Assessment of Severity of Coronary Stenoses Using a Doppler Catheter
Validation of a Method Based on the Continuity Equation

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The coronary Doppler catheter has been used primarily in the measurement of coronary vasodilator reserve, most often as the ratio of peak to resting velocity in response to an intracoronary dose of papaverine. We have developed a new method based on the continuity equation using a Doppler catheter for the assessment of stenosis severity in the coronary circulation by means of quantitative velocity measurements obtained by complex spectral analysis of the Doppler signal. With this system we have been able to detect a high-velocity stenosis jet in a canine model of coronary stenosis of known cross-sectional area. Using the peak velocity obtained by complex spectral analysis, we found a strong correlation between cross-sectional areas determined by the continuity equation and known cross-sectional areas \((r=0.93, \text{SEE}=0.23 \text{ mm}^2)\). We also found a strong correlation between the ratio of peak stenosis velocity to proximal vessel velocity and percent diameter and percent area stenosis \((r=0.91 \text{ and } 0.92, \text{ respectively})\). When the velocity was determined with conventional zero-crossing methods for these parameters, there was no correlation between calculated and known values for cross-sectional area and percent diameter or area stenosis. Measurements of the vasodilator reserve in response to intracoronary papaverine before and after implantation of the stenosis did not correlate with any of the anatomic parameters of stenosis severity regardless of the method of signal analysis (zero-crossing or complex spectral analysis). The measurement of quantitative peak coronary velocity with a Doppler catheter using complex spectral analysis may provide an accurate method for determining the severity of a coronary stenosis. *(Circulation 1989;80:625–635)*

Recently, pulsed Doppler coronary catheter measurements of coronary vasodilator reserve have been validated and used to assess vasodilator reserve in patients with multivessel coronary disease, isolated single-vessel disease, and in patients after bypass surgery and percutaneous transluminal coronary angioplasty (PTCA).1–4 Initial published data emphasized a poor correlation between measurements of vasodilator reserve and angiographic parameters of stenosis severity (both visual estimates and parameters derived from quantitative arteriography). More recently, the use of vasodilator reserve derived from Doppler catheter velocity measurements has come under closer scrutiny with questions raised about the importance of confounding variables such as coronary perfusion pressure, acute or chronic myocardial conditions that affect the coronary pressure-flow relation at maximal vasodilation, and the type of vasodilator stress employed.5,6 In a recent study by Wilson and colleagues2 of patients with limited coronary artery disease (i.e., isolated lesions in one or two vessels), vasodilator reserve was assessed with a Doppler catheter at the time of cardiac catheterization. Patients with myocardial hypertrophy, prior infarction, or segmental wall motion abnormalities were excluded. In this highly selected patient group a good correlation between three different quantitative anatomic parameters of stenosis severity and vasodilator reserve was found. It would appear from these studies that Doppler measurements of coronary vasodilator reserve pro-

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Figure 1. Schematic diagram of Doppler catheter in position to range-gate into coronary stenosis from proximal position. Note that both proximal and stenosis velocity may be obtained with the catheter in a proximal position by adjusting sample volume position on the pulsed Doppler velocimeter. V, velocity; CSA, cross-sectional area; V(Sten), stenosis velocity; V(Prox), proximal velocity; CSA(Sten), cross-sectional area of stenosis; CSA(Prox), cross-sectional area of proximal region of the vessel.

To implement a method based on the continuity equation, accurate quantitative measurement of stenosis velocity jets is necessary. Most recent studies involving intracoronary Doppler techniques have used a catheter with a side-mounted Doppler crystal that is aligned at an angle of 45\(^\circ\) to the direction of flow along with a Doppler velocimeter using zero-crossing counter techniques. Since these methods are known to be inaccurate in areas of disturbed flow and incapable of detecting true peak velocities,\(^{10,11}\) we have developed a pulsed Doppler velocimeter that analyzes the Doppler shift signal by a fast Fourier transform (FFT) method. The system is based on a commercially available pulsed Doppler catheter with an end-mounted Doppler crystal and a 20-MHz velocimeter (Millar Instruments, Houston, Texas). These standard components have been linked by a custom-designed gain control to an FFT spectrum analyzer (Meda Sonics, Mountain View, California) such that the Doppler shift signal may be analyzed by both conventional zero-cross techniques and by complex spectral analysis in real time. With this system we have been able to detect accurate peak and mean velocities from the velocity distribution in a sample volume that is aligned coaxially with the direction of coronary blood flow.

