ABSTRACT

Pulmonary vascular input impedance and hydraulic power were measured at various heart rates in 29 anesthetized and 5 unanesthetized dogs. Hydraulic power at the pulmonary veno-atrial junction was measured in 5 dogs. The pulmonary vascular impedance spectrum in the unanesthetized dogs did not differ significantly from that in the anesthetized dogs. Average pulmonary arterial power in the anesthetized dogs was 157 milliwatts (mw), of which 108 mw was associated with mean pressure and flow, and 49 mw with the pulsations around these means. Seventy-eight per cent of this input power was dissipated in passage through the pulmonary bed. Kinetic energy accounted for 2% of the total input power.

Because of a steep fall in impedance between zero and 3 cycles/sec, and a rate-dependent change in the harmonic structure of flow pulsations, there was an inverse relationship between heart rate and the input power for a given mean flow, up to 180 beats/min. Pulmonary vascular dimensions and elasticity, which determine impedance, thus embody a mechanism whereby tachycardia can increase pulmonary blood flow by as much as 35% with an increase in pulmonary arterial input power of less than 5%, without the intervention of vasomotor activity.

Additional Key Words

pulmonary artery  pulmonary veins  pulmonary circulation
vascular impedance  blood pressure  cardiac output  dogs

The physical characteristics of the normal pulmonary vascular bed are such that its input impedance varies considerably with frequency.1-3 The hydraulic power, or work per unit time, associated with blood flow in the pulmonary artery must be similarly frequency dependent. Pulmonary arterial pressure and flow waves in vivo can be shown by harmonic analysis to be equivalent to a collection of sinusoidal waves at many different frequencies, but the largest single component of each pulse is the fundamental harmonic, and the

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Supported in part by Grant HE-7174 from the National Heart Institute, U. S. Public Health Service and by the Clayton Fund. The computations were done in part at the computing center of the Johns Hopkins Medical Institutions, supported by Research Grant FR-00004 from the National Institutes of Health.

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Accepted for publication May 8, 1966.
frequency of this fundamental harmonic is equal to the heart rate. These facts suggested to us that heart rate might be a major factor in determining the hydraulic power at the inlet of the pulmonary bed. Pulmonary input impedance falls rather sharply as frequency rises from zero to 3 cycles/sec (Fig. 1), a range that includes fundamental harmonics for the heart rates usually found in the dog under resting conditions or during mild exertion. At heart rates up to 180 beats/min, therefore, an inverse relationship between heart rate and the hydraulic power required for a given pulmonary blood flow might be expected.

To test this hypothesis and explore its implications, we have measured pulmonary arterial input impedance and hydraulic power in anesthetized and unanesthetized dogs at various heart rates. An artificial pacemaker was used to provide a wider range of rates in some experiments. Most experiments were concerned with input impedance and power, but in one group of animals, pressure, flow, and power were measured in a pulmonary vein near its junction with the left atrium, as well as in the pulmonary artery, so that the energy dissipated by blood flow through the pulmonary bed could be calculated. Pulmonary impedances at very low frequencies, which could not be determined accurately at spontaneous heart rates, were defined by experiments on dogs with surgically induced complete heart block.

From the harmonic content of flow pulsations at different rates, and the impedance spectrum, which is independent of heart rate, it was possible to derive the general relationship between heart rate and hydraulic input power under the conditions of our experiments. The results confirmed the hypothesis that the power required for a given mean flow into the pulmonary bed decreases as rate increases, up to 3 cycles/sec. At heart rates up to about 180/min, in other words, increases in pulmonary blood flow can be accomplished more economically, in terms of the hydraulic power required, by increasing heart rate than by increasing stroke volume.

