Fluid Dynamics of Coronary Artery Stenosis

ROBERT E. MATES, RAMJI L. GUPTA, ADAM C. BELL, AND FRANCIS J. KLOCKE

SUMMARY A large-scale model of the coronary circulation, instrumented to permit detailed pressure and velocity measurements, has been used to study flow through isolated stenotic elements in large coronary arteries. Pulsatile aortic pressure and instantaneous peripheral resistance were simulated with servovalves. A variety of axisymmetric and asymmetric stenoses were studied and flow separation was found to occur for all but very mild stenoses. Pressure recovery downstream of the stenosis throat was limited and, in some cases, no recovery was observed. Pressure drop was primarily dependent upon the minimum area of the stenosis and relatively independent of stenosis geometry. Flow was quasi-steady at normal heart rates, and simple steady flow theory proved adequate to describe the pressure drop through the stenosis. The theory yielded results that agreed well with published data for dogs and appears promising for predicting effects of hemodynamic variables on a given stenotic lesion. Thus, principal findings of the study are that a relatively severe stenosis behaves essentially like an orifice and that a simple quasi-steady theory appears adequate to predict effects of a stenosis on coronary flow.

IT HAS BEEN known for some time that a relatively severe constriction of a coronary artery is required to alter mean resting coronary flow significantly. Studies in open-chested dogs have delineated the following characteristics. An area occlusion of approximately 80-85% (corresponding to a diameter reduction of 60-80%) is required to reduce resting coronary flow. This value has been referred to as a “critical” occlusion. As this critical occlusion is approached, reactive hyperemia is almost entirely abolished indicating that full peripheral vasoconstriction has already occurred. For smaller degrees of occlusion, reactive hyperemia is reduced in comparison to the control state indicating partial vasoconstriction. Similar results were obtained in steady flow in vitro experiments conducted using postmortem coronary arteries with partial occlusion. In this study, in which perfusion pressure and downstream resistance could be independently controlled, it was found that at low flow rates stenotic resistance (ratio of pressure drop to flow) was essentially constant, suggesting fully developed laminar flow. At high flows resistance increased with flow, indicating the importance of turbulence, flow separation, or entrance effects. It was further observed that the resistance of the stenosis was primarily dependent on its minimum cross-sectional area rather than its length.

The foregoing studies have been confined to a description of the overall behavior of coronary flow in the presence of a stenosis. Because of the small size of the arteries, it is very difficult to make any detailed pressure or flow measurements to describe in detail the flow mechanism responsible for stenotic resistance. In order to study the flow in detail, it is necessary to develop large scale laboratory models. Most studies of flow through an isolated area of stenosis reported to date have been confined to steady flow studies in rigid tubes. Forrester and Young studied the flow in a converging-diverging tube with a geometry resembling observed arterial stenoses. Their experiments indicated that, for any given geometry, there was a critical value of Reynolds number above which flow separation occurred downstream to the stenosis. The critical Reynolds number at which separation occurred was a function of the area ratio and shape of the stenosis. At large flow rates, the observed pressure drop was found proportional to the square of the flow rate (resistance increasing linearly with flow) in agreement with the results of Logan for human coronary arteries. In subsequent studies, Young and Tsai found that resistance is larger for asymmetric than for axisymmetric stenoses. They also performed some experiments on unsteady flow with similar results. Golia and Evans also studied steady flow through a stenosis and developed an empirical expression for the separation Reynolds number and the length of the separated region. Analytical models also have been developed in an effort to predict the pressure drop caused by a given stenotic lesion. These have ranged from relatively simple one-dimensional models to sophisticated analyses based on the governing partial differential equations for a viscous fluid. The latter generally have been restricted to relatively mild stenoses where flow separation does not occur.

The present study was undertaken in an attempt to provide a more detailed understanding of the fluid mechanical behavior of an isolated stenotic lesion in the coronary circulation. A large scale model was chosen to allow the recording of detailed pressure measurements as well as velocity profiles. A dynamic model of the coronary circulation was constructed in which both time-varying supply pressure and peripheral resistance could be simulated. This permits simulation of the influence of systolic extravascular compression on the coronary flow. The results were used to develop a simple mathematical simu-
lution to assess the effect of a stenosis under varying hemodynamic conditions.

