Pressure-Flow Relations In Dog Arteries

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ABSTRACT

The propagation of the arterial pressure and flow pulses through the vascular beds is characterized by marked changes in shape and magnitude. In order to understand better the mechanisms which account for these changes, instantaneous pressures and flows were measured simultaneously at various sites in the arterial tree of 12 anesthetized dogs, using Statham strain gauges and electromagnetic flowmeters. The data were processed by on-line Fourier analysis and the frequency spectra of pressure, flow and vascular impedance at these sites were evaluated. In these dogs about 10% of the cardiac output was distributed to the head, 14% to the kidney, 35% to the gastrointestinal tract, 14% to the pelvic organs and 6% to the legs. The input impedances of the various beds were frequency dependent, the frequency-dependent components varying between 2 and 45% of the corresponding D-C impedances (peripheral vascular resistance). The viscous losses associated with pulsatile flow were larger than those for corresponding steady flows. The excess losses varied between 9 and 30% of the mean flow losses. The marked decrease in cross section down the aorta lead to considerable acceleration of the mean blood velocity and corresponding pressure losses despite the drainage through aortic branches. Comparisons of measured flows with those predicted from Womersley’s theory showed satisfactory agreement for short vascular segments. However the theory underestimates the losses occurring in the nonuniform arterial tree. On the basis of anatomic measurements, the space dependence of resistance, iner- tance and vascular distensibility was evaluated.

ADDITIONAL KEY WORDS vascular geometry elastic tapering frequency spectra of arterial pressure and flow vascular impedance on-line Fourier analysis distribution of mean flow arterial models energy losses in blood flow anesthetized dogs

Theoretically it is possible to calculate dynamic blood flows from instantaneous pressure measurements if the physical properties of the blood and blood vessel walls are known. Hamilton and Remington were among the first to make systematic use of such a possibility for the determination of cardiac output. Their approach is based on Frank’s Windkessel theory and requires only the measurement of a pressure pulse at one site. More refined theories predicting flow in terms of the pressure gradient have recently been advanced by Womersley and by others (see reference 4 for review). However, all theories depend upon simplifying assumptions relating to geometry of the blood vessels, visco-elastic properties of the vessel walls and rheology of the blood itself.

The primary purpose of the present study was to investigate experimentally the relationship between dynamic blood flow and blood pressure at various points of the arterial tree of anesthetized dogs and to compare the results with those predicted by Womersley’s theory. This work has been made possible by technical advances in the measurement of instantaneous blood flow and by the development of automatic computing devices which provide rapid and continuous solutions for the complex Fourier analysis of pressure and flow recordings. Our results...
show good agreement with Womersley’s theory, provided the theory is applied to short uniform vascular segments. However, the theory appears to be inadequate for the prediction of flow from the pressure gradient in more complex vascular beds.

Methods

The experiments were done on 12 anesthetized dogs, weighing between 12.5 and 30 kg. After anesthesia with pentobarbital sodium (30 mg/kg) and intubation of the trachea, the carotid and femoral arteries were exposed, and catheters and flow probes were introduced at the desired locations. After a series of measurements had been obtained the animal was connected to a Harvard respirator and the chest was opened. In order to secure adequate space for manipulation, one or two ribs were removed. Flow probes and catheters were then placed at different points on the thoracic aorta and another series of measurements was made. Finally, the abdomen was opened and additional catheters and flow probes were introduced at one or two sites in the abdominal aorta and the superior mesenteric artery. (In some animals the measurements in the abdominal arteries were made before the chest was opened.) In this final stage of the experiment as many pressure and flow measurements as possible were recorded simultaneously. The aortic mean pressure was monitored continuously throughout the experiment and slow continuous infusion of dextran was given into the femoral vein in order to minimize the effects of blood losses. The rate of infusion was adjusted to keep the mean pressure constant. Flow was measured in the ascending aorta in eight dogs, in the abdominal aorta in five, in the superior mesenteric artery in four, in the carotid in seven and in the femoral artery in eight. Except for one experiment, at least four simultaneous pairs of pressure and flow recordings were obtained in each animal.

1. PRESSURE MEASUREMENT

Pressures were measured by means of Statham strain gauges, either of the SF-1 or the P23 series. The catheter manometers (SF-1) which have a natural frequency of about 2000 cycles/sec were used only in the aorta and were introduced either through a femoral or a subclavian artery. They were capped with plastic plugs containing two side holes, so that lateral pressure was measured. The P23 gauges were connected to the vessel by means of nylon catheters, 5 cm long. The latter were introduced through side branches at the point of measurement and also sensed side pressure. The natural frequency of the catheter-manometer system was at least 80 cycles/sec depending upon the size of the catheter and the type of gauge used. All catheter-manometer systems were calibrated statically against a water manometer and also dynamically by the Hansen “pop” technique.

2. FLOW MEASUREMENT

Volume flow was measured by means of a four-channel electromagnetic flowmeter (Medicon) using either type K or type Q probes. Care was taken to provide a snug fit between the probe and the vessel wall. The static flowmeter calibration was done as follows: at the end of the experiment Bardic catheters of appropriate size were tied into the vessel upstream and downstream to the flow probe. The downstream catheter was connected to a reservoir, which provided the distending pressure observed during the experiment, and the upstream catheter to a double-barrel, variable speed Harvard injection pump, which delivered flow rates from 0.027 to 720 cm³/min. Higher flow rates (1 to 3 liters/min) were obtained by replacing the pump with a pressurized reservoir. After all side branches had been tied it was possible to calibrate several flow probes on the same vessel simultaneously. Dynamic calibration was performed in some of the experiments by using a servo-controlled pump with variable frequency and a sinusoidal output. The connections for dynamic calibration were identical with those for the static calibration, and the difference between the two calibrations did not exceed 10% from zero to 25 cycles/sec. The ratio of the two calibrations was 0.98 ± 0.03 (mean and SEM), over this frequency band and the phase lag was linear, amounting to 0 = (2 f ± 0.83)° (mean and SEM), where f is the frequency in cycles/sec. The dog’s own blood was used as a perfusion liquid during the calibration procedure.

