critical evaluation of the double sucrose gap voltage clamp technique by other investigators using this technique in order that the limitations of the technique can be better defined and the tension-voltage-current relationships in frog myocardial tissue better understood.

References

Measurements of Disordered Flows Distal to Subtotal Vascular Stenoses in the Thoracic Aortas of Dogs

DON P. GIDDENS, PH.D., ROBERT F. MABON, M.D., AND ROBERT A. CASSANOVA, PH.D.

SUMMARY Instantaneous blood velocity measurements employing a constant temperature hot film anemometer were obtained in the region distal to externally enforced, subtotal vascular stenoses in the descending thoracic aortas of anesthetized dogs. Our objectives were to determine alterations in velocity waveforms and energy spectra as the degree of stenosis was increased. We paid particular attention to descending thoracic aortas of anesthetized dogs. Objectives were

The predilection for cholesterol-laden plaques to develop at certain discrete sites in the vasculature has prompted numerous investigators to hypothesize that the local hemodynamic environment is an active agent in the pathogenesis and proliferation of atherosclerosis. Although several causative factors have been suggested, among the more prominent are turbulence and unstable stress patterns. Turbulence also has been proposed as a probable mechanism in the production of poststenotic dilation. It has been postulated that turbulence within the blood vessel may cause intimal injury by increasing the local shear stress acting on the endothelial surface or by a vibrational effect akin to the phenomenon of fatigue in engineering materials.

There is also experimental evidence that unstable flow patterns lead to a randomness in endothelial cell orientation. Any of these mechanisms might diminish the effectiveness of the normal vascular barrier to lipid accumulation within the intima.

Although the existence of turbulence has been reported in canine and equine aortas, little is known of the extent of its occurrence in the normal vasculature of man. However, the production of an unstable, fluctuating flow distal to obstructions in a relatively large artery is a natural supposition of the fluid dynamist. Physicians often have attributed various sounds and bruits to turbulence; and recently, direct verification in vivo of the existence of intensely turbulent fields in the presence of vascular stenoses was reported. An interesting corollary of these fluctuating velocity fields created by a pathological state is the potential for utilizing characteristics of the disordered flow to interpret the struc-
ture and location of the diseased site. Attempts at identifying localized vascular stenoses through interpretation of sounds created by turbulence are routinely undertaken by the clinician, using a stethoscope. Recently, Lees and Dewey\(^7\) have reported on the implementation of externally applied transducers for receiving sounds generated within stenosed carotid and femoral arteries and on use of the power spectra of these signals to estimate the reduction in vessel diameter in the occluded region.

Previously reported fluid dynamic studies of stenotic flows have focused on simulation of arterial stenoses in vitro.\(^{6,9,10}\) These investigations have for the most part employed steady mean flow through constrictions in rigid tubing, although some aspects of pulsatile flow were also considered.\(^6,11\) Energy spectra for the distal velocity fields generally indicate greater turbulence as the degree of stenosis is increased. We have completed a series of studies on pulsatile flow in vitro which were designed to provide basic information for guidance of work in vivo. These experiments are described elsewhere.\(^{12-13}\)

The work reported here is derived from measurements in vivo of the velocity fields created by subtotal, externally placed vascular stenoses imposed on the descending thoracic aorta of mongrel canines. The characterization of these fields in terms of velocity waveforms and their associated energy spectra should contribute to the description of hemodynamics in occlusive vascular disease and delineate possibilities for earlier and more quantitative diagnosis of localized vessel constrictions.

**Methods**

**Instrumentation**

**Volume Flow Rate**

The instantaneous volume flow rate was measured with a Carolina Medical Electronics electromagnetic flowmeter. A cuff-type probe was located at a site several centimeters distal to the region where the velocity was studied.

**Velocity**

The local instantaneous velocity was obtained by a DISA Electronics model 55D01 anemometer and 55D10 linearizer. We employed hot film probes (DISA Electronics) mounted on the conical tip of a hypodermic needle 1.0 mm in diameter or a catheter 2.0 mm in diameter. Since the calibration curves, hence the linearization of the velocity vs. voltage output, change with different fluids, it was necessary to calibrate each probe in the blood of the dog under investigation.