Methods

Since total coronary flow is less than 10% of systemic flow, one can assume that the aorta acts as a pressure source, i.e., variations in coronary flow do not affect the aortic pressure directly. Variations in right atrial pressure are small compared to aortic pressure changes and hence, for the purposes of modeling, one can assume a constant outflow pressure.

The detailed fluid mechanical behavior of the coronary circulation is complex owing to the effects of vessel elasticity and taper and wave reflections from branch sites. Investigations of the systemic circulation have demonstrated that simplified linear lumped parameter models are adequate to describe the main features of the flow. Such a model has been employed in this investigation. The impedance of the circulation includes capacitive (C), inductive (L), and resistive (R) elements. In the normal coronary circulation, viscous resistance is concentrated almost entirely in the small vessels while capacitive and inductive effects are predominant in the larger arteries.

Inertial and capacitive effects may be estimated by considering flow through the large coronary arteries. The values of inertance and capacitance were estimated on the basis of data published for the canine coronary artery and summarized in Table 1. With these values, a simple lumped parameter RLC analogue was used to compute the time varying resistance of the coronary circulation. Experimental values for aortic pressure and coronary flow were obtained by hand fitting the data of Menno et al. as shown in the upper panel of Figure 1. The driving force for coronary flow is the difference between aortic and right atrial pressure. Reported aortic pressure measurements such as those used here are normally referenced to atmospheric rather than atrial pressure. Unless atrial pressure is abnormally elevated, this difference should have little effect on the simulated resistances. The computed values of resistance are shown as the solid circles in the bottom panel of Figure 1.

For comparison, the resistance was also computed neglecting inductive and capacitive effects by taking the instantaneous ratio of aortic pressure to coronary flow, that is, the equivalent resistance for a purely resistive system. Solid circles represent the resistance computed from an RLC analogue circuit of the coronary circulation.

In the modeling, blood was assumed to behave as a Newtonian fluid. Blood exhibits a number of non-Newtonian characteristics. At shear rates less than about 50 sec⁻¹, the apparent viscosity increased markedly. Whitmore has shown that the mean shear rates in the systemic circulation are above this value except perhaps in the large veins. Additionally, in small vessels, the Fahraeus-Lindqvist phenomenon results in a reduction in apparent viscosity. This effect becomes appreciable for tube diameters less than approximately 1 mm. Thus it is not expected that this effect would be significant in the unobstructed artery, although it might begin to play a role in stenosed sections with an effective diameter less than 1 mm, which would correspond to an area obstruction of greater than 90%. It was therefore felt that the use of a Newtonian model should provide results quite comparable to those existing in the actual coronary artery. Since the non-Newtonian properties of blood are complex, it would be extremely difficult to model these effects in a large model. Use of the Newtonian model enables one to employ standard stimulation methods.

Although stenoses are most likely to occur near bends or branches in the artery, this investigation was concerned with the simple case of an isolated stenosis in a straight, rigid tube. In order to ensure dynamic similarity between the model and the coronary artery, both the mean Reynolds number, Re = 4pQrd, and the pulsatile flow parameter, α = (d/2) (ωap/μ), were simulated. Here p is the fluid density, Q the mean coronary flow, μ the fluid viscosity, d the vessel diameter, and ω the basic frequency of the system or the reciprocal of the heart

<table>
<thead>
<tr>
<th>Variable</th>
<th>Numerical value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Length (L)</td>
<td>100 mm</td>
</tr>
<tr>
<td>Wall thickness (t)</td>
<td>0.32 mm</td>
</tr>
<tr>
<td>Diameter (d)</td>
<td>3.2 mm</td>
</tr>
<tr>
<td>Young's modulus (E)</td>
<td>7.7 x 10⁸ dyn/cm²</td>
</tr>
<tr>
<td>Blood density (ρ)</td>
<td>1.0 g/ml</td>
</tr>
</tbody>
</table>
rate. The model was made sufficiently large (19 mm inside diameter) to allow detailed velocity measurements in the vicinity of the stenosis. Automotive ethylene glycol antifreeze, which has a viscosity appropriate for the model, was chosen as the working fluid. The ratios of model to actual parameters are summarized in Table 2.

