Details of coronary stenosis morphology influence its hemodynamic severity and distal flow reserve.
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Details of Coronary Stenosis Morphology
Influence Its Hemodynamic Severity and Distal Flow Reserve

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Differences in coronary flow reserve with anatometrically similar coronary artery stenoses have been attributed to 1) nonstandard physiologic conditions, 2) inadequacies of measurements of coronary artery stenosis dimension and/or coronary blood flow, and 3) inadequate hyperemic stimulus. Our study tested the hypothesis that details of coronary artery stenosis geometry, which may or may not be apparent on coronary angiograms, also may contribute importantly to such differences. A simple and complex coronary artery stenosis, each of which reduced vessel cross-sectional area by 84%, was introduced in random order into the left anterior descending coronary artery of nine closed-chest, sedated swine. The simple stenosis had a single lumen while the complex stenosis had five small lumena whose combined area equaled that of the single lumen stenosis. Measurements of hemodynamics and regional myocardial blood flow (microspheres) were made at control and after 10 minutes of adenosine infused at 400 μg/min and then at 800 μg/min distal to each stenosis. Both heart rate and aortic mean pressure were controlled and thus did not change versus initial baseline (129±4 minutes and 120±10 mm Hg, mean±SD, respectively) during the study. Baseline total flow (ml/sec) distal to the stenosis was similar at each control (1.05±0.35 vs. 0.92±0.34, simple versus complex, respectively; p=NS). At maximal adenosine, total flow with the simple stenosis was 3.44±0.92 versus 2.77±0.51 for complex (p<0.05). Distal zone maximal endocardial flow with the simple stenosis (3.83±1.16 ml/min/g) also was greater (p<0.01) than that with the complex stenosis (2.76±0.83 ml/min/g). Stenosis gradient at identical levels of flow both at baseline and at maximal hyperemia was approximately twofold greater with the complex lesion than with the simple stenosis. Thus, details of coronary artery stenosis geometry (simple versus complex) may contribute importantly to differences in coronary flow reserve (especially in the endocardium) despite comparable reduction in vessel cross-sectional area. (Circulation 1989;80:636–642)

Characterization of coronary artery stenosis severity has proven difficult from a physiologic point of view. An early effort to correlate visual estimates of anatomic stenosis severity with hyperemic flow reserve was unsuccessful.1 Subsequently, computer-based measurements of anatomic stenosis severity have been used and have been useful in improving the correlation between anatomic and physiologic stenosis severity,2,3 although problems remain.4 Recently, it also has been pointed out that a variety of baseline factors such as major determinants of myocardial oxygen demand and the presence or absence of left ventricular hypertrophy need to be considered in the interpretation of hyperemic flow reserve distal to a coronary arterial stenosis.4,5 Raising or lowering the baseline value of coronary flow obviously will influence the maximal hyperemia-to-baseline flow ratio that is commonly used to define the physiologic severity of the lesion.

Another problem that may contribute to difficulty in correlating the physiologic severity of a coronary stenosis with its apparent anatomic severity is the inability to appreciate morphologic details of the stenosis on clinical coronary arteriograms. Although several studies have called attention to irregular-appearing lesions and their frequent association with unstable angina pectoris,6–10 the focus of such studies has been on the clinical implications of presumed plaque rupture and thrombosis and not on the hemodynamic effects of irregular stenosis morphology per se. Indeed, there is no published

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information available concerning the potential effects of stenosis morphology on physiologic severity of the lesion in an intact animal model. Furthermore, data obtained from in vitro experiments have not been consistent. Results of one well-designed study indicate that stenosis morphology may not have a major impact on its hemodynamic severity. In contrast, results of other in vitro experiments indicate that stenosis morphology per se may have an important influence on its pressure-flow relations and, hence, physiologic severity. Previous authors also have stressed the difficulty in formulating mathematical models to predict the behavior of irregular-shaped stenoses and the need for additional studies. Accordingly, the present study tested the hypothesis that the stenosis model which details of stenosis anatomy may contribute importantly to its physiologic severity.