The right angle, hypodermic needle probe was placed within the vessel in the following manner. It was aligned axially, facing proximally, by retracting it against the vessel wall where it could be palpated. It then was rotated until judged to be parallel to the longitudinal axis of the aorta. This technique is satisfactory because a conical-tipped probe is not sensitive to small errors in alignment, as has been verified repeatedly during experiments in vitro. The probe was positioned radially by traversing it across the vessel lumen using a micrometer. The velocity drops to zero when the probe presses against the vessel wall. These positions can be noted on the micrometer and the midline thus determined. This degree of control is not possible with the catheter probe, and measurements taken with this device are subject to errors in positioning, particularly in the radial direction.

It is important to designate the inherent limitations of hot film anemometry. One handicap obviously is that the presence of the sensor and its mounting structure may disturb the flow being measured. This is a difficulty in any experimental study. In the present work this problem was minimized by using probes which were small in diameter (1.0- to 2.0-mm probe body) in comparison with the internal dimensions of the blood vessel (12-16 mm in diameter). A second limitation of the hot film probe is its inability to sense direction of the flow. Accordingly, the conical-tipped instrument must be used in regions where the total velocity component of the flow is essentially aligned with the axis of the shank upon which the sensor is mounted. Furthermore, probes mounted on the tips of needles or catheters cannot accurately measure reverse flow.

Another difficulty in using these probes to measure blood flow results from the fact that the velocities to be measured range from zero to approximately 100 cm/sec. Precise linearization of the calibration curve over such a span is difficult. In the procedures employed in the present study the linearization was accurate to within ±3 cm/sec. However, at low velocities this variation, although small, represents a substantial percentage error. This, coupled with the fact that reversal of flow may occur in the aorta during parts of the cardiac cycle, means that inaccuracies may be present during the low velocity phase of the measured waveform. This point will be discussed further in the section describing the analysis of data.

Finally, if the flow is essentially unidirectional and the probe is aligned properly, little error can be expected in describing the major velocity component. However, if fluctuating velocities occur in a turbulent flow, the signal detected by the hot film element actually is a composite of the fluctuating components which cannot be resolved with a single sensor.

Despite these limitations of hot film probes, they are extremely valuable in studies of blood flow. During much of the cardiac cycle the flow in arteries is forward and, in the straight segments of these vessels, is primarily axially oriented. The response of these probes up to the frequencies reported here (1,000 Hz) is good under these conditions. We have compared measured energy spectra obtained for turbulent flows in pipes in our laboratory with data from other investigators,\(^14\) and the spectral shapes are in good agreement. Furthermore, we have compared results obtained by these hot film probes with data obtained by use of a laser velocimeter (DISA model Mark II) to measure energy spectra in turbulent water flow. The curves agree to a point at which Doppler ambiguity occurs for the laser system (approximately 200 Hz for the particular optical configuration employed). Thus, we believe that velocity and spectral data obtained with hot film probes provide valuable data in blood flow studies.

**Electrocardiogram**

As an aid in maintaining reasonably normal and constant status in the dog, the ECG was monitored and recorded on...
tape. This information also furnished reference points in the cardiac cycle which may be useful to describe events in the measurement of other parameters such as the velocity waveforms.

**Viscosity**

The blood viscosity was measured from samples drawn from the right femoral artery at selected times during the experiment. A Wells-Brookfield microviscometer (cone-in-plate) was employed and the non-Newtonian apparent viscosity was determined at 37.5°C over a shear rate range from 11.5 to 230 sec⁻¹. A plot of apparent viscosity vs. shear rate was extrapolated, if required, to give an estimate of the high shear rate asymptotic value of the viscosity coefficient, \(n_w\), which is used in the calculation of the Reynolds number.

**SURGICAL PROCEDURES**

The animals used were mongrel dogs, weighing 18−25 kg. Each dog was anesthetized initially with 2.5% thiopental sodium (Surital sodium) (10−20 ml, iv). This was followed immediately by injection of atropine sulfate (0.4 mg, iv) and later, sodium pentobarbital (150 mg). The dog was maintained in a satisfactory anesthetized state by administering sodium pentobarbital at a rate of 200 mg/hour.

The thoracic descending aorta was exposed for a distance of approximately 15 cm. An electromagnetic flowmeter was placed about the vessel at the most distal accessible position above the diaphragm. The probe was then inserted into the aorta and stationed in the midline.