The experimental model, shown schematically in Figure 2, consisted of a pressure source simulating aortic pressure, a test section in which an isolated stenosis could be placed, and a variable hydraulic resistance. The pressure source was a single stage three-way servovalve supplied with constant pressure by a positive displacement gear pump. The servovalve was designed for a maximum flow rate of 9.6 liters/min at a pressure of 800 mm Hg. The sine wave frequency response of the pressure servovalve was flat to 2 Hz, well above the nominal operating frequency of 0.083 Hz. A flow straightener downstream of the valve was employed to relaminarize the flow leaving the servovalve. The pressure output from the servovalve was measured downstream of the flow straightener and compared with a desired reference signal. Proportional-integral control was used to provide pressure regulation.

The coronary resistance simulator consisted of a servovalve identical to that used for the pressure source. The instantaneous resistance was obtained by simultaneously measuring the pressure drop across one of the spool orifices (the other was plugged) and the flow through the orifice. The ratio of these two signals was compared with a reference value and the amplified error used to drive the torque motor of the servovalve. The system was linear over the range of resistances used in the experiments.

The test section consisted of 1-foot-long segments of transparent polycarbonate, each equipped with a static pressure tap. The stenosis could be inserted between any two segments thus allowing an investigation of entrance and exit length effects on the flow through the stenosis. A variety of axisymmetric and asymmetric stenoses was fabricated. Their geometries are summarized in Figure 3. The stenoses M-1 through M-5 were constructed by casting a liquid plastic monomer in an aluminum mold with a removable aluminum core of the desired geometry using the technique of Young and Tsai.* Asymmetric models M-6 through M-11 were made by casting paraffin wax in one of the straight sections of tubing and then machining the core to the required geometry. The exit from the test section consisted of multiple radial outlets to minimize exit effects. A traverse mounted on the downstream end of the test section permitted axial and radial traverses using a Disa hot film anemometer probe for local velocity measurements.

Preliminary experiments were carried out for both steady and pulsatile flow through the test section without a stenosis to ensure the existence of a fully developed laminar flow upstream of the stenosis. Entrance effects were confined to the first 25 cm of the test section and exit effects were negligible.† In Figure 4, the nondimensional peak pressure drop for an oscillating flow with a peak Reynolds number of 340 is plotted as a function of $\alpha$. The solid line was calculated from Womersley's theory for fully developed unsteady laminar pipe flow. Good agreement was obtained with the data. At low values of the pulsatile flow parameter $\alpha$ the pressure drop asymptotically approaches the steady state value $(64/\text{Re})(f/d)$ where Re is the Reynolds number, $f$ is the test section length, and $d$ the test section diameter. At the nominal operating frequency of 0.085 Hz, $\alpha = 1.7$. The

![Figure 2](image_url)

**Figure 2.** Schematic diagram of apparatus. Simulated aortic pressure is supplied to the test section through a spool-type servovalve and a transition section. Coronary resistance is simulated by an identical downstream valve. The test section containing the stenotic element is instrumented for pressure and velocity measurements.
solid curve indicates that dynamic effects should be quite small at this frequency.

The desired aortic pressure and coronary resistance wave forms were produced by digital-to-analogue conversion of the desired signal on a PDP-8/E digital computer and recording on a 4-channel Ampex FM tape recorder. In this way, the system could be run at steady state for long periods of time. The pressure and resistance signals from Menno and Schenk38 shown in Figure 1 were used in the tests. Pressure and flow data were recorded on an 8-channel Honeywell FM tape recorder for A/D conversion and analysis on the PDP-8/E.

Results

Steady Flow through a Mild Stenosis

Several investigators have studied steady flow through mild stenoses. The study most closely related to our problem is the recent work of Young and Tsai.8 We obtained complete pressure-flow characteristics of a mild
The pressure drop across stenosis (M-1) is plotted nondimensionally in Figure 6 as a function of Reynolds number. The solid circles represent the experimental data and the dotted line the experimental results of Young and Tsai.6 Excellent agreement was attained. Young and Tsai found a critical Reynolds number of 190, in close agreement with the value of 200 which we observed. For comparison, the solid line represents the nondimensional viscous pressure drop for fully developed flow through an unobstructed tube of the same length and nominal test section diameter.