Experiments were conducted in sedated, closed-chest domestic swine under carefully controlled conditions. Each animal served as its own control and was tested with both a simple and a complex artificial coronary arterial stenosis. Each stenosis produced the same reduction in vessel cross-sectional area. The simple stenosis, however, had only a single lumen available for passage of blood. In contrast, the complex stenosis used five lumens whose combined cross-sectional area equaled the area of the single lumen device. The multilumen device was selected because it offered a quantifiable model (in terms of lumen surface area) that could be compared fairly with a simpler lesion and thus a more precise quantitative estimate of the effects of complex geometry on stenosis hemodynamics could be obtained.

Methods

Animal Preparation

Farm-bred domestic swine (n=9; mean weight, 58 kg; range, 45–74 kg), after an overnight fast, were premedicated with sodium thiamylal (total dose, 0.5–1.0 g i.v.), intubated, and anesthetized with halothane (0.5–1.0%) and 60% NO2-40% O2. The animals were ventilated with a volume-cycled respirator through which supplemental oxygen was delivered at 2–3 l/min mixed with room air and anesthetic gases. Arterial blood gases were monitored frequently and were maintained at physiologic levels (pH 7.39–7.45; PCO2, 35–45 mm Hg; PO2, 100–125 mm Hg) throughout each study. After induction of anesthesia, each animal was anticoagulated with heparin (225 IU/kg i.v.). Full anticoagulation was maintained by administration of approximately half the initial loading dose every 2–3 hours. Each animal also was given aspirin 325 mg i.v. just before instrumentation.

The animal was instrumented for the study as follows. A 7F Eppendorf catheter was advanced under fluoroscopic control from the right femoral artery to the left ventricle and then retrograde across the mitral valve to the left atrium. This catheter was used for administration of radioactive microspheres for measurements of regional myocardial blood flow. The left femoral artery was used to introduce a balloon in the descending aorta above the level of the renal arteries. This balloon was inflated, if necessary, so that mean aortic pressure remained constant throughout the protocol. An 8F double-lumen catheter was inserted into the right brachial artery and then advanced to the ascending aorta. This catheter was used to monitor arterial blood gases and for reference withdrawal for microsphere determinations for regional myocardial blood flow. The femoral veins were cannulated bilaterally with 8F catheters that were used for administration of fluids and medications during the course of the study. Next, a 7F bipolar pacing catheter was inserted in the left internal jugular vein and subsequently advanced to the coronary sinus. The pacing catheter was used to maintain the animal’s heart rate at 125–135 beats/min throughout the study.

Before placement of the coronary arterial stenosis (see below), halothane and nitrous oxide were discontinued and the animal was permitted to awaken sufficiently to breathe spontaneously and exhibit modest tremulousness. A constant intravenous infusion of sodium thiamylal was begun at 200–800 mg/hr to maintain sedation and ensure the animal was free of pain. Once the animal had stabilized for approximately 20 minutes, the experimental protocol was begun. In all instances, intravenous drugs and medications were administered in normal saline.

Experimental Protocol

Two coronary arterial stenoses were used (Figure 1). Each was mounted on a double-lumen catheter and introduced into the left anterior descending artery of the animal as previously described. The “simple” stenosis was a plastic cylinder (7-mm long) with outer diameter of 4.0 mm tapering to 3.75
mm distally. The stenosis had a single straight, 1.6-mm diameter circular lumen that reduced vessel diameter by 60% and cross-sectional area by 84%. Surface area of the stenosis lumen was 35 mm². The complex stenosis had the same external dimensions. However, five separate lumens, each 0.7 mm in diameter, were made through the device to allow for passage of blood. The multilumen device also reduced vessel cross-sectional area by 84%. Its total luminal surface area was 77 mm². Both stenoses were placed in random order in each animal's left anterior descending artery.

After stenosis placement, baseline measurements of hemodynamic parameters (i.e., heart rate and aortic, left atrial, and distal coronary pressures) and regional myocardial blood flow (microspheres) were obtained. Repeat measurements of all experimental parameters were obtained after a 10-minute intra-coronary administration of adenosine distal to the stenosis at 400 and 800 μg/min.