3. RECORDING AND ANALYSIS OF DATA

The transducer outputs were fed through Sanborn amplifiers into a switch panel and from there in parallel into two eight-channel Sanborn monitoring scopes, a 14-channel tape recorder (Ampex CP-100) and the analog to digital (A-D) converter of the LINC computer. The LINC is a small digital computer with a core memory of 2048 words and a 12 bit word length. Additional storage is provided by two digital tape units each of which can handle 512 blocks of 512 words. The A-D converter has seven channels with a maximal conversion speed of 25,000 samples/sec and accepts an analog voltage from −1.0 to +1.0 volts with a resolution of 1:256, i.e., 7.8 mv. Zero suppression on the amplifiers was used to eliminate the D-C components so that the oscillatory components could be ampli-
fied to the maximum compatible with the converter range. The digital signals were monitored on the oscilloscope of the LINC in order to assure appropriate amplification without clipping. The mean values of pressure and flow were obtained from the recordings of a Sanborn oscillograph. All the variables were calibrated in terms of cm H₂O or cm³/sec.

During the experiment selected cycles were marked on an additional channel of the tape recorder. Simultaneously the computer determined the period of the marked cycle, using a one-volt level of the amplified R wave of the ECG as a triggering signal. The tape was then played back, the marked cycles were sampled (usually at 96 samples/cycle) and the data subjected to Fourier analysis. The Fourier coefficients obtained were then converted to appropriate units, the modulus and the phase were calculated for the desired number of harmonics and stored on digital tape. A read-out of the coefficients on the oscilloscope permitted evaluation of these preliminary results within minutes after an experimental run had been performed. The validity of the Fourier analysis technique as applied to the cardiovascular system has been discussed in detail elsewhere. An advantage of on-line Fourier analysis is that spurious harmonic contents may be determined before artifacts, such as damping, become apparent in the analog tracing. Steps can then be taken to remove the cause of these artifacts during the experiment, rather than to process a large amount of unreliable data. From the pressure and flow data the input impedances into the various beds were calculated and printed out by means of a Teletype. For each part of one experiment eight to ten cardiac cycles were analyzed and the values for pressure, flow, and impedance averaged and printed out with their standard deviation in terms of modulus and phase.

The results reported are based on the analysis of 580 cardiac cycles.

The static measurement errors evaluated at the computer output are ±0.2 cm H₂O for the pressure transducers and ±0.1 cm³/sec for the flowmeters. The cardiac frequencies encountered in these studies lie between 1 and 3 cycles/sec. Since only the components of the pressure and flow pulses below the sixth harmonic are of significant magnitude (table 4 and fig. 2), the frequency spectrum of these variables is accurately reproduced by a measuring system with a flat frequency response from 0 to at least 20 cycles/sec. (This is also a necessary requirement for the measurement of mean flow and mean pressure.) The static measurement accuracy can be maintained over this frequency range by careful coupling of the transducers to the cardiovascular system. Such coupling requires short, rigid and bubble-free liquid connections between blood and pressure transducer, as well as a snug and invariant fit between vessel and flow probe.

A 10% decrease in vessel radius during an experiment, for instance, during periods of hypotension, may alter the calibration of the flowmeter by a factor of two or more. Referring again to figure 2 and table 4, it will be noted that the magnitudes of the higher harmonics are frequently within the range of measurement errors. The error in the calculation of the impedance functions of the blood (equation 3, below) or of the vessel walls at these frequencies therefore becomes large. Additional errors are introduced in the determination of the phase of the impedance if the pressure is not measured at the center of the flow probe. Assuming a wave velocity of 6 m/sec and a heart rate of 2 cycles/sec a distance of 5 cm between the sites of pressure and flow measurements would introduce an error of 6° in the determination of the phase of the

### Table 1

Average values for mean flow and D-C impedance (peripheral resistance) obtained in 12 anesthetized dogs (weight 22 ± 1.5 kg, heart rate 2.64 ± 0.03 cycles/sec, mean arterial pressure 140.7 ± 4.2 cm H₂O).

<table>
<thead>
<tr>
<th>Artery</th>
<th>Number of runs*</th>
<th>Mean flow</th>
<th>D-C Impedance</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>cm³/sec</td>
<td>10⁶ × dynes cm⁻¹sec</td>
<td></td>
</tr>
<tr>
<td>Ascending aorta</td>
<td>20</td>
<td>28.7 ± 1.2†</td>
<td>4.7 ± 0.3</td>
</tr>
<tr>
<td>Descending aorta</td>
<td>25</td>
<td>21.0 ± 1.0</td>
<td>6.9 ± 0.5</td>
</tr>
<tr>
<td>Abdominal aorta, high</td>
<td>4</td>
<td>10.6 ± 0.6</td>
<td>12.5 ± 0.7</td>
</tr>
<tr>
<td>Abdominal aorta, low</td>
<td>2</td>
<td>6.3 ± 0.3</td>
<td>21.0 ± 1.5</td>
</tr>
<tr>
<td>Superior mesenteric artery</td>
<td>23</td>
<td>2.7 ± 0.3</td>
<td>62.0 ± 12.8</td>
</tr>
<tr>
<td>Carotid artery</td>
<td>11</td>
<td>2.6 ± 0.2</td>
<td>60.5 ± 0.8</td>
</tr>
<tr>
<td>Femoral artery</td>
<td>14</td>
<td>1.0 ± 0.1</td>
<td>150.0 ± 14.2</td>
</tr>
</tbody>
</table>