The purpose of this study was threefold: 1) to attempt to determine minimal cross-sectional stenosis area in a canine model of coronary stenosis using the continuity equation and stenosis velocities obtained by complex spectral analysis and zero-cross methods, 2) to test whether the ratio of stenosis jet velocity to proximal velocity could provide a useful measurement of stenosis severity, and 3) to compare complex spectral analysis and conventional zero-crossing methods for analyzing the Doppler shift signal from an intracoronary Doppler catheter.

**Methods**

**Doppler Catheter**

The features of this catheter have been described elsewhere.\(^{12}\) An annular 20-MHz ceramic crystal capable of both transmitting and receiving acoustic signals was mounted on the tip of a woven Dacron catheter (Millar Instruments). The catheter was 4F in the main body, with a wire weave for torque control, and the distal 20-cm tip tapered to 3F (1 mm o.d.). Two wires attached to the crystal passed within the catheter between the outer catheter sheath and an inner lumen tubing that traversed the full length of the catheter. The internal lumen was 0.015 in. and would accommodate a standard 0.014-in. PTCA guidewire. The proximal main body of the catheter terminated in a Luer hub that allowed flushing of the inner lumen with the two wires exiting and terminating in a separate connector for attachment to the velocimeter.

**Instrumentation**

A 20-MHz pulsed Doppler velocimeter, based on the design of Hartley and Cole\(^{13,14}\) and manufac-
tured by Millar Instruments, was coupled to the Doppler catheter. A 20-MHz carrier frequency was provided by a crystal oscillator. The basic frequency was divided by 320 to produce a pulse repetition frequency of 62.5 KHz with 0.4-μsec pulses containing eight cycles of 20 MHz. These pulses were amplified and converted to sound pulses by an ultrasonic transducer. Acoustic pulses were reflected by any structures in the sound beam, and the returning echoes returned to the transducer where they were compared in phase to two signals from the master oscillator. The quadrature phase detector had two outputs with a 90° phase difference between them, representing the instantaneous phase difference between the echo signal and the 20-MHz carrier frequency with the sign of the phase shift determined by the direction of motion (toward or away from the transducer). Since the echoes from each reflector returned to the transducer delayed in time proportional to distance, sampling the reflected signal after a certain delay corresponded to sensing motion at a specific distance from the transducer (range gate), which for this instrument varied from 1 to 10 mm from the catheter tip. The quadrature audio signals were filtered to remove high frequency components at the pulse repetition frequency and low frequency components below about 200 Hz and then were processed by a frequency-to-voltage converter or broadcast by a speaker. The unamplified audio signal was also directed to external outputs for recording or further decoding (in this case for complex spectral analysis by FFT).

The quadrature audio signal was processed internally by a zero-crossing detector that operated as follows. The two audio inputs were converted to square waves by a dual comparator with an offset trigger that created a dead zone around zero. Individual sensitivity controls were set internally such that background noise was within the dead zone and signal above it. The comparators triggered pulse generators, one of which produced positive pulses and the other negative pulses. They were mutually inhibited so that pulses were only produced by one generator at a time. The resulting pulse trains were summed and averaged, producing a voltage proportional to the pulse frequency with the sign indicating the direction of the velocity at that point in time. In our laboratory this output was recorded on a Gould four-channel recorder with channels for phasic velocity with a low-pass cutoff frequency of 50 Hz and for mean velocity with a low-pass cutoff frequency of 0.25 Hz.