Sinusoidal Flow through a Mild Stenosis

Pulsatile flow through the mild stenosis was investigated by superimposing a sinusoidal flow with a maximum oscillatory Reynolds number of 63 on a steady flow with a Reynolds number of 207. The peak dimensionless pressure drop is plotted for various frequencies of oscillation in Figure 7. Also shown by the solid line is the predicted pressure drop for fully developed flow through an unobstructed tube of the same length based on Womersley's theory.21 At low frequencies, the flow is quasi-steady and the dimensionless pressure drop becomes independent of frequency. At higher frequencies, the pressure drop increases with frequency. The experimental results show a higher pressure drop than predicted by the Womersley theory, the additional drop being contributed by the constriction. It is interesting to note that, although the curve has been shifted vertically, the break frequency has been maintained. This probably indicates proportional viscous and inertial increments. Young and Tsai's6 results are represented by the dotted line. Their results show a

(55%) stenosis (M-1), geometrically identical to one of theirs, to validate our measurement techniques.

The steady flow nondimensional pressure distributions along the stenosis for various Reynolds numbers are shown in Figure 5. Nondimensional variables have been chosen since they are equal for the model and prototype. Axial distance along the stenosis is nondimensionalized by the tube diameter d. Pressure drop $\Delta p$ is normalized by half the product of density $\rho$ and the square of the mean flow velocity $V^2$. The pressure drop was measured from a reference upstream pressure tap to various taps along the stenosis and downstream from it. The pressure falls rapidly in the converging section of the stenosis, primarily due to the Bernoulli effect as the flow accelerates. Downstream to the throat, there is a tendency for pressure recovery due to flow deceleration. In the absence of viscous effects, the pressure would rise to the value upstream of the stenosis. This pressure rise is counterbalanced by viscous effects that tend to reduce the pressure. For very low Reynolds numbers, viscous effects dominate and no pressure recovery is observed. As the Reynolds number increases, some pressure recovery is observed in the diverging section. However, this adverse pressure gradient causes flow separation, producing a recirculating vortex that dissipates the kinetic energy of the fluid and limits pressure recovery. For this stenosis, separation occurs at a Reynolds number of approximately 200. This was verified using the hot film anemometer velocity profiles.17
somewhat lower pressure drop. The value of Reynolds number at which they conducted their experiments is not given in the paper and this perhaps explains the discrepancy. The subsequent experiments using actual aortic pressure and resistance signals were carried out at a fundamental frequency of 0.083 Hz which corresponds to a heart rate of 60 beats/min. These results would indicate that unsteady effects should be minimal at this frequency, although the pressure and resistance signals contain higher harmonics and are not quantitatively predictable from these results.

Simulated Coronary Flow through a Stenosis

Following these preliminary experiments, the entire series of stenotic elements was inserted into the test section and studied under the time-varying pressure and resistance described above. Pressures were recorded continuously throughout the cardiac cycle from a series of pressure taps located along the stenosis and downstream.

Typical systolic and diastolic pressure recordings are shown in Figures 8 and 9 for M-3 and M-5, respectively. The characteristics are similar to those presented earlier for steady flow. The 90% stenosis exhibits some pressure recovery downstream of the throat. The 95% stenosis causes a much larger pressure drop than the 90% stenosis.

Velocity profiles were obtained using the hot film anemometer probe for models M-1 through M-5. Typical results are shown in Figures 10 and 11. Figure 10 presents velocity profiles, normalized with respect to maximum velocity, during systole and diastole for the 90% axisymmetric stenosis (M-3). The velocity profile at the throat is very blunt due to flow acceleration in the converging section. Separation occurs downstream of the throat. Since the hot film probe is not sensitive to reverse flow, we were not able to measure the velocity in the separation region. The areas of zero velocity shown on the figures.
include recirculating flow regions. The dotted lines represent an estimate of the region of flow separation. Separation was observed both during systole and diastole for all but the mildest stenosis (M-1). For the asymmetric stenosis (Fig. 11), normalized velocity profiles were obtained across two perpendicular diameters at various stations downstream of the throat. Results are presented only for diastole. The upper panel, set A, shows a large separation region and a jet of fluid issuing from the throat of the stenosis. This separation region is larger than that observed for the axisymmetric stenoses and probably accounts for the total absence of pressure recovery shown earlier in Figure 9. The velocity traverses in set B show an initially blunt profile which develops a central peak that later disappears. Flow separation is not observed in this plane. Approximately five diameters downstream of the throat, the jet has largely disappeared and the flow has reattached to the wall.