Several precisely measured, notched, plastic strips were placed at predetermined locations about the aorta for the extravascular occlusions. These strips then were attached individually to a device specially fabricated to cause varying degrees of occlusion by use of a micrometer adjustment. This device gives a measurement of external occlusion diameter. The internal diameter is computed by increasing the constriction until flow is totally obstructed, noting the resultant external diameter, and then assuming that the vessel wall is deformed isovolumetrically at each stage of occlusion. This method was checked for the case at zero occlusion by comparing the determined value with the internal diameter ascertained by traversing the hot film probe across the lumen as described earlier. Such comparisons were consistently within 1.0 mm. The strips employed as constricting bands were 3.0 mm in width. The length of the resulting stenosis length consequently ranged from 4.0 to 7.0 mm as the occlusion passed from mild to severe. At the conclusion of the experiment the dog was killed by injection of potassium chloride through a jugular vein.

**DATA REDUCTION**

The intra-aortic pressure, blood volume flow rate, anemometer signal, linearized output (velocity), ECG, and pressure indication of the respiratory cycle were recorded on an Ampex model 1300, 14-channel FM tape recorder for subsequent analysis.

**Mean Volume Flow Rate**

The recorded instantaneous volume flow rate signals were integrated to obtain mean values.

**Velocity Waveform Analysis**

There are several important methods for analysis of the velocity waveform. Two important examples are (1) time ensemble averaging of velocity waveforms, and (2) determination of various energy spectra. Each of these is discussed briefly.

**Time Ensemble Averaging.** This is accomplished by digitizing the velocity waveform, always beginning the digitization at a specific point in the cardiac cycle, and forming a linear average over many heart beats which have the same duration. The onset of the QRS complex of the recorded ECG signal is used as a reference to ensure correct triggering at the same point in each cycle. This trigger activates a variable delay circuit which is set to give a 1-μsec voltage pulse at any desired time subsequent to the ECG trigger. This pulse is used to initiate the analog-to-digital conversion in the Hewlett-Packard model 5451A Fourier analyzer. Typically, 200 analog signals 1 second in duration are analyzed to form a time ensemble average, each analog signal being represented by 512 digital points. For the heart rates of the animals under study, each complete velocity waveform is represented by over 200 discrete points. Use of the velocity waveform itself as the triggering signal rather than the ECG is not sufficiently accurate because of variations in the velocity waveform from cycle to cycle, particularly once turbulence develops in a poststenotic flow.

**Energy Spectra.** The energy spectrum of a velocity signal is a measure of the kinetic energy (square of the velocity) contained within a frequency band width, \(\Delta f\). A steady, laminar flow would have energy contained only at zero frequency, whereas the presence of turbulence would distribute energy into higher frequencies. There are several approaches to studying the energy spectra, each offering certain information which complements that obtained with the others.

1. **Analysis of total waveforms:** In this method the entire velocity waveform is analyzed. For the data reported here the frequency band width was 1 Hz, and 50 samples of the velocity signal, each comprised of 1,024 digitized data points, were employed. The duration of each analog sample obtained from the tape was 1 second. The maximum frequency shown in the figures is 512 Hz. Higher frequencies in the analyzed signal were filtered out to prevent aliasing.

2. **Analysis of segments of waveforms:** Energy spectra can be obtained for specific segments of the velocity waveform by using the ECG triggering method described above and by sampling data for a desired time interval. As the sampling time is decreased, the resolution of the frequency band width becomes wider since the band width, \(\Delta f\), is inversely proportional to the sampling time, \(T\). For the time-resolved data reported here the sampling time was 0.1 second and \(\Delta f\) was 10 Hz. The maximum frequency studied was 1,280, and 50 samples were taken to form the energy spectra.

3. **Analysis of velocity disturbance:** It is possible to obtain a disturbance velocity, \(u'(t)\), by subtracting the time ensemble waveform from a given velocity waveform. This process also can be accomplished for segments of the cycle.

4. **Analysis of time ensemble average:** Another method of examining the energy spectra is to perform a frequency
analysis of the time ensemble average itself. This type of analysis may prove useful in searching for vortex shedding frequencies.

We have endeavored to discuss briefly various methods of studying disordered arterial flows and to point out the specific types of information which can be provided by these techniques. Clearly, to perform an exhaustive study along each line would be a monumental task and would require considerable space to report. Although we have performed analyses using each of these techniques, for the present paper we have focused attention on presenting time ensemble waveforms and on analyzing the energy spectra for the entire velocity waveform. Illustrative examples will be given for alternative ways of investigating the nature of flow disorder.

