations for $\beta$, $K_v$, and $K_v$, it is
possible to write Equation 13 in the form

$$\Delta p = \Delta U + B U \beta,$$

(12)
where $A$ and $B$ can be calculated for a given fluid and
stenosis geometry. The form of Equation 12 is the same as
that previously suggested by May et al.\cite{2} Equation 14 is a
reasonable approximation for hollowed cylindrical plugs
but would have to be modified for other stenosis geometries.\cite{26}

Acknowledgments

We are grateful to Joyce Feavel for her technical assistance throughout
the experimental studies.

References

1. Mann FC, Herrick JF, Essex HE, Baldes EJ: Effect on blood flow of
decreasing the lumen of a blood vessel. Surgery 4: 249-252, 1938

2. May AG, DeWeese JA, Rob CG: Hemodynamic effects of arterial


4. Kindt GW, Youmans JR: The effect of structure length on critical

5. Shipley RE, Gregg DE: Effect of external constriction of a blood


7. Keitzer WF, Fry WJ, Kraft RO, DeWeese MS: Hemodynamic mecha-
nism for pulse changes seen in occlusive vascular disease. Surgery 57:
163-174, 1965

8. Kreuzer W, Schenk WG Jr: Effects of local vasodilatation on blood

9. Youmans JR, Kindt GW: Influence of multiple vessel impairment on


11. Roth AC, Young DF, Cholvin NR: Effect of collateral and peripheral
resistance on blood flow through arterial stenoses. J Biomech 9: 367-
375, 1976

12. Young DF, Tsai FY: Flow characteristics in models of arterial steno-

13. Young DF, Cholvin NR, Roth AC: Pressure drop across artificially
induced stenoses in the femoral arteries of dogs. Circ Res 36: 735-
743, 1975


reserve and resistance. Am J Cardiol 34: 48-55, 1974

16. Seeley BD, Young DF: Effect of geometry on pressure losses across
Hemodynamics of arterial stenoses at elevated flow rates.
D F Young, N R Cholvin, R L Kirkeeide and A C Roth

Circ Res. 1977;41:99-107
doi: 10.1161/01.RES.41.1.99

Circulation Research is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1977 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7330. Online ISSN: 1524-4571

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circres.ahajournals.org/content/41/1/99