**Quasi-Steady Model**

While the detailed experimental results are helpful in understanding the fluid mechanical behavior of a given stenosis, it would be desirable to develop a mathematical model characterizing these results in order to simulate the
effect of a given stenosis on the coronary circulation under varying hemodynamic conditions. The results presented above were obtained for an equivalent heart rate of 60 beats/min. The preliminary sinusoidal flow experiments indicated that frequency had very little influence on measured velocities and pressures, indicating that the system is quasi-steady. We therefore attempted to develop a simple quasi-steady model to explain the observed pressure drops.

At each instant of time during the cardiac cycle, the pressure drop through the stenosis was assumed equal to that which would occur through the same geometry under steady flow conditions. First, the pressure drop caused by viscous wall shear was calculated. For the asymmetric stenoses, the cross-section of the stenosis is noncircular. For noncircular ducts, the viscous pressure drop can be approximated by that through a circular duct with the same equivalent diameter. The equivalent diameter is defined as four times the cross-sectional area divided by the perimeter of the area. The flow is assumed to be laminar and fully developed so that the local pressure gradient dp/dx is given by the Poisson equation, 

\[ dp/dx = 128\mu Q/\pi d^4 \]

where \( \mu \) is the fluid viscosity, \( Q \) the mean flow, and \( d \) the tube diameter. The total viscous pressure drop through the stenosis up to the separation point was obtained by integrating this expression along the length of the stenosis. Since equivalent diameter varies in a very complex fashion with distance \( x \), the integration was performed numerically. The blunt velocity profiles observed near the throat of the stenosis indicated that flow was not fully developed. For such a blunt profile, the wall shear is larger than that in a fully developed flow. A correction was applied to account for this increase by pressure drop based on the results given by Goldstein.

In the separated flow region, energy is dissipated by the recirculating vortex. Since the experiments indicated that in most cases the pressure recovery observed downstream of the throat was very small, the recovery was assumed to be zero. This is equivalent to treating the stenosis as an orifice of the same area as the stenosis throat in series with a viscous resistance. Results for stenosis M-6 through M-11 are shown in Figures 12 and 13. In Figure 12, the pressure drop during diastole is plotted as a function of stenosis length for a series of 95% stenoses with sharp throats. The solid line indicates the prediction of the simple theory while the circles represent experimental data. The circle corresponding to a span of 0 represents flow through a sharp orifice. The pressure drop rises nearly linearly with the total stenosis span. In Figure 13, the pressure drop is plotted as a function of the throat length for a series of long 95% stenoses. Again the solid line represents the prediction and the solid circles the experimental results for diastolic pressure drop. Here the predicted drop increases less steeply with length as the throat length increases. In both cases, good agreement was achieved between the predicted value and the measured value.

The preceding results give the pressure drop at a particular time during the cardiac cycle. Of greater interest is the mean flow produced in the coronary circulation for a given stenosis geometry. In our model, the coronary circulation is represented by two series resistances, the stenosis and the peripheral bed. The pressure-flow relationship for the stenosis is nonlinear because the stenosis resistance varies with the flow rate. Since initially the flow is unknown, it is not possible to specify the resistance in advance. The mean flow \( Q \) was calculated in the following manner. The driving pressure and peripheral resistance were taken as known functions of time as described earlier. The pressure-flow relation for the system is given by

\[ \Delta p = R_1 Q + K_1 Q^2 + K_2 Q^2 + R_2 Q \]

where \( R_1 \) represents the viscous resistance of the stenosis, \( K_1 \) is the entrance loss coefficient, \( K_2 \) is the loss coefficient for the separated flow, and \( R_2 \) is the peripheral bed resistance. The coefficients are given by

\[
R_1 = 128 \mu L/\pi d_0^4 \int \frac{dx}{(d/d_0)^4}
\]

\[ K_1 = (1.14p)/d_0^4 \]

\[ K_2 = (2C_0\mu^2/\pi d_0^4)(d_0/d_1)^4 - 1 \]

where \( L = \) stenosis length, \( d_0 = \) tube diameter, \( d_1 = \) minimum equivalent diameter of stenosis, and \( C_0 = \) orifice discharge coefficient (0.63). The quadratic equation may be solved for \( Q \)

\[
Q = \frac{(R_1 + R_2)}{2(K_1 + K_2)} \left( -1 + \sqrt{1 + 4\Delta p(K_1 + K_2)/(R_1 + R_2)^2} \right).
\]

This gives the instantaneous flow at each point during the cardiac cycle. Finally, mean flow was determined by averaging over the cardiac cycle. Typical results are shown in Figure 14 for the limiting cases of a sharp orifice and a long asymmetric stenosis (M-11). The upper two curves in the figure were calculated for a peripheral bed resistance equivalent to that achieved under full vasodilation. Vasodilation was simulated by assuming that the compressive portion of resistance remained unchanged while the noncompressive resistance decreased; the noncompressive resistance, which includes viscous and autoregulatory
components, was calculated from the minimum resistance during the diastolic portion of the nominal cycle. This value was decreased to simulate vasodilation.