The unamplified audio signal (through the external output) was also directed to a spectrum analyzer (Model SP25A, Meda Sonics) via a circuit that provided amplitude attenuation with a custom-designed gain control. The quadrature audio signals underwent internal amplification and then passed to two analog-to-digital converters that sampled the analog signals and converted them to a stream of digital samples. The sampling rate was adjustable. Digital samples were temporarily stored in a data buffer, and periodically 256 digital samples from each channel were transferred to the Fourier processor. Here, an FFT was performed on the data, converting the 512 input samples into 256 frequency amplitudes with one complete FFT executed in about 0.005 second. The frequency amplitudes generated by the Fourier processor were compressed in logarithmic form to minimize memory requirements and were periodically transferred to the display memory. The output from the display memory was gray scale mapped and converted into a video signal by the video generator. This output was passed to a standard VHS video cassette recorder and then to a high resolution monitor via the video input and output jacks. For our experiments audio and spectral data were recorded on standard ½-in. videotape during the study.

Coronary Stenosis Implants

The production and placement of these implants have been described previously.\textsuperscript{15} They were fabricated from urethane plastic and precision molded into tapered cylindric sections approximately 20 mm in length and narrowing from about 3.8 mm proximally to 2.5 mm distally. The internal lumen was either round or irregular in shape and of known dimension (Figure 2). Sections 2 mm in length were cut from each conical section to produce individual implants of known external diameter that were used to create discrete coronary stenoses in each canine preparation. Vessel size was estimated from coronary angiography performed at the time of the study, and stenosis implants of particular external diameters were selected so that the implant would settle in a straight portion of the coronary artery away from major side branches.
**Animal Studies**

Eleven large mongrel dogs (25–35 kg) were anesthetized with pentobarbital sodium and ketamine and mechanically ventilated with room air. A 12F sidearm sheath was placed in the left carotid artery for arterial access. An 8F K-9 guiding catheter (Advanced Cardiovascular Systems, ACS, Mountain View, California) was advanced via the left carotid artery to the left main coronary ostium with position confirmed by contrast injections (Hypaque-76). The Doppler catheter, preloaded with a 0.014-in. high torque floppy guidewire (ACS), was advanced to the left main ostium and the guidewire advanced to the distal circumflex coronary artery. The Doppler catheter was advanced to a proximal or midposition in the circumflex, and velocity signals were obtained. Velocity signals were optimized with both the spectral display and the audio signal. Papaverine 3–9 mg was administered via the guiding catheter and the hyperemic response recorded. Hyperemia runs were repeated one to three times and the results averaged. Papaverine-induced hyperemia was also measured in the left anterior descending coronary artery (LAD) in the same fashion. The Doppler catheter and guidewire were then removed, and a 0.035-in. guidewire was placed in the circumflex coronary artery. The guiding catheter was removed, and a stenosis implant, 2 mm in length, was passed over the guidewire into the circumflex and wedged into place with a "push catheter" fashioned from an 8F guiding catheter with the secondary bend removed. Position of the stenosis was confirmed angiographically and the 0.035-in. guidewire removed.

The Doppler catheter/0.014-in. guidewire system was again advanced into the circumflex and the guidewire directed through the stenosis and out distally in the vessel. The Doppler catheter was advanced to a position just proximal to the stenosis where velocity recordings were obtained. The range gate control was then adjusted such that the sample volume moved out from the catheter tip (usually about 3–5 mm) to insonify the region of the stenosis without moving the catheter itself. Several recordings were obtained in both the stenosis high velocity jet position and the proximal position. Care was taken to ensure that the highest velocity during the range-gating procedure was recorded. Next, the catheter was moved back to the immediately proximal position, and measurements of papaverine-induced hyperemia were obtained using 3–9 mg injected through the guiding catheter. The entire process was repeated for the LAD (stenosis implantation, measurement of high velocity stenosis jet and proximal velocity, and repeat hyperemia runs) (Figure 3).