At the conclusion of the study, myocardium distal to the stenosis was marked by injection of radiolabeled microspheres through the coronary infusion catheter attached to the stenosis. Afterward, the animal was given a large intravenous dose of sodium thiamylal and killed 5 minutes later by an intravenous injection of potassium chloride. Next, the chest was opened after which the stenosis-catheter system was removed from the left anterior descending coronary artery and the vessel itself was inspected for evidence of gross intimal damage. After this, the heart was rinsed thoroughly in tap water, refrigerated in buffered saline, and later sectioned for determination of microsphere activity (see below).

**Hemodynamics**

Heart rate (lead II of the surface electrocardiogram) and arterial, left atrial, and distal coronary artery pressures were monitored continuously throughout the study and recorded on chart paper with a Hewlett-Packard eight-channel recorder (Model 5588A). Aortic balloon inflations were made as necessary to maintain mean aortic pressure constant throughout the protocol. Intravascular and intracardiac pressures were recorded from fluid-filled catheters connected to Hewlett-Packard force transducers (Model 1280A). In addition to analog recordings, data were also digitized, displayed, and stored on-line with an IBM-AT laboratory computer system. Software developed in our laboratory to record and analyze data has been described previously.

**Regional Myocardial Blood Flow and Stenosis Resistance**

For each experimental condition, approximately $4 \times 10^6$ radiolabeled microspheres (15 μDIA, 85–105 μCi total radioactivity) were injected via the left atrial catheter to determine regional myocardial blood flow. A different radioisotope was chosen at random for each flow determination. Details of microsphere methods used in our laboratory have been published. The technique used to accurately count as many as seven isotopes in the same tissue sample is based on the method of Baer et al and has been used in our laboratory since 1984. Briefly, an energy spectrum for each isotope is acquired by counting the isotope alone. The data are used in a computer program that uses a least-squares method to determine true counts for each isotope in the tissue sample by relating observed counts in each energy window to spectra obtained from pure isotopes.

Stenosis resistance (mean) was calculated by dividing the mean pressure gradient across the stenosis in millimeters of mercury by total transmural coronary blood flow (ml/sec) distal to the stenosis. Total flow was determined by identifying and counting all areas of myocardium (including the right ventricle and interventricular septum) that were distal to the stenosis. Appropriate regions of right ventricle and interventricular septum were marked by injecting methylene blue distal to the stenosis just before the potassium chloride injections. Stenosis pressure gradient versus total flow was plotted for group mean (±SEM) data for each stenosis and fit to a second-order polynomial by means of a commercially available computer program (Sigma-Plot, Jandel Scientific, Corte Madera, California).

**Stenosis In Vitro Pressure-Flow Relations**

The pressure-flow characteristics of each stenosis also were assessed under steady-flow conditions in vitro. Whole blood was obtained from one pig and stored in CPDA-1 solution (63 ml citrate-phosphate-dextrose-adenine 450 ml whole blood). On the day of the in vitro study, the blood was gently mixed and warmed to =37°C. Each stenosis was secured.
within a short length of firm plastic tubing (i.d., 3.2 mm), which in turn was connected to a tubing adaptor with a side port. Accordingly, the tubing containing the stenosis could be interchangeably attached to a common length of the same tubing, which in turn was connected to a blood-filled reservoir. The stenosis was held at a fixed height above the laboratory bench and the height of the reservoir above it varied to produce flow rates between 1.0 and 4.0 ml/sec. Pressure was recorded 3.5 cm upstream of the stenosis with zero reference set at the level of the stenosis itself. Blood flowed through the tubing and out of the stenosis to atmosphere where it was collected in a preweighed plastic container with acquisition time (20 seconds) carefully noted. Flow (ml/sec) was determined by reweighing the container after the timed collection, subtracting (postcollection weight minus precollection weight), dividing by time, and then dividing the result by 1.06. Each stenosis was tested at a given reservoir height after which the height of the reservoir was readjusted. The procedure was repeated twice for each stenosis until the full range of flows with corresponding pressure drop had been recorded. Duplicate values agreed closely and were averaged in the data analysis. A commercially available computer program (Sigma-Plot, Jandel Scientific) was used to fit pressure-flow data to a second-order polynomial.