Results

We performed a series of seven experiments in which velocity waveforms were recorded at various locations distal to partial occlusions of the descending thoracic aorta. The important similarity parameters for a confined pulsatile flow are the frequency parameter, \(\alpha = R \sqrt{\omega/\nu}\) (where \(R = \) radius, \(\omega = \) fundamental heart frequency in radians per second, and \(\nu = \) kinematic viscosity), and the Reynolds number, \(Re = 2UR/\nu\) (\(U = \) velocity). The ranges spanned by these experiments are \(9 < \alpha < 16\) and \(1,800 < Re < 2,300\), where the Reynolds number is based on peak velocity as measured by the hot film probe. Table 1 presents a summary of the various parameters for experiment 102974 in which a hypodermic needle hot film probe was employed. Table 2 shows similar data for experiment 52075 in which we used a catheter-mounted hot film probe. Since the focus of the research was on disordered poststenotic flows and since substantial disorder was encountered for many stenotic conditions, no attempt was made to alter flow conditions by the use of drugs which affect cardiac output and heart rate. Subsequent studies at other Reynolds numbers certainly would be appropriate.

For presentation of data we decided to give representative results from the series of experiments. To include data from all studies would yield an excessively lengthy manuscript; and, for the experiments reported here, a statistical compilation is inappropriate because each dog served as its own "control." All seven experiments produced the same trends and this supports the postulate that the reported observations are not artifacts.

The occurrence of disturbances in the velocity waveforms recorded distal to the imposed stenoses was readily apparent. This is illustrated by the curves in Figure 1 from experiment 102974. For these data the hot film probe was located 2 cm distal to the constriction. Figure 1A shows the measured velocity at the center line of the unoccluded aorta. The shape of the waveform is typical of those found in the descending thoracic aorta in numerous experiments, inas-

### Table 1 Data for Experiment 102974

<table>
<thead>
<tr>
<th>Percent occlusion (reduction in area)</th>
<th>Constriction (cm)</th>
<th>Peak local velocity (cm/sec)</th>
<th>Peak Reynolds number</th>
<th>Mean Reynolds number</th>
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<tbody>
<tr>
<td>0</td>
<td>1.2</td>
<td>59</td>
<td>31</td>
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</tr>
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<td>2100</td>
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<td>41</td>
<td>30</td>
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<td>48</td>
<td>29</td>
<td>2000</td>
</tr>
<tr>
<td>88</td>
<td>0.4</td>
<td>57</td>
<td>27</td>
<td>2400</td>
</tr>
</tbody>
</table>

Frequency parameter: \(\alpha = 13.8 (\omega = 15.0 \text{ rad/sec}, \nu = 0.030 \text{ poise})\). Hot film probe location: 2 cm distal to minimum area of constriction.

### Table 2 Data for Experiment 52075

<table>
<thead>
<tr>
<th>Percent occlusion</th>
<th>Constriction diam. (cm)</th>
<th>Peak local velocity, probe pos. 1 (cm/sec)</th>
<th>Peak Reynolds number</th>
<th>Peak local velocity, probe pos. 2 (cm/sec)</th>
<th>Peak Reynolds number</th>
<th>Peak local velocity, probe pos. 3 (cm/sec)</th>
<th>Peak Reynolds number</th>
</tr>
</thead>
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<td>2500</td>
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<td>2600</td>
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<td>2500</td>
<td>70</td>
<td>3700</td>
<td>52</td>
<td>2800</td>
</tr>
</tbody>
</table>

Frequency parameter: \(\alpha = 15.8 (\omega = 11.7 \text{ rad/sec}, \nu = 0.032 \text{ poise})\). Probe position 1, 1 cm distal to stenosis; probe position 2, 5 cm distal to stenosis; probe position 3, 7 cm distal to stenosis.

**Figure 1** Velocity recorded at the center line of the canine aorta in experiment 102974. The hot film sensor was located 2 cm distal to the area of maximum constriction.
much as the curves are relatively smooth, well defined, and repeatable, although occasional irregularities can be observed near the maximum velocity, during the deceleration phase of systole, and in some cases during diastole. Because of problems in measuring reverse flow discussed earlier, the irregularities observed near zero velocity in the waveforms of Figure 1A could be attributable to artifacts produced by the physical presence of the probe even though the electromagnetic flowmeter indicated that the mean flow was always forward. This point will be considered again in the discussion of the energy spectra.