At the end of the experiment, the dog was killed using high concentration intravenous potassium chloride. The heart was harvested, and the coronary arteries were examined by dissection to assess the position of the stenosis implants and check for evidence of thrombus formation or gross intimal damage. The stenosis implants were then removed, and external and internal diameters (e.d. and i.d., respectively) were measured with digital calipers (Mitutoyo, Tokyo, Japan), and cross-sectional areas were derived from the relation \( A = \pi r^2 \) for a circle for the proximal portion of the vessel (external diameter of the stenosis implant) and for the stenosis. For the irregular orifice shapes the implants were photographed under \( \times 24 \) magnification, and the internal area was planimetered with a digital planimeter (Tamaya Technics, Tokyo, Japan). The percent diameter stenosis and percent area stenosis were calculated for all implants with circular orifices as \( e.d. - i.d. / e.d. \times 100 \) and proximal area–stenosis area/proximal area \( \times 100 \), respectively. The percent area stenosis was calculated for the stenosis implants with irregular orifice shapes.

**Data Analysis**

Peak and mean velocity determinations from the spectral data were obtained as follows. Spectral data from the spectrum analyzer were transferred into a host computer (Compaq 386) and stored for postprocessing. With the use of an algorithm for the determination of peak and mean frequencies from the spectral display (Meda Sonics) modified for implantation on the host computer, time-averaged peak and mean velocities could be determined. Peak and mean velocities for each beat were obtained by averaging instantaneous velocity data.
FIGURE 4. Tracing of velocity data from the zero-cross detector during papaverine-induced hyperemia run to assess vasodilator reserve. Values used for instantaneous peak phasic and mean velocity during the study are indicated by the small arrows. Large arrow indicates time of papaverine administration.

over a one-beat time interval. Several beats (four to eight) were averaged to obtain a peak and mean velocity determination for each condition (proximal to stenosis, in stenosis jet, resting, and hyperemic velocities). To select beats for analysis, the entire recording was reviewed, and as many as four high-quality beats were selected and freeze-framed for uploading to the computer. This procedure was done for as many beats as possible for each run (all high quality beats were analyzed): resting, hyperemia, proximal vessel, or stenosis velocity. Several screens of data had to be uploaded in most cases, but no more than eight beats were analyzed for each condition.

For data from the zero-crossing detector, instantaneous peak phasic and mean velocities were taken as the peak of each tracing from the recorder for each condition (Figure 4). Stenosis cross-sectional area was determined using the continuity equation and solving for stenosis cross-sectional area:

$$A_{\text{sten}} = V_{\text{prox}} \times A_{\text{prox}} / V_{\text{sten}}$$

where $$A_{\text{sten}}$$ is stenosis cross-sectional area, $$A_{\text{prox}}$$ is cross-sectional area of segment immediately proximal to stenosis, $$V_{\text{prox}}$$ is velocity in normal segment just proximal to the stenosis, and $$V_{\text{sten}}$$ is velocity in stenosis. The ratio of stenosis velocity to proximal velocity was calculated for every stenosis implant as $$V_{\text{sten}} / V_{\text{prox}}$$. Vasodilator reserve was assessed as the ratio of peak to resting blood flow velocity after a maximally vasodilating dose of intracoronary papaverine. Determinations of stenosis cross-sectional area and $$V_{\text{sten}} / V_{\text{prox}}$$ were made for each velocity parameter measured: spectral peak velocity, spectral mean velocity, zero-cross peak phasic velocity, and zero-cross mean velocity. Determinations of vasodilator reserve were likewise made for all four velocity parameters.

Statistical Analysis

Cross-sectional areas derived from the combination of stenosis velocities and the continuity equation were compared with known cross-sectional areas. Stenosis velocity ratios were compared to three anatomic parameters of stenosis severity using linear regression analysis for each velocity parameter measured. To quantify the strength of the association of these variables, the Pearson product-moment correlation coefficient was also calculated for each velocity parameter measured. To assess the variability about the regression line for each observation, the standard error of the estimate (SEE) was calculated for the four velocity parameters measured. Finally, as an additional measure of the accuracy of the technique, the mean absolute difference between the derived cross-sectional area and the known cross-sectional area was calculated and given as mean±SEM. The significance of the change in coronary vasodilator reserve with the presence of the stenosis implant was assessed with a Student’s t test. The strength of the association of vasodilator reserve with the anatomic parameters of
stenosis severity were assessed with the Pearson product-moment correlation coefficient.