Statistics
The significance of group mean changes in hemodynamics, regional myocardial blood flow, and stenosis resistance parameters were assessed by means of a blocked one-way analysis of variance and Dunnett's t test.21 Results were considered statistically significant when p<0.05. All values are expressed as mean±SD.

Results

Hemodynamics
In accordance with the study design, there were no significant changes in heart rate, mean arterial pressure, or tension time index versus initial control at any time during the study (Table 1). Mean left atrial pressure also did not change compared with initial control at any time during the study. As anticipated, transstenotic pressure gradient increased versus respective baseline in response to each dose of adenosine for both simple and complex stenoses (p<0.01). The pressure gradient across the stenosis also was significantly (p<0.01) greater for the complex versus simple stenosis at each dose of adenosine. Mean distal coronary pressure during the 800 μg/min adenosine infusion also was lower with the complex versus the simple stenosis (71±9 vs. 78±14 mm Hg, respectively; p<0.02). In addition, endocardial perfusion pressure (distal mean diastolic pressure minus mean left atrial pressure plus 7 mm Hg22) was lower (p<0.03) with the complex stenosis at maximal adenosine (46±12) in comparison with the simple stenosis (55±16). The transstenotic pressure gradient for the simple stenosis increased significantly at the 800 μg/min versus the 400 μg/min adenosine dose but did not change significantly with the complex stenosis (400–800 μg/min adenosine).

Regional Myocardial Blood Flow and Stenosis Resistance

Baseline values of regional myocardial blood flow were comparable in distal zone endocardial and epicardial layers of the heart before adenosine intervention for each stenosis (Table 2). The distal to circumflex flow ratio for each layer of the heart also was comparable before adenosine intervention with each stenosis, as was the distal zone endocardial to epicardial flow ratio.

Adenosine caused a significant (p<0.01) increase versus baseline in distal zone flow in each layer of the heart for both simple and complex stenoses. Although flow values in all myocardial layers tended to be higher for each stenosis at the 800 μg/min compared with the 400 μg/min adenosine dose, the differences were not statistically significant.

Distal zone endocardial, transmural, and total flows were significantly greater at the 800 μg/min dose (p<0.01, endocardium; p<0.05, transmural and total) with the simple stenosis in comparison with the complex lesion. In addition, the ratio of complex to simple stenosis for maximal endocardial
TABLE 2. Distal Zone Blood Flow and Stenosis Resistance

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<th>Simple stenosis</th>
<th>Complex stenosis</th>
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<tr>
<td></td>
<td>Control  400 µg adenosine 800 µg adenosine</td>
<td>Control  400 µg adenosine 800 µg adenosine</td>
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<tr>
<td>Flow (ml/min/g, mean±SD)</td>
<td>Endocardium 1.71±0.61 3.35±1.69* 3.83±1.16* 1.54±0.38 2.63±0.78* 2.76±0.83†*</td>
<td>Epicardium 1.58±0.67 5.12±2.46* 6.34±2.07* 1.28±0.42 4.65±1.42* 5.58±1.47*</td>
</tr>
<tr>
<td></td>
<td>Transmural 1.71±0.66 4.56±2.13* 5.33±1.68* 1.46±0.40 3.78±1.10* 4.28±1.22†*</td>
<td>Transmural 1.24±0.20 0.60±0.24* 0.50±0.09†</td>
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<td>Ratio endo:epi 1.11±0.12 0.75±0.30* 0.66±0.31* 1.24±0.20 0.60±0.24* 0.50±0.09†</td>
<td>Total flow (ml/sec) 1.05±0.35 2.89±1.18* 3.44±0.92* 0.92±0.34 2.45±0.47* 2.77±0.51†*</td>
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<td>Stenosis resistance (mm Hg/ml/sec, mean±SD) 5.8±2.5 11.5±2.7* 12.6±3.9* 12.0±5.1†† 20.6±4.7‡* 21.0±5.2‡*</td>
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*p<0.01 vs. respective control, †p<0.05 vs. simple stenosis, ‡p<0.01 vs. simple stenosis.