In the actual circulation, autoregulation acts to reduce peripheral resistance as the stenotic resistance increases in order to maintain required coronary flow. Thus, ideally the flow could remain constant until the peripheral bed was fully dilated. This is shown schematically by the lower curve in Figure 14. This curve was chosen to represent resting conditions, with the mean flow approximately one-fourth of that which would be produced with no stenosis in the fully dilated condition.

This theory indicates that, whereas the long asymmetric stenosis produces a larger pressure drop and consequently a lower flow through the system, this difference is most pronounced for moderate stenoses and disappears as the stenosis becomes more severe. Under normal conditions, when an approximately 90% stenosis is required to produce any reduction in coronary flow, the orifice and the long stenosis behave essentially identically. Once the stenosis is increased even a small amount beyond this critical value, the flow in the coronary circulation will drop precipitously. Under stress conditions, when the demand is increased, the lower curve in this figure will shift upward and a less severe stenosis will then become flow-limiting. Thus, the coronary reserve available to meet increased demand is reduced even by moderate stenoses.

Discussion

A number of recent in vivo studies have demonstrated that relatively severe constrictions of a coronary artery are required to cause a reduction in coronary flow. These studies have been confirmed by in vitro studies using postmortem human coronary arteries. The present study was intended to investigate in more detail the fluid mechanics of the isolated stenosis and to develop a simple mathematical model for predicting mean coronary flow in the presence of a given lesion under varying hemodynamic conditions.

Although the actual coronary circulation is extremely complex, a very simple model was chosen for initial studies using a straight rigid tube with stenotic elements inserted in series with the peripheral bed resistance.

While the use of scale models is routine in studying the behavior of physical systems, modeling biological systems must be approached with more care because of their...
inherent complexities. It is clearly impossible to develop a large scale laboratory model which is an exact replica of the coronary circulation. In addition to simplifying the geometry, we made two significant assumptions in developing the laboratory model. First, it was assumed that the superficial coronary artery is a rigid tube. Second, we assumed that blood flowing in the coronary artery behaves as a Newtonian fluid. Both of these assumptions have been discussed previously and are believed reasonable for the purpose of the model. The validity of the model results depends upon similitude between the model and the coronary artery. This requires that the appropriate nondimensional parameters, in this case the Reynolds number and the α parameter, be the same in the model and prototype. If these conditions are met, then the nondimensional pressure distributions, velocity profiles, and streamline patterns must be the same in the two situations. The measurements represent the detailed flow of a Newtonian fluid through an isolated stenosis in a rigid coronary artery. The extent to which these results duplicate the flow in the actual artery can be assessed only by comparison with results of in vivo measurements. Since detailed measurements are not available, the model can be assessed only by comparing the predicted overall behavior of the stenosis with that observed in animal preparations. Some comparisons are discussed below.

The detailed pressure and velocity measurements indicated that flow separation occurs even for relatively mild constriction of the tube, well below that required to limit coronary flow under resting conditions. Thus, an appreciable portion of the pressure drop is a result of energy dissipation in the recirculating flow region downstream of the stenosis throat. For relatively severe stenoses, essentially no pressure recovery was obtained in the region distal to the location of minimum area. In such cases, the total pressure stop becomes primarily a function of minimum stenosis area and relatively independent of the detailed geometry. This is in agreement with the observations of Logan4 based on the steady flow through postmortem human coronary arteries.

Despite the fact that both the driving pressure and peripheral bed resistance are time-varying, dynamic effects appear to be minimal. The simple quasi-steady theory provides an adequate representation for the measured pressure drop. The experimental results were obtained in a laboratory model that does not include the effects of vessel elasticity. While our initial calculations indicated that this elasticity is not of major importance in defining the impedance of the coronary circulation, the inclusion of vessel elasticity would tend to increase dynamic effects. Further, the experiments were conducted at a nominal heart rate of 60 beats/min. At higher heart rates, dynamic effects may become important, although our preliminary experiments with a sinusoidal flow indicate that the flow through the system remains quasi-steady to frequencies approximately 10 times the nominal value. Since the actual pressure and resistance curves are not truly sinusoidal but contain higher harmonics, dynamic effects may become important at relatively lower frequencies.