Results

Stenosis Implants

Eleven dogs had 21 stenosis implants into their coronary arteries (left anterior descending or circumflex arteries). There were two dissections and acute vessel closures during placement, and two implants were noted to be tipped obliquely within the vessel by angiography, later confirmed at autopsy. Significant aliasing of the Doppler signal precluded analysis in two implanted coronary arteries, and in one implanted coronary artery the Doppler signal was of such poor quality that spectral analysis and signal processing were not possible. A total of seven implanted coronary arteries were therefore excluded from analysis, leaving 14 coronary arteries with successfully placed stenosis implants suitable for analysis. Twelve of the stenosis implants had circular orifices with cross-sectional areas ranging from 1.27 to 2.60 mm²; two had irregular orifices with planimetered cross-sectional areas of 2.08 and 2.16 mm² (Figure 2). Stenosis diameters ranged from 37% to 67%, and stenosis areas ranged from 60% to 89%.

Velocity Measurements

Proximal vessel velocities were recorded as the velocity in the segment just proximal to the stenosis implant. These typically were similar to the velocity in more proximal segments of the vessel. With the range-gate control on the pulsed Doppler velocimeter, the sample volume could be moved further out from the catheter tip. Generally, proximal velocities were obtained in the 2.0–2.5 mm range (distal to the catheter tip) with analyzable signals obtained by range-gating as far out as 6.0 mm. There was a clear increase in velocity as the range-gate control moved the sample volume into the region of stenosis in all successfully implanted vessels. In most cases, the signal was of high quality (Figure 5). The highest velocities obtained by range-gating into the stenosis were used as the stenosis velocity. By using the range-gate control to move the sample volume from an area of low velocity proximally into the region of stenosis without moving the catheter into the stenosis itself, further obstruction by the catheter in the stenosis and artificial elevation of the stenosis velocity were prevented.

Stenosis Cross-Sectional Area

Cross-sectional areas derived from the continuity equation, using the spectral peak velocity as the velocity term, correlated best with true cross-sectional area, giving a correlation coefficient of 0.93. The regression line for these two variables closely approximated the line of identity with a slope of 1.12 and y intercept of 0.10; the SEE for the cross-sectional area determined from the spectral peak velocity was 0.23 mm² (Figure 6). Cross-sectional areas using the spectral mean velocity were only weakly correlated with true cross-sectional area, giving a correlation coefficient of 0.53. There was no correlation between cross-sectional areas using either zero-cross peak phasic velocity or zero-cross mean velocity and true cross-sectional area (Figure 6). The mean absolute difference from the true cross-sectional area for the cross-sectional area determined from the spectral peak velocity was 0.22±0.03 mm² (mean±SEM), compared with 0.51±0.12 mm² for cross-sectional area determined from the spectral mean velocity, 0.61±0.15 mm² for cross-sectional area determined from the zero-cross peak phasic velocity, and
0.65±0.12 mm² for cross-sectional area determined from the zero-cross mean velocity.

**Stenosis Velocity Ratio**

We also examined the relation of the stenosis velocity to proximal vessel velocity ratio (V_{sten}/V_{prox}) and three anatomic parameters of stenosis severity, namely, percent diameter stenosis, percent cross-sectional area stenosis, and minimal cross-sectional area. There was a strong correlation between V_{sten}/V_{prox} and percent diameter and percent area stenosis when the spectral peak velocity was used as the velocity term (r=0.91 and 0.92, respectively) (Figure 7). There was also a correlation between the stenosis velocity ratio and minimal cross-sectional area measured in the same fashion, although not as strong (r=-0.76). When the spectral mean velocity was used as the velocity term in the V_{sten} to V_{prox} ratio, the correlation with the three anatomic parameters of stenosis severity was weak: r=0.70 for percent diameter stenosis, r=0.65 for percent area stenosis, and r=-0.56 for minimal cross-sectional area. There was no correlation between V_{sten}/V_{prox} and the anatomic parameters of stenosis severity when the zero-cross peak phasic or zero-cross mean velocities were used as the velocity term (r=0.34 to −0.31 for peak phasic and 0.27 to −0.14 for mean velocity).