Figure 1B illustrates the velocity waveform for a relatively slight degree of occlusion (20%). Although the flow could scarcely be described as turbulent, comparison with curve A of that figure clearly indicates the appearance of higher frequency disturbances near the maximum velocity and during the deceleration phase. This is evident also in the energy spectra, as will be shown subsequently. This result was found to be typical of all experiments, provided the measurement was made near the constriction; that is, when the degree of occlusion approached 20%, notable disturbances in the high velocity region of the waveform were detected, particularly during deceleration.

As the degree of stenosis was increased, the flow became more obviously disturbed (Fig. 1C–F). For a 40% reduction in area the flow was markedly disordered during deceleration although the basic pulsatile nature was clearly evident. However, for severe degrees of occlusion the pulsatile aspect diminished so that the distal flow resembled a quasi-steady, turbulent jet.

TIME ENSEMBLE AVERAGE WAVEFORMS
The velocity measurements from experiment 102974 were used to form time-varying mean velocity waveforms using methods described under Data Reduction. These results are given in Figure 2 for 40% and 58% stenosis and can be compared with the corresponding individual waveform results of Figure 1. The average waveforms possess a very striking feature. Although the individual waveforms contain disturbances which appear random, their time averages exhibit distinct double- or multiple-peak patterns. This feature also appeared in all other experiments. The similarity of these records to waveforms we have observed in vitro using flow visualization are suggestive of vortex shedding. Additional evidence for this interpretation is the fact that the multiple-peaked patterns do not appear at all axial stations, but rather disappear, as would occur upon vortex breakup, as the anemometer probe is moved downstream. However, interactions of the flow with the distensible vessel cannot be discounted. These may take the form of resonance of the wall or of wave reflections associated with the changing cross-sectional area presented by the stenoses. Therefore, until further analysis of the waveforms, their energy spectra, and comparisons with data from in vitro studies have been completed, we present these as an observed—and potentially descriptive—but unexplained, phenomenon.

ENERGY SPECTRA
In the present study we performed spectral analysis of the entire velocity waveform for several reasons. First, it is this entity which affects interaction of flow with the vessel wall. Second, the analysis of the total waveform yields information on harmonics of the fundamental heart rate frequency which might be interesting. Furthermore, since frequency band width varies inversely with the sampling time, resolution of the data is sacrificed by resorting to shorter time intervals. Finally, once the stenosis is almost complete, the pulsatile nature of the flow is greatly subdued so that the spectra tend to become independent of position in the cardiac cycle. We shall return to this point shortly.

Examples of energy spectra are illustrated in Figure 3. These correspond directly to the velocity waveforms shown in Figure 1A–F. The plots are in log-log coordinates with the ordinate being $10 \log (u^2/U_{ref})^2$, where $u^2$ is the square of the instantaneous velocity and $U_{ref}$ is a reference velocity which...
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arbitrarily was taken to be 100 cm/sec for the data here. This represents the energy contained within a frequency band width, Δf, which is 1 Hz for the data presented. The abscissa is the frequency coordinate.

Before discussing the experimental results, the general character of the spectral curves should be described. Consider the energy spectrum for the case with no constriction. Were the velocity waveform a perfect sine wave at a frequency of 2 Hz, then the plot of the energy spectral content for our procedure of data analysis would be a delta function 1 Hz in width and centered about a value of 2 Hz on the frequency axis. The height of this delta function would be such that the area beneath the curve would represent the total energy supplied by the velocity pulse. Next, consider a waveform which is periodic, but not a pure sine wave. Also, assume that no random fluctuations occur so that the velocity is completely laminar throughout. If this periodic wave is predominantly (but not exclusively) at one fundamental frequency, then the energy spectrum would have a maximum peak at this fundamental and less prominent peaks at the various harmonics. The height of the spectral curve would decay rather rapidly with increasing frequency. Such appears to be the situation for the velocity waveform (Fig. 1A) and the corresponding energy spectrum (Fig. 3) for the case of no occlusion in the aorta. It would be presumptuous to state that this flow is totally "laminar," since a close inspection of the waveform trace indicates some irregular behavior in each cycle which is not strictly repeatable for every heart beat. However, the velocity for this particular case is "predominantly" laminar, and the associated energy spectrum is composed primarily of frequencies which should be attributed to the decomposition of a curve which is almost periodic, rather than to turbulent velocity fluctuations.