**Methods**

The experiments were carried out on mongrel dogs, weighing 11.7 to 28.9 kg (mean 18.7 kg). The methods employed in the anesthetized dogs and our techniques of data analysis were described in an earlier paper. In all experiments, pulmonary arterial blood flow was measured with an electromagnetic flowmeter (type K-2000 or M-4000, Medicon Division, Statham Instruments, Inc., Los Angeles, California), using hinged coreless probes. The frequency response of the flowmeters was determined, and appropriate correction factors were applied in the calculation of results. Pulmonary arterial pressures in the acute open-chest experiments, and left atrial pressures in all experiments, were measured with Statham P23Db gauges. The dynamic response of the catheter-gauge system was tested after each experiment and usually demonstrated a natural frequency of 90 to 110 cycles/sec, with relative damping of 0.3 to 0.4. In the unanesthetized animals pulmonary arterial pressure was measured with a Statham SF-1 catheter-tip gauge, which we found to have a natural frequency above 1,000 cycles/sec, with relative damping of 0.016.

Four kinds of preparation were studied.

1. Anesthetized, open-chest (25 animals): Pulmonary arterial pressure and flow and left atrial pressure were measured in this group, which included 13 control studies summarized in a previous paper. Input impedance and power at spontaneous heart rates (117 to 194 beats/min) were calculated in each of these animals; in two of them similar measurements were made after heart rate had been increased by electrical pacing through a small bipolar electrode on the right ventricle.

2. Anesthetized, open-chest, with complete atrioventricular block (4 animals): In these dogs, chronic complete atrioventricular block was produced by placing sutures through the endocardial surface of the right atrium in the region of the atrioventricular node. Twelve to twenty-one days after this operation, the same experimental procedures as in Group 1 were carried out. In each experiment input impedance and power were measured at the spontaneous rates, which ranged from 0.40 to 0.71 beats/sec, and at faster rates (up to 4.42 beats/sec) produced by electrical stimulation of the right ventricle.

3. Anesthetized, open-chest, pressure and flow measured simultaneously in pulmonary artery and a pulmonary vein (5 animals): In addition to the pulmonary arterial flowmeter probe and catheter, a second probe (Medicon, type ST, lumen diameter 2 to 4 mm) was placed around one of the left pulmonary veins 1 to 3 cm from its junction with the left atrium. Pressure at this site in...
PULMONARY BLOOD FLOW AND HYDRAULIC POWER

the vein was measured through a small polyethylene catheter introduced into a small tributary vein and advanced downstream.

4. Unanesthetized (5 animals): These dogs were subjected to thoracotomy under sterile conditions and general anesthesia, and a flowmeter probe placed around the pulmonary artery, using surgical procedures similar to those previously described for acute experiments. The probe cable and external plug were brought out through the dorsolateral thoracic wall on the left side. In some instances, the flared end of a length of polyethylene tubing was inserted into the left atrium through its appendage, anchored with sutures, and brought out through the posterior chest wall, so that left atrial pressures could also be measured. This catheter was kept filled with heparinized saline, and its external end closed with a metal fitting when not in use. After this operation, the animal was brought to the laboratory almost daily and trained to lie quietly while the flowmeter cable was connected and recordings made. The measurements of input impedance and hydraulic power reported here were carried out 11 to 61 days after implantation of the flowmeter probe. On the day of the experiment, the right jugular vein was exposed under local anesthesia and a miniature catheter-tip manometer (Statham type SF-1) advanced through this vein into the pulmonary artery under fluoroscopic guidance. When this catheter had been placed so that its tip lay in the main pulmonary artery just beyond the flow probe, a ligature was placed around the vein to hold it in place, and the incision temporarily closed and bandaged. The animal was then allowed to move around the laboratory more or less freely for a period of 10 to 20 minutes, after which he was placed on a table, on which he was accustomed to lying quietly, either prone or somewhat on his right side. The heart rate under these conditions usually slowed to 100 beats/min or less, often with a prominent sinus arrhythmia, and pressures and flow were then recorded.

Data were recorded both graphically (model DR-8 multi-channel recorder, Electronics for Medicine, White Plains, New York) and on magnetic tape (model 8100 tape recorder, Honeywell, Inc., Denver, Colorado). Analog-to-digital data conversion was accomplished electronically in most cases (Computer of Average Transients, model 400-B, Technical Measurements Corp., North Haven, Connecticut), and with a manual reading device applied to the graphic record (Datascaler, Westfield, Massachusetts) in others. Digital data were stored on punched cards and analyzed on an IBM 7094 digital computer.