** Vasodilator Reserve**

Vasodilator reserve was measured as the peak to resting velocity ratio before placement of the coronary stenosis in the portion of the coronary artery selected as the site of stenosis implantation and again in that same site after the stenosis was implanted. Papaverine 3–9 mg was administered through the guiding catheter for each measurement; most animals achieved maximal hyperemia with 6 mg. There was a statistically significant decrease in mean vasodilator reserve when the hyperemic response in all vessels was averaged (p<0.001–<0.002) using all of the four methods of signal analysis (Figure 8).

**Discussion**

The coronary Doppler catheter has been used primarily as an instrument to measure the reactive hyperemic response in the human coronary circulation as an indication of the severity of a coronary stenosis. Investigators at the University of Iowa were the first to validate a subselective coronary Doppler catheter that could be placed in branches of major epicardial coronary arteries in humans at the time of cardiac catheterization. This catheter, which uses a Doppler crystal mounted at an angle of 45° to the vessel wall on the side of the catheter proximal to the tip, has been used in several studies to measure the peak to resting velocity ratio in human coronary arteries, most often in response to a dose of intracoronary papaverine. Since a velocity ratio (more accurately, a kilohertz shift ratio) is always measured, no attempt has been made with this catheter and analysis system to
make quantitative statements about coronary flow velocity. The catheter used in our study, which uses an end-mounted Doppler crystal, is better suited to quantitative flow velocity measurements since the Doppler sample volume is aligned coaxially with the flow stream, and angle corrections in the Doppler equation should not be necessary. This system could potentially be used to measure absolute coronary flow velocity, although the validity of this measurement was not directly assessed in the present study. The movable guidewire system, which is usually placed through a stenosis and out distally in the vessel, should help to keep the catheter aligned down the center of the vessel and, hopefully, in the core of the forward streamline of flow. The guidewire may be particularly important in allowing positioning of the sample volume within the region of the coronary stenosis, which was critical in our study.

Flow interference effects from the catheter were felt to be a potential problem with the end-mounted design; indeed, absence of flow interference has been claimed as a major advantage of the side-mounted design of the Iowa catheter. In studies using flow models designed to replicate the coronary artery with the catheter in position, we have found that flow interference effects extend about one catheter diameter, or about 1 mm beyond the tip for flows in the range of coronary flow (20–100 ml/min). Since all of our measurements are made with the sample volume at a distance of at least 2 mm from the tip, flow interference effects are believed to be negligible in this preparation.

Previous studies employing coronary Doppler catheters have used an analysis system based on the zero-crossing detector to evaluate the Doppler shift signal. Our study is the first that has used complex spectral analysis by FFT to measure the Doppler shift from a coronary Doppler catheter. Complex spectral analysis has several major advantages over the zero-crossing detector. In a nondisturbed, laminar flow field neighboring targets are moving at the same velocity, so that all red cells within a given sample volume have the same velocity. The zex-cross method, which computes an average velocity in the sample volume at each point in time based on the frequency of zero crossings of the complex Doppler shift signal, is theoretically accurate for this type of flow. However, in the case of disturbed flow, or given a nonuniform flow profile (i.e., parabolic), the zero-cross method computes only an average velocity in the sample volume; the true peak velocity cannot be measured. This problem has been documented in human coronary arteries by Kajiya and coworkers who showed that the zero-cross method is inaccurate in disturbed flow fields and that only FFT analysis techniques are capable of detecting the characteristic spectral broadening. This observation is particularly true in regions near coronary stenoses where turbulent effects are prominent. Furthermore, zero-cross methods are subject to errors caused by motion of tissue