Returning now to discussion of the experimental results, several characteristics of the spectra are apparent. The maximum energy is contained at the frequency corresponding to the fundamental heart rate. Thus, the highest peak in each of the spectral plots occurs at 2 Hz, which is the discrete frequency in the digitized spectra that is nearest the heart rate. Further peaks in the spectra occur near harmonics of this value.

If the data for no occlusion are compared with the remaining curves in Figure 3, it may be seen that, as the degree of occlusion is increased, the energy content in the higher frequencies is elevated. For severe stenoses the sharp, low frequency peaks which are characteristic of pulsatile flow are virtually absent, indicating that the flow immediately distal to the constriction approaches a quasi-steady, turbulent state.

We now consider the problem of uncertainty of results obtained by the hot film anemometer near zero velocity which was mentioned earlier. As pointed out, one cannot be sure that the low velocity segment of the waveform which occurs during diastole is accurately representative of the actual velocity, since the probe cannot distinguish reverse flow. Use of the ECG trigger previously described can eliminate this uncertain segment from consideration and we have performed spectral analysis over that part of the waveform beginning with the onset of systole and terminat-

ing when the velocity fell, following the systolic peak, to a value below approximately 10 cm/sec in the unoccluded case. This same time interval then was maintained for analysis of the cases with occlusion present. When this procedure was applied, it was found that for zero occlusion there is a small contribution of the low velocity region over a wide frequency range. For occlusion of 40%, on the other hand, the spectra with and without exclusion of the low velocity region are scarcely distinguishable from each other. This holds true for all higher degrees of occlusion.

The energy spectra indicate that the degree of disorder in the flow decays as one proceeds distally. Figure 4 illustrates this for one of the experiments (No. 52075), for a stenosis of 50%. The spectrum for no occlusion is shown for reference. From the viewpoint of fluid dynamics the decay is rather rapid. This is likely caused by a combination of mechanisms, among them viscous dissipation of eddies, convection of the disturbance from confinement in a jetlike core to the entire vessel lumen, and absorption of energy by the aortic wall. Further experiments to make measurements at additional radial stations at varying distance from the center line and at several axial locations will be helpful in determining which effect is dominant.

As discussed earlier the consideration of a time history of the energy spectra is of great interest. Although we have generated many such spectra in our data analysis, we shall present here only one particularly interesting example. A later paper will develop this topic in more detail.

If one examines the velocity waveforms for cases of zero to moderate stenoses, it is seen that the acceleration phase of systole has virtually no observable high frequency content, whereas that portion of the curves corresponding to peak velocity and the deceleration phase has higher frequency disturbances present. This is to be expected since an accelerating flow is inherently more stable than a decelerating one. Thus, we have selected for display examples of energy spectra from these two phases of the flow. The specific segments examined are shown in Figure 5 for the
FIGURE 5 Segments of waveform selected for comparison of acceleration and deceleration phases of systole.

“no occlusion” waveform. The acceleration phase is taken to begin prior to the onset of systole, and data are analyzed for 0.1 second. The end of this period occurs slightly prior to the time of peak velocity. The deceleration phase is taken to begin at the termination of the acceleration phase, and data are analyzed for 0.1 second. The same time intervals using the ECG trigger are maintained for each degree of occlusion. Because of the shortened time for analysis, the frequency band width resolution is 10 Hz. Figure 6 gives the computed energy spectra for several degrees of occlusion for experiment 102974. For the case of no occlusion, differences in the spectra for acceleration and deceleration are not large. The corresponding spectra for 20% stenosis also are shown. The disorder during the deceleration phase is dramatically greater than that during acceleration. As the degree of occlusion increases and disturbances begin to occur throughout the waveform, the differences between acceleration and deceleration spectra become less. This is illustrated by the two remaining curves, for 88% occlusion, in Figure 6. It can be seen that the degree of disorder is very similar for both phases. This is, of course, entirely consistent with the results one would anticipate by examining the velocity waveforms themselves. Instabilities begin first in the immediate neighborhood of peak systolic velocity, usually just after the peak occurs. As the degree of stenosis increases, these disturbances extend over a greater portion of the deceleration phase until, finally, virtually the entire waveform is turbulent and quasi-steady.