blood flow (0.73±0.14) did not differ significantly from the ratio of complex to simple stenosis for endocardial perfusion pressure 0.85±0.11. Thus, the relative decline in maximal endocardial flow (complex versus simple stenosis) was roughly proportional to the decline in effective perfusion pressure. The distal to circumflex flow ratio for endocardial and transmural layers also was significantly (p<0.01 and p<0.05, respectively) greater at the 800 µg/min dose of adenosine with the simple stenosis (2.1±0.7 and 3.1±1.0, respectively) compared with the complex one (1.4±0.3 and 2.4±0.5, respectively). Distal zone epicardial flows at the 800 µg/min adenosine dose also were higher with the simple device (seven of nine animals) in comparison with the complex one. The difference, however, was of borderline statistical significance (p=0.05). Distal zone endocardial to epicardial flow ratios declined (p<0.01) compared with respective control in response to adenosine for both simple and complex stenoses. However, the ratio was lower (p<0.05) for the complex versus the simple lesion at the 800 µg/min adenosine dose.

Mean stenosis resistance, as expected, increased significantly (p<0.01) after adenosine administration with each stenosis. However, despite the fact that flows with the complex stenosis were similar or lower compared with the simple lesion, both baseline and maximal mean stenosis resistances were greater with the complex stenosis (p<0.01) than with the simple lesion. Figure 3 demonstrates the relation between group mean (±SEM) values of total transstenosis flow and mean pressure gradient for each stenosis. A clear-cut upward shift of the curve is apparent for the complex stenosis (compared with the simple stenosis) and demonstrates the greater pressure loss across it at any given level of flow. It also is apparent that the curves diverge considerably as flow increases. Results of the in vivo study are consistent with results of the in vitro steady-flow, perfusion experiment (Figure 4). Thus, the in vitro gradient–flow curve for the complex stenosis is steeper than that for the simple stenosis and diverges from it at higher flow rates. The value of r² for each of the in vitro and in vivo curves was more than 0.98 (p<0.01). Differences in absolute values of stenosis pressure gradient between in vitro and in vivo studies, especially at higher flow rates, likely reflect added energy losses due to pulsatile perfusion in vivo. The gradient–flow curves obtained in the in vivo study are otherwise virtually identical to those obtained in the in vitro experiment.

**Discussion**

The present study tested the hypothesis that complex morphology will amplify the hemodynamic severity of a coronary arterial stenosis in comparison with a simple lesion that causes the same reduction in vessel cross-sectional area. To test the hypothesis, it was necessary to use a complex stenosis whose cross-sectional area could be accurately known, hence the five-hole device. It should be stressed that multilumen stenoses have been described in postmortem studies of human coronary arteries (Figure 16 of Reference 23). The frequency with which such lesions occur in living patients is unknown but may be more common than suspected given the high prevalence of thrombus in acute coronary syndromes.6–10 Multilumen stenoses are thought to represent an evolutionary stage of prior coronary artery thrombosis.23 Furthermore, the purpose of the present study was to use a quantifiable stenosis model capable of producing greater energy losses across it because of increased frictional drag (and possibly turbulence as well) at any given level of flow.12–14 The complex stenosis, therefore, was compared with a simpler lesion to estimate the increment in physiologic severity that could result from unfavorably altering stenosis morphology while holding minimal cross-sectional area, length, and locus constant. The estimated increment in physiologic severity produced by the multilumen device likely is conservative in comparison with naturally occurring lesions that may have more complex, irregular shapes (i.e., greater surface area) and would be expected to generate greater energy losses.