*8 to 20 cycles each.
†Mean and se of mean.
The average values for heart rate, mean arterial pressure, and mean flow observed in all arterial beds of 12 anesthetized dogs are listed in table 1. Opening of the chest was followed in 35 cm H2O of mean blood pressure of 25 cm H2O (range: -70 to +15 cm H2O) by an average fall in mean blood pressure of

| TABLE 2 |

<table>
<thead>
<tr>
<th>Segment</th>
<th>A*</th>
<th>R</th>
<th>L</th>
<th>C1</th>
<th>C2</th>
<th>Q0</th>
<th>V0</th>
<th>ΔP0</th>
<th>Z0</th>
<th>Z0/Z0</th>
</tr>
</thead>
<tbody>
<tr>
<td>no.</td>
<td>cm²</td>
<td>dyn cm⁻³</td>
<td>cm²</td>
<td>dyn cm⁻³</td>
<td>cm²</td>
<td>dyn cm⁻³</td>
<td>cm²</td>
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</tr>
<tr>
<td>1</td>
<td>1.57</td>
<td>1.6</td>
<td>3.2</td>
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<td>29.3</td>
<td>28.7</td>
<td>18.4</td>
<td>43</td>
<td>4.7</td>
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</tr>
<tr>
<td>2</td>
<td>1.13</td>
<td>2.2</td>
<td>4.4</td>
<td>16.7</td>
<td>24.2</td>
<td>24.8</td>
<td>21.8</td>
<td>54</td>
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<td></td>
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<tr>
<td>3</td>
<td>.71</td>
<td>2.8</td>
<td>7.2</td>
<td>9.0</td>
<td>12.5</td>
<td>21.0</td>
<td>30.5</td>
<td>157</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>.50</td>
<td>15.0</td>
<td>10.0</td>
<td>.64</td>
<td>8.5</td>
<td>21.0</td>
<td>35.1</td>
<td>224</td>
<td>6.9</td>
<td>.10-18</td>
</tr>
<tr>
<td>5</td>
<td>.50</td>
<td>15.0</td>
<td>10.0</td>
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<td>8.5</td>
<td>21.0</td>
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<td>224</td>
<td>6.9</td>
<td>.10-18</td>
</tr>
<tr>
<td>6</td>
<td>.376</td>
<td>28.8</td>
<td>13.3</td>
<td>4.8</td>
<td>3.5</td>
<td>13.5</td>
<td>36.0</td>
<td>300</td>
<td>12.5</td>
<td>.14-21</td>
</tr>
<tr>
<td>7</td>
<td>.300</td>
<td>40.0</td>
<td>16.4</td>
<td>3.9</td>
<td>2.8</td>
<td>6.0</td>
<td>19.5</td>
<td>242</td>
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<td></td>
</tr>
<tr>
<td>8</td>
<td>.298</td>
<td>42.4</td>
<td>16.9</td>
<td>3.8</td>
<td>3.8</td>
<td>6.0</td>
<td>20.0</td>
<td>254</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*A = cross section, R = resistance, L = inductance, C = capacitance, Q0 = mean flow, V0 = average velocity, ΔP0 = pressure difference associated with mean flow, Z0 = D-C impedance, Z0/Z0 = pulsatile impedance/D-C impedance. Measured data based on 12 dogs; C1 assumes a constant modulus of elasticity E = 5.8 × 10⁸ dyn cm⁻², C2 assumes elastic tapering E = 4 to 9 × 10⁸ dyn cm⁻².
backflow decreases progressively and there appears a second period of forward flow, which is considerably smaller than the one associated with the cardiac ejection period. The flat portion of the flow curve immediately preceding the systolic ejection represents zero flow only in the ascending aorta; toward the periphery, this level increases toward a positive value.

The harmonic content of the pressure and flow curves is illustrated for different arteries in figure 2. The data for this figure were obtained from three dogs with identical heart rates and comparable flows in the ascending and descending aorta and in the carotid artery. The mean pressures in the three experiments were 90 cm H₂O, 120 to 130 cm H₂O and 160 cm H₂O. (Mean pressure, P₀, and mean flow, Q₀, for each tracing are indicated in the figure.) The magnitudes of all the harmonics are expressed as fractions of the corresponding mean value. While the magnitude of the lower harmonics of the flow pulse is similar to that of the mean flow, the corresponding values of the oscillatory pressure components represent only a small fraction of the mean (usually less than 20%). It is therefore apparent that the frequency-dependent components of the impedance (pressure divided by flow) must be considerably smaller than the D-C impedance.

The magnitude of the harmonics of the pressure pulse increases toward the periphery. This increase is more marked toward the hind part as compared to the front part of the body (compare mesenteric and femoral vs. carotid). The magnitude of the first harmonic is usually considerably higher than that of the higher frequency components. Frequently, only the second harmonic is of the same order of magnitude, and above the sixth harmonic, the frequency content tends to disappear within the noise level of the measuring equipment. Note that the magnitudes of the harmonics at two measuring sites in the same vessel are different (distance between proximal (car. 1) and distal (car. 2) pressure measuring site in the carotid was 19.5 cm; between proximal (desc. 1) and distal (desc. 2) thoracic aorta, 14.5 cm; and between upper (abd. 1) and lower abdominal (abd. 2) aorta, 6 cm). In the abdominal aorta and the carotid artery, the magnitudes of the harmonics decrease from the proximal to the distal measuring site. In the descending aorta, on the other hand, all frequency components except the first harmonic increase in size between the proximal and distal measuring site. These observations are in agreement with earlier reports in which the overall increase in the magnitude of the pressure harmonics is characterized by an undulatory pattern, consisting of peaks and troughs alternating as a function of space and frequency.¹⁰

Turning now to the frequency spectrum of the flow pulse, it will be noted that with the exception of the abdominal arteries the magnitudes of the first two harmonics are of the
same order as the mean flow. Frequently the second harmonic is larger than the first, particularly in the femoral and carotid arteries. Despite the relatively large pulsatile pressure components in the abdominal arteries, the magnitudes of the flow harmonics decrease rapidly with frequency; the third harmonic is already very small in these vessels. While the frequency spectra of flow at the two abdominal measuring sites are similar, there are considerable differences in the harmonic content of the flow pulses in the descending aorta and carotid artery. The more distal flows in both vessels are less pulsatile than the proximal flows. In general, the magnitudes of all flow harmonics decrease along the aorta, reaching a minimum in the abdomen and increasing again toward the femoral artery. However, the peaks and troughs which characterize the propagation of the pressure pulse appear less conspicuous in the propagation of the flow pulse.