We have examined the possibility of expressing the energy spectra in terms of similarity parameters. In general, one would not expect to correlate spectral curves for zero occlusion with those for severe stenoses, since the former typically possess little or no disorder while the latter are intensely turbulent. However, once the degree of vessel constriction is sufficient to create substantial flow disturbance, the existence of similarity in the energy spectra might prove useful. A frequency similarity parameter often employed in flow experiments is the Strouhal number, \( S \) (frequency \( \times \) characteristic length/characteristic velocity). Several different combinations for reference length and velocity were employed in an attempt to correlate the energy spectra by a Strouhal number. Although at present we view this problem as incompletely resolved, there does appear to be a definite, but limited, region distal to the stenosis for which the spectral data are reasonably correlated by introducing a semilocal Strouhal number, \( f_d/U_p \), where \( d \) is the constriction diameter and \( U_p \) is the peak value of the velocity measured by the hot film probe at this distal location. This is illustrated by Figure 7, which shows the similarity plots of data taken from experiment 11474 in which the right angle probe was stationed 4 cm distal to the stenosis. The energy axis has been nondimensionalized by \( U_p^2 \) in these curves.

FIGURE 6 Spectra for acceleration and deceleration phases of systole. Data derived from waveforms of experiment 102974.

FIGURE 7 Energy spectra using similarity coordinates. Data are for velocity waveforms of experiment 11474.
The spectrum for zero occlusion is distinctly separated from spectra for the occlusion measurements, as would be expected. The remaining spectral curves all lie quite close together, indicating good similarity. This result also was found in other experiments. We emphasize, however, that the suggested parameters appear valid only within a limited distal region whose boundaries are as yet ill-defined. If the measurement site is quite close to the constriction, the spectral curves do not show good correlation, possibly because the large scale eddies created have not had sufficient time to decompose before intercepting the probe.

Discussion

The studies reported here have examined poststenotic flow fields in the canine descending aorta with emphasis on describing the disordered flow characteristics which occur. We have demonstrated that clearly discernible alterations in the velocity waveform and corresponding energy spectra are found for mild degrees of stenosis and that very turbulent fields, which are quasi-steady in nature, are encountered for severe stenoses. Spectral analysis of the entire velocity waveform suggest the existence of a modified Strouhal number correlation over the near-field region distal to axisymmetric obstructions. Measurements over selected parts of the cardiac cycle indicate that the acceleration phase of systole is the most stable region of the waveform and is the last segment to encounter substantial disturbances as the degree of stenosis is increased. On the other hand, the deceleration phase immediately subsequent to the peak systolic velocity is the most unstable segment, and it is here that instabilities are first observed. The axial decay of disturbances also has been described and found to be rather rapid. This would indicate that disorder produced by a mild stenosis, by vessel branching, or by bifurcation would likely be dissipated quickly as the flow proceeds distally. Presumably, this would likewise be true for disturbances created by actual or artificial cardiac valves.

Although the present study has not examined directly the questions of intimal injury, poststenotic dilation, or atherogenesis, the measurements of the fluid environment distal to vascular stenoses can be hypothesized as having some relationship to these problems. Fry has indicated that disordered flow patterns may have an effect on endothelial cell orientation and possibly on the transport of lipoproteins into the vessel wall. Our studies in vitro of pulsatile flow through modeled stenoses indicate that the region immediately distal to the constriction is filled with a violently swirling flow. If the observations reported by Fry eventually are proved to be related to atherosclerosis, it may be possible that the flow field created by an early lesion could contribute to local enhancement of the disease. Roach has reported that the frequencies of vibration which lead to poststenotic dilation in arteries range from 25 to 400 Hz. The turbulent energy spectra described in our study indicate substantial energy content throughout this range, provided the stenosis is moderately large.

The importance of early detection of occlusive vascular disease leads us to speculate optimistically that the velocity waveforms and corresponding energy spectra may provide valuable clues to the extent of disease development before clinical symptoms become apparent. Any eventual application of fluid dynamic measurements as a diagnostic tool for detecting occlusive vascular disease in its initial stages of development would have to rely on noninvasive techniques. Clearly, the hot film anemometer would not be suitable. However, as refinements are made in Doppler ultrasound devices, eventually it may be possible to detect and quantify disorders of blood flow in a noninvasive,atraumatic manner.

References

Measurements of disordered flows distal to subtotal vascular stenoses in the thoracic aortas of dogs.

D P Giddens, R F Mabon and R A Cassanova

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