**Figure 7.** Plots of regression data for the ratio of stenosis jet velocity to proximal velocity and three anatomic parameters of stenosis severity: % diameter stenosis, % cross-sectional area stenosis, and true stenosis minimal cross-sectional area (CSA). Peak velocity from complex spectral analysis was used as the velocity term. V(STEN), stenosis jet velocity; V(PROX), proximal velocity; SEE, standard error of the estimate.
structures other than blood within the beam. With Doppler catheters movement of the walls of the coronary arteries within the ultrasound beam is frequently detected as a "wall thump" artifact. With care the Doppler catheter may be positioned within the vessel so as to minimize wall thump artifacts, but frequently they cannot be eliminated completely. The zero-crossing detector erroneously represents this artifact as a high velocity signal, while FFT shows it properly as a high amplitude but low velocity signal. The results of the present study, showing that the peak velocity determined from complex spectral analysis is most accurate in determining the anatomic severity of a coronary stenosis, suggest that FFT processing may be the most reliable technique for analyzing the Doppler shift signal when measuring coronary stenosis jet velocity with a Doppler catheter.

There are several important concerns in the interpretation of our data. Theoretically, the continuity equation applies to measurements using mean flow in a nonbranching system. Therefore, one would predict that the true mean velocity should be most accurate in making vessel cross-sectional area assessments based on continuity of flow. However, our data indicate that the cross-sectional area determined with the spectral mean velocity has only a poor correlation with true cross-sectional area of a stenosis and that velocities determined with zero-crossing methods, which theoretically are truly based on the average frequency in a sample volume of moving red blood cells, give cross-sectional areas that do not correlate at all with true stenosis cross-sectional areas. On the other hand, cross-sectional areas using the spectral peak velocity were highly correlated with true cross-sectional areas. The discrepancy may arise from errors in the measurement of mean velocity because of a number of factors. First, the Doppler sample volume with this catheter system is a three-dimensional structure with a length of approximately 0.45 mm. The sample volume may overlap regions of flow proximal to the stenosis even though it is predominantly positioned in the stenosis jet. This overlap would lower the calculated mean velocity to a variable extent based on the degree to which the sample volume involves these proximal regions. The peak velocity is not subject to error introduced by these lower velocity components. Second, in our system we were able to reduce wall thump artifacts with high-pass filters although these filters also factor out potentially important low velocity components and introduce error into the calculation of true mean velocity. This effect is particularly important in a disturbed flow field where spectral broadening occurs. Third, the Doppler beam may be narrow relative to the

**Figure 8.** Plots of changes in vasodilator reserve as assessed by the peak-to-resting velocity ratio after a maximally vasodilating dose of intracoronary papaverine. Vasodilator reserve was measured pre- and postplacement of the stenosis implant using the four velocity parameters derived from complex spectral analysis or the zero-crossing detector. While the mean change in vasodilator reserve for all vessels studied was statistically significant (p < 0.001 to p < 0.002) by all four velocity parameters, there was no correlation between the change in vasodilator reserve and any of the anatomic parameters of stenosis severity (r = −0.50 to 0.53).

Johnson et al  Doppler Assessment of Coronary Stenosis
Entrance effects lead to another theoretic concern with our measurements: the increase in velocity in the region immediately proximal to the stenosis. If proximal velocity determinations are made too close to a stenosis, the measured velocities will be spuriously elevated. Model calculations suggest that the area of acceleration of velocities occurs about one step diameter before the stenosis, corresponding to 1–2 mm for the stenosis implants used in our study (Figure 9). Since the sample volume was always range-gated at least 3 mm from the proximal area to obtain stenosis velocities in our study, entrance effects on proximal velocities should not have introduced additional error.

Another important limitation of the model presented here is the fact that the stenoses are “ideal” in the sense of being discrete and located in relatively straight segments. With longer or more complex stenoses, jets may be more difficult to record and may be particularly difficult to record in tortuous segments where, despite the centering effect of the guidewire on the catheter, the beam may not be directed into the peak velocities of the jet. Our model also ignores any dynamic component of actual stenosis geometry although in theory the Doppler technique allows for an instantaneous assessment of jet velocity and lesion cross-sectional area. In this ideal setting our method compares favorably with quantitative coronary arteriography and videodensitometry based on previously published data in a similar canine model of implanted stenoses. It would be useful, however, to compare these techniques directly using this model.