For comparison, the frequency spectra for the blood flow in the inferior and superior
Frequency spectrum of blood flow in the ascending aorta, the inferior and superior vena cavae, normalized with respect to mean flow. The ratio of superior vena cava flow to inferior vena cava flow is 1:3.3 in this anesthetized dog (heart rate 2.1 cycles/sec). Calculated from data of Pinkerson et al. 11

venae cavae calculated from data of Pinkerson et al. 11 are shown in figure 3. Note that in the superior vena cava the first two pulsatile components amount to about 40 to 60% of the D-C component, while in the inferior vena cava the magnitude of the highest harmonic is only about 15% of the D-C value.

Impedance is generally analyzed in terms of sinusoidal variables and the computation of impedances in the vascular system requires the transformation of the observed periodic variables into equivalent sinusoidal frequency distributions. This is accomplished by Fourier analysis. 8-10 Thus the observed blood pressure $P(t)$ can be represented by the Fourier series:

$$P(t) = \sum_{n=0}^{N} P_n \cos(n \omega t - \theta_n) \quad (1a)$$

or by the real part of the equivalent exponential form:

$$P(t) = \sum_{n=0}^{N} P_n e^{i(n \omega t - \phi_n)} \quad (1b)$$

Similarly, for the observed blood flow at the same point in the vessel we can write:

$$Q(t) = \sum_{n=0}^{N} Q_n e^{i(n \omega t - \phi_n)} \quad (2)$$

where: $\omega$ is the angular frequency
$n$ is the harmonic under consideration
$t$ is time
$j$ is $\sqrt{-1}$
and $\theta$ and $\phi$ are phase angles referred to the beginning of the period over which the signal is analyzed.

The vascular input impedance, $Z(\omega)$, (pressure divided by flow) is frequency dependent and has to be calculated separately for each harmonic:

$$Z_n = \frac{P_n}{Q_n} e^{i(\theta_n - \phi_n)}; \quad n = 0,1,2,\ldots \quad (3)$$

For $n = 0$, equation 3 represents the ratio between mean pressure and mean flow, commonly called vascular resistance. For other values of $n$, equation 3 describes the pressure-flow relation for the different harmonics. In contrast to the D-C impedance, these impedances are characterized by a magnitude $(P_n/Q_n)$ and an angle $(\theta_n - \theta_0)$. This means that their values depend not only on the viscous properties of the blood and the radius of the vessel (as the peripheral resistance), but also on the distensibility of the vessel wall and the inertia of the fluid. Referring to the model discussed in the last section of this paper, one can associate the real part of the input impedance (magnitude times the cosine of the phase angle) with the energy dissipated by the blood stream, and the imaginary part (magnitude times the sine of the phase angle) with the energy exchanged back and forth between blood and vascular structures.

The average values of the input impedances of the various beds, calculated for comparable frequencies (±0.2 cycle/sec), are illustrated in figure 4 (mean and sem). From a maximum at zero frequency the impedance de-
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creases rapidly in an oscillatory fashion to a fraction of the D-C value. The locations of the minima and maxima depend both on frequency and on the site of measurement. In the ascending aorta this oscillation is smallest, ranging from a minimum of 4% of the D-C value for the third harmonic (about 8 cycles/sec) to a maximum of 9% for the sixth harmonic. The phase angles are negative below 8 cycles/sec (flow is leading pressure) and become positive at higher frequencies. In the descending aorta the behavior of the impedance is more oscillatory, varying between 11 and 18% of the D-C value. A first maximum appears for the third harmonic, and minima for the first and fourth harmonic. Except for the second and fifth harmonics, the phase angles are negative. In the abdominal aorta the impedance decreases steadily until the third harmonic and increases thereafter, oscillating between 14 and 22% of the D-C value. The phase angles become positive above the second harmonic. In the mesenteric artery the input impedance represents a considerably larger fraction of the D-C impedance than in the other vascular beds (38 to 44%) and the phase angles are negative at all frequencies. As shown in figure 2, the magnitude of the flow components in the mesenteric artery decreases much faster with frequency than that of the corresponding pressure components, indicating that the effects of vascular distensibility (Windkessel effect) are much larger than in the other beds studied. In the femoral artery the impedance decreases steadily with frequency (12 to 4% of the D-C value) and the phase angles become positive above the third harmonic. In the carotid artery the values range between 10 and 24% of the D-C value, with a minimum for the fourth harmonic. The phase angle behavior is similar to that in the femoral artery. At the origin of the carotid artery, the impedance behavior appears more oscillatory than in the more distal part over the frequency range observed. The presence of these maxima and minima...
clearly indicates the presence of wave reflections in the arterial system. However, the magnitudes of these oscillations are much smaller than those one would expect if the reflections were originating at a few specific sites. It has been shown that the effects of wave reflection in the arterial tree are markedly attenuated by other factors such as geometric and elastic tapering of individual arteries as well as the overall geometry of the vascular bed. In fact, the transmission characteristics of the arterial tree appear to decouple the heart from its peripheral load so that external cardiac work becomes remarkably independent of heart rate.

On the basis of the flow and impedance data the energy dissipation due to the pulsatile components has been calculated following the procedure outlined by Uchida. It amounts to about 10% of the dissipation due to mean flow in the ascending aorta, 30% in the descending aorta, 16% in the abdominal aorta, 13% in the mesenteric artery, 24% in the carotid artery and 30% in the femoral artery. In terms of the power output of the heart and its energy requirements, such losses are not negligible. Only about two-thirds of the dissipation due to mean flow is associated with the blood flow through the arterial bed and about one-third with the capillary and venous beds. In contrast, nearly all the losses introduced by the pulsatile components occur in the arteries.

**Discussion**

**a) DISTRIBUTION OF MEAN FLOW**

The amount of a metabolite transported through the larger vascular channels is proportional to its concentration and to mean flow. As far as this function of the cardiovascular system is concerned, the pulsatile components can affect it only by providing a more uniform mixing. Taking into account that the coronary circulation absorbs about 5% of the cardiac output, our data indicate that in supine anesthetized dogs, approximately 10% of the total output is distributed to the head, 14% to the kidney, 35% to the gastrointestinal tract, 14% to the pelvic organs and 6% to the legs. Correspondingly, the peripheral resistance into a femoral artery is about 35 times greater than the total resistance of the peripheral circulation. Figures derived from the *Handbook of Physiology* indicate that the "average" cardiac output is distributed as follows: coronary circulation 10%, brain 15%, kidney 25%, gastrointestinal tract 25%, muscle 15% and skin 10%. Using a dye dilution technique, Evans et al. found in anesthetized dogs that about equal amounts of the ascending aortic flow go to the head and forelimbs (1/3), the portal and hepatic systems (1/3) and the kidneys and hindlimbs (1/3), the flow to the kidneys amounting to about 2/9 of the total flow. Although the fraction of the cardiac output going to a given vascular bed is subject to considerable changes, depending upon the vasoactive state, the three sets of values are in good agreement.