Irregular-appearing stenoses have been observed with increasing frequency both in clinical coronary angiograms6–9 and by coronary angioscopy.10 It also should be noted that some stenoses that appear simple or regular on coronary angiograms in fact may be quite irregular when viewed by angioscopy.10
Irregular, ragged, or shaggy lesions have been associated with unstable angina pectoris and an unfavorable clinical course.\textsuperscript{6-10} Plaque rupture and thrombosis are thought to be responsible for the irregular appearance of the lesion.\textsuperscript{6-10,24} It also has been hypothesized that platelet aggregation and release of vasoactive compounds (e.g., thromboxane A\textsubscript{2}, 5-hydroxytryptamine) could contribute to the unstable clinical course by either inducing large-vessel vasospasm at the site of the lesion\textsuperscript{25} or impairing arteriolar dilation distal to it.\textsuperscript{17} The effects of morphology per se on the physiologic severity of the stenosis, however, have not been considered other than in terms of reduction in vessel minimal cross-sectional area, percent diameter reduction,\textsuperscript{2,3} and length.\textsuperscript{3} Data obtained in the present study demonstrate fine details of stenosis morphology that may or may not be apparent on clinical coronary arteriograms also may contribute importantly to the physiologic severity of the stenosis.

Thus, as shown in Figures 3 and 4, stenosis pressure gradient at comparable flow levels was approximately twofold greater for the complex stenosis compared with that of the simple model. In addition, absolute values of mean stenosis resistance at both peak flow and baseline were higher for the complex stenosis even though maximal hyperemic flow was significantly lower and baseline flow slightly (albeit not significantly) lower with the complex lesion. Further, as a result of roughly doubling lumenal surface area, maximal endocardial blood flow distal to the complex stenosis was reduced by 25–30\% in comparison with the more streamlined simple stenosis. Such a reduction in flow reserve could help to explain why, other things being equal, two patients with stenoses of similar anatomic severity (i.e., minimum cross-sectional area) could exhibit very different clinical patterns. Complex stenosis morphology per se may be sufficient to convert an otherwise tolerable lesion into one associated with a more ominous clinical course and vice versa.

It is important to emphasize that the animals used in the present study were instrumented with a rigid plastic stenosis that could not change shape and that all animals were vigorously anticoagulated with heparin and pretreated with aspirin to prevent platelet aggregation and thrombosis on the stenosis. Furthermore, in a previous study with a similar animal model,\textsuperscript{17} we have shown that endothelial damage at the stenosis site is minor and that platelet aggregates either within or distal to the stenosis lumen are rare in heparinized, aspirin-pretreated animals. In addition, the order in which simple and complex stenoses were placed in the coronary circulation was randomized. Thus, it is unlikely that the results of the study can be attributed to factors such as vascular injury, coronary vasospasm, or platelet aggregation and thrombus formation on the stenosis. Because the purpose of our study was to examine the effects of stenosis morphology per se on physiologic severity of the lesion, the fixed, rigid nature of the artificial stenosis along with the ability to exclude platelet-related vasoactive and/or thrombotic effects represents a distinct advantage of the model.

A disadvantage of the preparation relates to the inability to measure instantaneous coronary blood flow. Because instantaneous values of flow exceed mean values recorded with radiolabeled microspheres, it is likely that differences between the two stenoses with regard to impedance to flow may have been even greater at some points in the cardiac cycle, particularly diastole. Similarly, moments in the cardiac cycle with very low flow may have shown smaller differences. Nevertheless, even baseline transstenotic pressure gradient for the complex stenosis was almost twice that of the simple device. The same was true of calculated mean stenosis resistance at control. Thus, on balance, inability to measure phasic flow in our study probably worked in the direction of minimizing rather than accentuating differences in physiologic severity between the stenoses. In any case, it should be stressed that measured mean stenosis gradient and flow reflect the influence of transients, both high and low, and thus are valuable in terms of describing the cumu-
lative effects of each stenosis throughout the cardiac cycle.

In conclusion, the results of the present study demonstrate that specific details of coronary stenosis morphology may have an important influence on the physiologic severity of the lesion. Such effects could be responsible, at least in part, for apparent discrepancies between anatomic assessment of stenosis severity and variables such as clinical course or functional effects of the stenosis on the coronary circulation. Conversely, streamlining stenosis morphology may represent another mechanism by which coronary angioplasty augments flow reserve in the coronary circulation.

Acknowledgments

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References


Key Words: coronary circulation • endocardium • adenosine • stenoses