Finally, it is important to note that in our study changes in vasodilator reserve showed no correlation with any of the anatomic parameters of stenosis severity. This observation is inconsistent with findings in previous animal studies that increasing stenosis severity lowered vasodilator reserve in a predictable fashion. In the present study, however, it is likely that resting flow was altered by ischemia as a result of placement of the stenosis, manipulation of the large diameter intracoronary guidewire, and prolonged engagement of the 8F guiding catheter. This effect is comparable to the situation seen clinically when measuring Doppler flow reserve during percutaneous transluminal coronary angioplasty where baseline conditions may be altered by induced ischemia. Thus, ischemic change has emerged as an important limitation of the vasodilator reserve method in this context.

The Doppler continuity method described here is theoretically independent of any alterations in coronary vascular tone and can be potentially applied in patients with left ventricular hypertrophy, other forms of diastolic dysfunction, and prior myocardial infarction. In this ideal setting our method appears to give an accurate determination of minimum lesion cross-sectional area, which is based on a straightforward fluid dynamics calculation. It should be emphasized, however, that although the velocity

vessel diameter. Particularly in the case of proximal recordings, the catheter may rest off center in the vessel so that a relatively high proportion of low velocity signals from the region of the vessel wall are represented in the mean velocity determination. Nevertheless, the peak velocity measurement will be accurate as long as some portion of the beam insonifies the high velocity center stream.

Additional important fluid dynamic issues arise in making these measurements with an intracoronary Doppler catheter. In all of the stenosis implants studied, the guidewire was placed through the stenosis and out distally in the coronary artery. The guidewire thus reduces the actual cross-sectional area of the stenosis. The 0.014-in. guidewire used in our study has a cross-sectional area of 0.10 mm² so that on this basis the cross-sectional area of the stenosis should be underestimated by 0.10 mm². The true cross-sectional area of the stenosis jet in our preparation would also be reduced by the “vena contracta” phenomenon: the effective area of flow through the stenosis is somewhat less than the cross-sectional area of the narrowing. This phenomenon is particularly important in the case of an abrupt, nontapering entrance region, as shown in the schematized stenosis implant in Figure 9. For the ratio of stenosis to proximal area of 0.6–0.9 in this study, the cross-sectional area of the stenosis jet (the vena contracta) would be expected to be in the range of 10–30% smaller than the actual cross-sectional area of the stenosis with greater underestimation of area for tighter stenoses. This effect would contribute to the negative y intercept and slope greater than unity in the regression line for the spectral peak velocity presented in Figure 5.

Figure 9. Schematic diagram of a coronary vessel with an implanted stenosis showing an abrupt, nontapering entrance region and its effect on the flow profile. The region of acceleration of velocity occurs about 1 step diameter back from the stenosis. The actual cross-sectional area of the stenosis jet, the vena contracta, is less than the anatomic cross-sectional area of the stenosis. The continuity equation would therefore underestimate anatomic cross-sectional area, with greater underestimation as the stenosis becomes tighter. See text for details.
ratio method is a new way of looking at relative cross-sectional areas, it does not escape the limitations inherent in lumen area measurements. Specifically, the method does not account for the fact that the extent of disease of the proximal, reference segment is unknown. In addition, although the velocity ratio method gives an estimate of minimal cross-sectional area, this parameter itself is not a sufficient or complete measure of the physiologic impact of a stenosis. We anticipate that the main practical utility of the velocity ratio method will be in the context of catheter-based interventions, where the goal is to remodel a stenosis in order to create a local flow condition that is more similar to the adjacent regions. Measurement of the velocity ratio can be performed quickly and easily in this setting and may provide additional information that helps to clarify the physiologic significance of a particular lesion.

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