**b) RELATION BETWEEN PULSATILE PRESSURE AND FLOW**

Using Womersley's theory, the flow through an arterial segment can be calculated if the pressure gradient is known. For a sinusoidal pressure gradient, \( \Delta P = M \cos(\omega t - \theta) \), the solution for volume flow \( Q \) is:

\[
Q = \frac{\pi r^4 M'}{\mu \alpha^2} M \sin(\omega t - \theta + \varepsilon)
\]

where

\[
\alpha^2 = \frac{r^4 \omega}{\nu}
\]

\( r \) = inside vessel radius
\( \mu \) = viscosity of the blood
\( \omega \) = angular frequency
\( \nu \) = kinematic viscosity
\( M' \) and \( \varepsilon \) are functions of \( \alpha \) and have been tabulated by Womersley.

When \( \alpha \) goes to zero (steady flow), \( M'/\alpha^2 \rightarrow 0 \) and \( \varepsilon \rightarrow 0 \), i.e., the equation reduces to that obtained by Poiseuille. The behavior of pulsatile flow is determined primarily by the nondimensional parameter \( \alpha \).

Figure 5 shows a comparison of measured flows with those predicted by Womersley's theory. The data are based on a pressure gradient measured over a distance of 5 cm and values of the inside radius estimated from measurements of the external diameter and wall thickness in the descending aorta. The external diameter was measured in vivo by

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Comparison of calculated and measured flows in a short segment of the descending aorta. Ratio of the two values is plotted on the ordinate. For calculations, two different radii and three different viscosities have been assumed \( r_0 \) = outside radius, \( h \) = wall thickness, \( \mu \) = coefficient of viscosity, \( \Delta L \) = length over which the pressure difference was measured.

The ratio, measured/calculated flow, is plotted on the ordinate. Assuming a ratio wall thickness/outside radius \( h/r_0 \) of 0.22, the top three curves are obtained. The differences between these three curves are due to the values of viscosity assumed (0.01 to 0.03 poise). The lowermost curve shows the result when the measured value of wall thickness is introduced into the calculation. It will be seen that the agreement between experimental and theoretical values depends markedly on the value chosen for the inside radius of the vessel.

In similar comparisons done for data obtained in the femoral artery and carotid artery, it has always been possible to match experimental and predicted data both with respect to magnitude and phase, provided an appropriate value for the inside vessel radius is substituted into equation 4.20 Errors in the estimate of viscosity are less critical because of the way \( \mu \) and \( \alpha \) appear in equation 4, and will be most noticeable at low values of \( \alpha \). Both in Poiseuille's and in Womersley's equation the radius appears in the fourth power and a small error in its estimate produces a correspondingly large error in the predicted result. In contrast to the outside dimensions of the vasculature, which can be measured accurately to at least \( \pm 0.02 \) cm in vivo, there exists at present no reliable method for the direct measurement of the inside diameter. It is generally stated that the ratio of wall thickness to outside radius is approximately constant in the larger arteries and the values given range between 0.12 and 0.16.10 Hence the inside radius derived from such an estimate may be in error by 25% or more.20 Furthermore, the wall thickness decreases as the distending pressure increases,21 and the measured value depends markedly upon the method used for the preparation of the specimen.22 Until better methods for radius measurements become available, Womersley's theory appears to be adequate for the prediction of pulsatile flow from the pressure gradient in a short vascular segment without branches, where uniform wall properties are a reasonable assumption. For reasons discussed in the next section, the pressure gradient has to be measured over a short distance. Under these conditions the pressure difference associated with the mean flow is extremely small and close to the limits of accuracy of modern manometers (table 2).

The frequency dependence of the input impedance has been investigated repeatedly for long, rigid and elastic tubes and found to be in good agreement with the results predicted from hydrodynamic theory.4 Corresponding data for the vasculature of the living animal are scarce, primarily because of the problems associated with the techniques of measurement and data analysis.25-29 The only comparable data obtained in a series of experiments are those of Patel et al.24, 25, 26 and Bergel et al.28 Our results concerning the behavior of the input impedance of the ascending aorta in the dog are practically identical with those in Patel's study. In man, where the cardiac output is about four times larger than in the dog, the D-C impedance is correspondingly lower, but the ratio between the frequency-dependent components \( Z_r \) to the
D-C value \((Z_0)\) is of the same order. The D-C impedance into the femoral bed of man is about one-third of that found in this study, and the ratio of the frequency-dependent components to the D-C value is considerably higher. However, Patel also showed that this ratio depends markedly upon the state of the vasculature. During reactive hyperemia, the D-C value fell to 75% of the control value, and the change in the ratio \(Z_p/Z_0\) was small. After the administration of norepinephrine the D-C value rose to 230% of the control value, while the ratio \(Z_p/Z_0\) fell to between 0.07 and 0.12. Furthermore, the marked increase in the magnitude of the components of the pressure toward the hindpart of the body was observed only in the region of the inguinal ligament. Thereafter, the magnitudes appeared to decrease progressively toward the foot, indicating a predominance of viscous losses. Under such conditions the magnitude of \(Z_p/Z_0\) depends strongly on the site of the pressure measurement. It is noteworthy that while the D-C value is only about one-seventh, the ratio \(Z_p/Z_0\) is considerably larger in the pulmonary vasculature as compared to the peripheral vascular bed.