The quasi-steady simulation indicates that, for relatively severe stenoses, it is primarily the minimum area of the stenosis rather than the detailed geometry which influences the maximum flow that can be produced in the coronary circulation. While our results predict that an approximately 80–90% area stenosis is required to reduce resting flow, coronary reserve is compromised by relatively moderate stenoses. Both Gould et al.1 and Furuse et al.5 have reported measurements of coronary flow through artificial stenoses in the dog. They found a stenosis that reduced area by approximately 80–85% was necessary to cause a reduction in resting flow, in good agreement with the prediction of our model. Using a temporary occlusion6 or a Hypaque injection,7 they measured maximum flow as a function of percent stenosis in order to obtain a measure of the available coronary reserve. Their results have been compared with the predictions of our simulation in Figure 15. The results are seen to be in relatively good agreement. These investigators also noted that diastolic flow was reduced considerably more than systolic flow. In some cases systolic flow increased slightly in the presence of the stenosis.4 These results are also consistent with our observations in that the pressure drop through the stenosis increases approximately as the square of the flow for severe stenoses. Thus, the stenosis presents a much larger effective resistance during diastole than during systole. The slight increase in systolic flow observed in the presence of an occlusion may be due to a localized reduction in myocardial contractility and consequently reduced extravascular compression of the peripheral bed. Alternatively, this increase in systolic flow may result from local peripheral vasodilation in epicardial layers of the myocardium. These effects have not been simulated in our model.

In summary, the primary findings of the study are that the hemodynamic effect of a given isolated stenotic lesion in a coronary artery can be approximated by a relatively simple quasi-steady analysis. Further, the pressure drop across a stenosis appears to be primarily a function of minimum stenosis area rather than the detailed geometry of the stenosis. Relatively severe stenoses are required to limit resting coronary flow; however, more moderate stenoses can cause a significant reduction in coronary reserve. The present study has been concerned only with total coronary flow and has not examined possible transmural flow variations. It is likely that reduced perfusion pressures caused by moderate stenoses would compromise subendocardial flow even when total flow remains adequate. While the results presented here are based on a highly simplified model, we believe that they are qualitatively correct and useful in understanding the phenomena involved in coronary artery disease.

References

Errata

In the article by C.P. Rose and C.A. Goresky, "Constraints on the Uptake of Labeled Palmitate by the Heart: The Barriers at the Capillary and Sarcolemmal Surfaces and the Control of Intracellular Sequestration," Circ. Res. 41: 534-545 (October), 1977, there should be a prime sign (') rather than three dots (\ldots) after the k's on the bottom of p. 538 (column 2).

The formulas on the top of p. 541 (column 2) should read as follows:

\[
\ln \left( \frac{C(t)_{\text{palmitate}}}{C(0)_{\text{palmitate}}} \right) = k' \gamma r_2(t) \frac{2P_c \tau_s(t)}{r(1 - Hct)}
\]

\[
\ln \left( \frac{C(t)_{\text{palmitate}}}{C(t)_{\text{sucrose}}} \right) = k' \gamma r_2(t) \frac{2P_c \tau_s(t)}{r(1 - Hct)}
\]

where \( P_s \) and \( P_p \) are the capillary permeabilities for sucrose and palmitate, respectively, \( r \) is the capillary radius, and \( Hct \) is the hematocrit. From this

\[
\ln \left( \frac{C(t)_{\text{palmitate}}}{C(t)_{\text{sucrose}}} \right) / \ln \left( \frac{C(0)_{\text{palmitate}}}{C(0)_{\text{sucrose}}} \right) = \frac{P_s}{P_p}
\]
Fluid dynamics of coronary artery stenosis.
R E Mates, R L Gupta, A C Bell and F J Klocke

Circ Res. 1978;42:152-162
doi: 10.1161/01.RES.42.1.152

Circulation Research is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1978 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/42/1/152

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation Research 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 Circulation Research is online at:
http://circres.ahajournals.org/subscriptions/