The computations of impedance and hydraulic power were based on harmonic analysis, by which the observed pressure and flow waves are expressed as Fourier series. Flow, for example, can be represented as a function of time, \( Q(t) \), by:

\[
Q(t) = Q_0 + \sum_{n=1}^{N} Q_n \sin(n\omega t + \phi_n)
\]

where \( Q_0 \) = mean flow; the subscript \( n \) indicates the harmonic number; \( N \) = the total number of harmonics included in the series; \( Q_n \) is the amplitude of the \( n \)th harmonic and \( \phi_n \) its phase angle, and \( \omega \) = the fundamental frequency of pulsation in radians/sec. Pressure waves can be expressed in the same form, and if \( P_0 \) = mean pressure, \( P_n \) = amplitudes of the pressure harmonics and \( \beta_n \) their phases, then the impedance modulus, \( Z_n \), at a frequency of \( n\omega \) is:

\[
Z_n = P_n/Q_n
\]

and impedance phase at that frequency, \( \Theta_n \), is:

\[
\Theta_n = \beta_n - \phi_n
\]

The hydraulic energy of the blood at any site in the circulation is of three different kinds:

1. Pressure energy, \( W \), a kind of potential energy. The product of intravascular pressure and the volume rate of blood flow at any instant equals potential power, or potential energy manifested per unit time.

2. Kinetic energy, \( K \), equal to one-half the blood mass, \( m \), multiplied by the square of its velocity \( (u^2) \). (\( m = \rho v \), where \( \rho \) = blood density, and \( v \) = the volume of blood being considered.)

3. Energy of position, or energy conferred by virtue of the vertical height of blood above some level of reference. Since these experiments were concerned with energy at the inlet and outlet of the pulmonary bed, which were at approximately the same level, this element was assumed to be negligible.

Accurate determination of the hydraulic energies involved in blood flow requires continuous measurements of instantaneous blood flow and pressure, and integration of the appropriate quantities throughout the heart cycle. Such continuous measurements have recently been used by a number of investigators for this purpose, in place of the older approximations based on mean pressure and flow, but the calculation of energy or power from the sinusoidal terms given by harmonic analysis of experimental data is still much more familiar in electrical engineering than in circulatory physiology. The present work is concerned principally with average power throughout the cardiac cycle, rather than instantaneous power variations as a function of time.

Hydraulic pressure energy and kinetic energy can each be divided into a portion associated
with mean pressure and flow (the energies that would exist if there were no oscillations around the mean), and a portion associated with the pulsations. Since our data were analyzed by making a finite number of consecutive measurements of the experimental observations, our computations took the form of finite series rather than integrals.

The average energy per unit time, or average power, as pressure energy associated with mean terms was calculated by multiplying mean pressure and mean flow:

\[ W_M = P_0 Q_o \]  \hspace{1cm} (4)

The oscillatory component of pressure energy, \( W_o \), which can be calculated by multiplying the oscillatory terms of the Fourier expressions for pressure and flow, was put in a form convenient for our purpose by substituting impedance-flow relationships (equations 2 and 3) for pressure, giving:

\[ W_o = \frac{1}{2} \sum_{n=1}^{N} (Q_n)^2 Z_n \cos \theta_n \]  \hspace{1cm} (5)

The total hydraulic power as pressure energy (\( W_T \)) is the sum of these two components:

\[ W_T = W_M + W_o \]  \hspace{1cm} (6)

The kinetic energy per unit time associated with mean flow, \( K_M \), is:

\[ K_M = \frac{1}{2} \frac{dm}{dt} u^2 \]  \hspace{1cm} (7)

Since \( \frac{dm}{dt} \) for mean flow equals blood flow \( (Q_o) \) times blood density \( (\rho, \text{ assumed to be 1.055 g/cm}^3) \), and velocity equals blood flow divided by the cross-sectional area of the vessel, \( A \), equation 7 becomes:

\[ K_M = \frac{\rho Q_o^3}{2 \pi A^2} \]  \hspace{1cm} (8)