Fry et al. measured the energy losses associated with blood flow in the descending aorta of five dogs and compared the results with those predicted by Womersley's theory. Their data indicate that the theory underestimates the viscous losses associated with pulsatile flow by a factor of three or more. Uchida calculated the energy losses associated with pulsatile flow in a circular, rigid pipe and compared them with those encountered in steady flow. He predicted that more work was required to provide a given flow rate by means of pulsatile than by steady flow. He suggested that this phenomenon might be related to the origins of turbulence.

In our calculations only the first and second harmonic of the flow pulse contribute significantly to the energy losses. For the corresponding range of \(\alpha (4\) to 22) the predicted extra losses are less than 10% of the losses due to mean flow. The observed values are considerably larger, ranging from 30% in the descending to 9% in the ascending aorta, and are of the same order of magnitude as those reported by Fry. Since this discrepancy is unlikely to be caused by nonlaminar flow patterns, one has to examine the second possibility he proposed, namely, the effects of the decrease in cross section of the individual arteries with distance (geometrical tapering) upon the pressure-flow relations.

c) ARTERIAL GEOMETRY AND BLOOD FLOW

From the aortic valves to the bifurcation both the cross section of the aorta and the mean flow decrease by a factor of 5, while the elastic modulus of the aortic wall increases fourfold (table 2). The changes in cross section are most marked at branch points where volume flow is diverted into several channels. In order to estimate the effects which might be expected from such nonuniformities we have measured the dimensions of the aorta at 14 sites and that of the proximal sections of the brachiocephalic and iliac-femoral arteries at six sites each. The values for the external radii were obtained from in vivo measurements in a 19-kg dog and, at corresponding points, are consistently 25% lower than those reported by Patel for the average aortic tree of 11 dogs with a mean weight of 22 kg. The thoracic aorta (length 25 cm) was divided into five, the abdominal aorta (length 15 cm) into three, and the brachiocephalic-carotid and the iliac-femoral main flow channels into seven segments each. The radii at both ends of each five centimeter segment were averaged in order to obtain the equivalent cross section of the segment. For the calculation of the inside radius the following values were assumed for the ratio wall thick-
ness/outside radius: 0.12 for the aorta, 0.15 for the carotid and 0.2 for the femoral artery. Neglecting longitudinal strains and defining:

\[ R = \frac{P}{\dot{Q}} = \frac{8\mu l}{\pi r^4} \]  

\[ C = \frac{dV}{dt} = \frac{3\pi r^2 (a + 1)^2 l}{E(2a + 1)} \]

\[ L = \frac{P}{dQ/dt} = \frac{\rho l}{\pi r^2} \]

where:  
- \( R \) = resistance of a vascular segment  
- \( C \) = volume distensibility of a vascular segment  
- \( L \) = inertance of the fluid in a vascular segment  
- \( P \) = pressure  
- \( \dot{Q} \) = volume flow  
- \( V \) = volume of vascular segment  
- \( r \) = inside radius of segment  
- \( l \) = length of segment  
- \( a \) = ratio or radius/wall thickness  
- \( \mu \) = viscosity of fluid  
- \( \rho \) = density of fluid  
- \( E \) = elastic modulus of wall material

one finds a progressive increase in resistance and inertance and a decrease in distensibility along the arterial tree, as well as marked changes in linear velocity. The values for distensibility were calculated for two cases:  

a) assuming a constant modulus of elasticity:  
- \( E = 5.8 \times 10^6 \) dyn cm\(^{-2}\) for the aorta,  
- \( E = 7.1 \times 10^6 \) dyn cm\(^{-2}\) for the brachiocephalic-carotid channel and  
- \( E = 13.0 \times 10^6 \) dyn cm\(^{-2}\) for the iliac-femoral channel.

b) assuming a progressive increase in the elastic modulus:  
- \( E = 4 \times 10^6 \) dyn cm\(^{-2}\) at the origin and  
  \( 9 \times 10^6 \) dyn cm\(^{-2}\) at the end of the aorta,  
- \( E = 5 \times 10^6 \) dyn cm\(^{-2}\) at the origin of the brachiocephalic artery and  
  \( 9 \times 10^6 \) at the end of the carotid artery,  
- \( E = 9.2 \times 10^6 \) dyn cm\(^{-2}\) at the origin of the iliac artery and  
  \( 20 \times 10^6 \) dyn cm\(^{-2}\) in the saphenous artery.

Considering first the aorta (table 2), it will be noted that the average linear velocity increases from 18 cm/sec at the root to 42 cm/sec at the diaphragm and then decreases again to 20 cm/sec at the aortic bifurcation (fig. 6). In the thoracic aorta the drainage of volume flow by the branch arteries is not sufficient to compensate for the decrease in cross-sectional area and a marked acceleration of the mean flow associated with additional dissipation of energy occurs. Note that the extra losses observed in our experiments are largest in this section of the aorta.

**FIGURE 6**

Variation in cross section, flow, and average blood velocity along the aorta of a 19-kg dog. Cross section changes most rapidly at the beginning and end of the thoracic aorta. At the latter site the average velocity has increased to twice its value in the ascending aorta and decreases rapidly thereafter. Changes in average velocity are due both to the progressive narrowing of the cross section and to the distribution of flow into major arteries. Relative importance of these two factors depends upon the location of a given segment. Arrows indicate anatomical landmarks. Abscissa: distance in cm from the aortic valve.
Because of the large increase in the total cross section of the vascular bed the average blood velocity must decrease toward the periphery. However, our data show that in individual vessels, such as the aorta, the drainage flow through branches may not compensate for changes in cross section and blood flow may be accelerated. Under such conditions, Poiseuille's law, which is based on a uniform cylindrical tube, is no longer valid. It is therefore of interest to compare the pressure differences predicted by Poiseuille's law with those which are actually observed. The pressure differences calculated for the observed mean flows in the individual segments are listed in column 9 of table 2. They increase from 43 dyn cm$^{-2}$ in the first segment to 360 dyn cm$^{-2}$ in the sixth segment and thereafter decrease again to 254 dyn cm$^{-2}$. The total pressure difference along the aorta amounts to about 1.6 cm H$_2$O if one neglects the convective acceleration, in contrast to the measured values which are in the order of 5 cm H$_2$O. One of the implications of this difference between observed and calculated values can be illustrated in the following example. In order to study arterial hemodynamics, models based on single uniform tubes have frequently been employed. In this case, wave reflection dominates the space- and frequency-dependent behavior of the pressure-flow relations. If one wants to represent the aorta by such an equivalent tube, keeping the total pressure difference constant, one has the choice between several possibilities, all of which lead to different results. From the sum of the individual resistances along the tube (table 2) one obtains a mean radius of 0.38 and a mean flow of 11.6 cm$^3$/sec. Averaging all the radii, the total resistance amounts only to 70 dyn cm$^{-5}$ sec and the mean flow becomes 23.5 cm$^3$/sec. Averaging the fourth power of the radius, the total resistance falls still further and the mean flow increases to 36 cm$^3$/sec. It is apparent from the data listed in table 2 that Poiseuille's law, and by inference, Womersley's theory, which were derived for circular tubes with uniform cross section, will have to be modified by the inclusion of terms accounting for the convective acceleration, if they are to be applied to whole arteries rather than to short segments.

Such modifications have been proposed by Hamel$^92$ and Streeter et al.$^93$ In the case of accelerated flows the velocity profile is considerably more blunt than that predicted for Poiseuille's flow and exhibits a marked boundary layer character. Except for a thin, peripheral layer where viscous forces are important, the flow behavior is determined primarily by inertial forces. In decelerating flows, on the other hand, the velocity profile resembles a cone, whose vertex angle depends heavily on the Reynolds number. Viscous forces are important throughout the cross section. Backflow, the early phase in the development of separation of the boundary layer, tends to develop. Since separation is associated with a retrograde pressure gradient and laminar flow can support only a very small pressure increase without the incidence of separation, the flow tends to become unstable.$^89$

Although small compared to the total pressure loss across a vascular bed, the pressure differences associated with geometrical tapering are considerably larger than those expected for a uniform tube. Their effects in terms of local flow and pressure patterns become significant in the larger vessels where tapering occurs. It has already been pointed out that geometrical tapering contributes markedly to the favorable transmission characteristics of the arterial tree by tending to stabilize the frequency-dependent parts of the input impedance.

The calculated changes in resistance, inertia and vascular distensibility (equation 5) associated with the geometrical tapering are illustrated for the individual segments of the aorta, the brachiocephalic-carotid and iliac-femoral arteries in figure 7. Distance is plotted along the abscissa, the aortic segments being in the center, the frontal part of the arterial tree to the left, and the hindpart to the right. As the cross section decreases, there is a progressive increase in resistance and inertia, while the distensibility diminishes markedly. The decrease in distensibility is con-
Changes in resistance (R), inertance (L) and distensibility (C) (calculated from equation 6) associated with the measured decrease in cross section (A) in the arterial tree of a 19-kg dog. From left to right: brachiocephalic-carotid channel, thoracic and abdominal aorta, and iliac-femoral channel. Distances are plotted on the abscissa, the head being on the left, and the feet on the right. Since there is no change in cross section in the proximal part of the carotid artery, the three parameters R, L and C are constant over a distance of 15 cm. Marked decrease in cross section between the origin and the bifurcation of the aorta leads to large changes in these three parameters. In the femoral artery the effects of geometrical tapering are greatest. Elastic tapering has been included in the calculation of C (C2 in Table 3). Note the discontinuities at the origin of the brachiocephalic and iliac arteries.

The observed increase in the elastic modulus of the vessel wall is included in addition to the geometrical tapering. This additional effect is much more marked in the peripheral arteries as compared to the aorta. The space dependence of the four parameters can be expressed as exponential functions in a first approximation (Table 3). Using the prediction formulas in Table 3 the values of the individual parameters were recalculated for each segment and compared with the initial data from which the prediction was derived. Despite the fact that none of the space dependences could be plotted on a straight line in Figure 6 (the deviation is rather large for the carotid artery, where no significant change in cross section occurs over a distance of 15 cm), the agreement between predicted and initial data is surprisingly good. In four other dogs the exponent characterizing the decrease in the cross section of the aorta varied between 0.022 and 0.036. Such an exponential space dependence of the various parameters could be introduced with relative ease into some of the more elaborate models of the arterial circulation if it can be established also for other major arteries than those investigated in this study. At present there are at least three models which represent the behavior of the input impedance at various sites in the arterial tree quite well.12-15 In each of these models the agreement between experimental and theoretical results has been achieved by a combination of the following factors:

1. The arterial tree is represented by a combination of small vascular segments to each of which the linearized Navier-Stokes equations, as developed by Womersley, are applied.
2. The large number of vascular segments permits the realization of geometrical tapering, although in a discontinuous rather than an exponential fashion.
3. Elastic tapering has been included either as a consequence of geometrical tapering (equation 6 and Tables 2 and 3) or by progressive alteration of the elastic modulus.

It remains to be seen how well these models perform in the simulation of cardiovascular control for which they were designed.

Appendix

ANALYSIS OF MEASUREMENT ERRORS

The measuring system employed in this study permits rapid analysis of large quantities of data. The errors associated with this analysis originate from four sources: 1) the transducers themselves, 2) the amplifiers and electrical connections, 3) the A-D conversion equipment and 4) the
Table 3

<table>
<thead>
<tr>
<th>Space Dependence of Vascular Parameters for Three Arterial Beds</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
</tr>
<tr>
<td>Aorta (n = 8)</td>
</tr>
<tr>
<td>Exp. approx.</td>
</tr>
<tr>
<td>Meas./pred.</td>
</tr>
<tr>
<td>Br. cephal.-carotid (n = 7)</td>
</tr>
<tr>
<td>Exp. approx.</td>
</tr>
<tr>
<td>Meas./pred.</td>
</tr>
<tr>
<td>Iliac-fem. (n = 7)</td>
</tr>
<tr>
<td>Exp. approx.</td>
</tr>
<tr>
<td>Meas./pred.</td>
</tr>
</tbody>
</table>

*Values for predicted changes in cross section (A), resistance (R), inertance (L) and volume distensibility (C) assumes constant elastic modulus, C2 assumes elastic tapering) are listed for three arterial beds.

†x is distance from origin in cm. Values for the ratio measured to predicted values (mean ± se) are shown below each prediction formula.

Mathematical computations in the computer. The calibration equipment (water manometer and infusion pump) is accurate to 0.1 cm H2O and 0.1 cm3/sec respectively. In order to minimize sampling errors, the pressure and flow pulses were amplified to the maximum compatible with the range of the A-D converter. Under these conditions the conversion error amounts to 0.2 cm H2O or to 0.1 cm3/sec. A comparable reading accuracy on an oscillograph would require that a 1-cm deflection of the tracing correspond to a pressure change of 4 cm H2O or to a flow change of 2 cm3/sec. The overall noise level of the system is less than 10 mv and of the same magnitude as the conversion error. The computational error in the calculation of the Fourier coefficient is negligible in comparison with the sampling errors. In order to reduce the overall measurement error from a given transducer output it is necessary both to decrease the noise level in the system and to increase the resolution of the A-D converter.

In a complex system significant errors are introduced if the impedances of the different components are not properly matched. Stray capacitances, invariably associated with multiple connections, cause errors both in magnitude and phase. For economic reasons electronic multichannel A-D conversion is usually achieved by means of multiplexers. The response of these electronic switches depends markedly on the output impedance of the previous stage in the system. If this output impedance is large, the accuracy of sampling may decrease significantly. For the low frequencies considered in this study, problems of impedance matching are less critical if one uses a system consisting only of transducers and a recording device.

Table 4 illustrates how the measurement errors increase for the higher harmonics. Eight randomly selected cardiac cycles were analyzed for the Fourier components of pressure and flow in the descending aorta at a heart rate of 2.38 cycles/sec, and the results averaged. For the first six harmonics the standard deviation of the modulus is with a few exceptions considerably less than 5% and that of the phase angle less than 8°. For the higher harmonics the magnitude of the modulus is less than 5% of the magnitude of the first harmonic and the errors for both modulus and phase become larger. Since the computer calculates pressure and flow values only to one decimal point (corresponding to the accuracy of the transducers), small values in the coefficients of the cosine or sine terms in the Fourier series may be in error by a factor of two. Both modulus and phase of the Fourier series described by equation 1 are calculated from these coefficients according to the relations:

\[
\sum_{n=0}^{N} c_n \cos (n\omega t - \theta_n) = \sum_{n=0}^{N} a_n \cos n\omega t + \sum_{n=0}^{N} b_n \sin n\omega t
\]

where \( c_n = (a_n^2 + b_n^2)^{1/2} \) and \( \theta_n = \tan^{-1} \frac{b_n}{a_n} \).

Hence it will be seen that if both the coefficients \( a_n \) and \( b_n \) are small, the error in the calculated moduli \( c_n \) and phases \( \theta_n \) become large. In terms of phase the error in the calculated angles will be largest if the sine (\( b_n \)) and cosine (\( a_n \)) coefficients are both small and of compar-
TABLE 4

Fourier Analysis of Flow, Q, and Pressure, P, in Descending Aorta of Dog

<table>
<thead>
<tr>
<th>Harmonic</th>
<th>Modulus cm³/sec</th>
<th>Phase °</th>
<th>Modulus cm H₂O</th>
<th>Phase</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>53.85 ± 1.91</td>
<td>133 ± 2</td>
<td>10.4 ± 0.8</td>
<td>199 ± 2</td>
</tr>
<tr>
<td>2</td>
<td>26.82 ± 0.64</td>
<td>235 ± 4</td>
<td>6.3 ± 0.3</td>
<td>289 ± 3</td>
</tr>
<tr>
<td>3</td>
<td>19.72 ± 0.64</td>
<td>341 ± 4</td>
<td>4.6 ± 0.1</td>
<td>37 ± 4</td>
</tr>
<tr>
<td>4</td>
<td>10.49 ± 0.53</td>
<td>69 ± 5</td>
<td>2.9 ± 0.0</td>
<td>136 ± 5</td>
</tr>
<tr>
<td>5</td>
<td>12.01 ± 0.64</td>
<td>184 ± 5</td>
<td>3.4 ± 0.1</td>
<td>244 ± 6</td>
</tr>
<tr>
<td>6</td>
<td>6.25 ± 0.42</td>
<td>271 ± 6</td>
<td>2.3 ± 0.0</td>
<td>5 ± 8</td>
</tr>
<tr>
<td>7</td>
<td>1.59 ± 0.42</td>
<td>18 ± 16</td>
<td>0.9 ± 0.1</td>
<td>123 ± 7</td>
</tr>
<tr>
<td>8</td>
<td>1.48 ± 0.42</td>
<td>6 ± 17</td>
<td>0.5 ± 0.1</td>
<td>172 ± 6</td>
</tr>
<tr>
<td>9</td>
<td>2.65 ± 0.53</td>
<td>137 ± 34</td>
<td>0.9 ± 0.0</td>
<td>270 ± 11</td>
</tr>
<tr>
<td>10</td>
<td>1.45 ± 0.32</td>
<td>230 ± 23</td>
<td>0.8 ± 0.1</td>
<td>42 ± 21</td>
</tr>
<tr>
<td>11</td>
<td>0.95 ± 0.32</td>
<td>259 ± 29</td>
<td>0.4 ± 0.4</td>
<td>155 ± 15</td>
</tr>
<tr>
<td>12</td>
<td>1.48 ± 0.42</td>
<td>2 ± 27</td>
<td>0.4 ± 0.0</td>
<td>215 ± 21</td>
</tr>
</tbody>
</table>

* Twelve harmonics were calculated and averaged over eight cardiac cycles, selected at random. The columns list the mean and standard deviation of the modulus ($c^2 = a^2 + b^2$), and the corresponding values for the phase angle ($\tan^{-1} \frac{b}{a} = \theta$). Note that the frequency content of pressure and flow above the sixth harmonic is small and the phase calculations therefore unreliable. Runs No. 51 to 60; $f = 2.38$ cycles/sec.

References


Pressure-Flow Relations in Dog Arteries
E. O. ATTINGER, H. SUGAWARA, A. NAVARRO, A. RICCETTO and R. MARTIN

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