The oscillatory component of kinetic power \( (K_o) \) can be calculated from the Fourier series representing flow, just as the oscillatory component of potential power \( (W_o) \) can be calculated from the Fourier series for pressure and flow (equations 1 to 5). The kinetic analogue of equation 5, however, contains an unwieldy number of terms, and we found it more convenient to compute \( K_T \) and \( K_0 \) from the original flow data (in digital form) than from their Fourier equivalent. Letting \( \Delta t \) represent the interval between successive readings of our pressure and flow observations (20 msec in the first 13 anesthetized dogs, 10 msec in all others), \( J = \) the total number of such successive readings so that the duration of the period analyzed, \( T = J \Delta t \), and \( Q_j = \) the observed flow at time \( = j \Delta t \), the total kinetic power (mean and oscillatory combined, \( K_T \)) becomes:

\[ K_T = \frac{\rho}{2} \sum_{j=0}^{J} (Q_j)^2 \]  \hspace{1cm} (9)

The oscillatory component, \( K_0 \), can then be calculated by subtracting the mean component from the total:

\[ K_0 = K_T - K_M \]  \hspace{1cm} (10)

The cross-sectional area of the pulmonary artery could be calculated fairly accurately by using the area of the lumen of the flowmeter probe and assuming a wall thickness equal to 10% of the radius. The total area of the pulmonary veins, needed to calculate \( K_T \) for the output of the pulmonary bed from equation 9, could not be determined exactly. Measurements of the circumference of each pulmonary vein in 2 dogs, however, indicated that total venous cross section was approximately equal to pulmonary arterial cross section, and this equivalence was assumed in our calculations.

Equations 4 to 10 give average hydraulic power during the period analyzed, which in these experiments always consisted of an integral number of cardiac cycles, ranging from 4 to 10. From the average power and the duration of a cardiac cycle, stroke work could be computed. If pressure is expressed in dyne/cm², flow in cm³/sec, impedance modulus in dyne sec/cm⁶, impedance phase in radians, velocity in cm/sec, area in cm², and time in sec, these equations give power in ergs/sec (1 mw = 10⁴ ergs/sec, = 0.102 g-m/sec).

Instantaneous power as a function of time was also computed from the data of some experiments, in addition to average power. The time course of 'resistive power' and 'reactive power' was calculated in these instances by summing separately the products of in-phase and out-of-phase pressure and flow terms.

\section*{Results}

\subsection*{GENERAL}

The average pulmonary arterial pressure and flow and other relevant variables in the 25 anesthetized dogs and 5 unanesthetized dogs are summarized in Table 1. Cardiac output (pulmonary blood flow) was somewhat lower in the unanesthetized animals, but this merely reflected the smaller size of the dogs in this group, since cardiac output per kilogram of body weight was not significantly different in the anesthetized and unanesthe-
TABLE 1

Hemodynamic Data from Anesthetized and Unanesthetized Dogs

<table>
<thead>
<tr>
<th>Variable</th>
<th>Anesthetized, open-chest</th>
<th>Unanesthetized</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean (n = 25) SEM</td>
<td>Mean (n = 5) SEM</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>18.71 ± 1.95</td>
<td>14.75 ± 1.26</td>
</tr>
<tr>
<td>Heart rate (beats/sec)</td>
<td>2.64 ± 0.07</td>
<td>1.66 ± 0.06</td>
</tr>
<tr>
<td>Stroke volume, RV (cm³/stroke)</td>
<td>16.2 ± 1.8</td>
<td>18.7 ± 0.9</td>
</tr>
<tr>
<td>Mean pressures:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>PA (mm Hg)</td>
<td>18.8 ± 0.7</td>
<td>17.2 ± 0.8</td>
</tr>
<tr>
<td>LA (mm Hg)</td>
<td>5.3 ± 0.4</td>
<td></td>
</tr>
<tr>
<td>Mean blood flow, PA:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>(cm³/sec)</td>
<td>42.0 ± 4.5</td>
<td>30.9 ± 1.3</td>
</tr>
<tr>
<td>(cm³/sec kg)</td>
<td>2.22 ± 0.16</td>
<td>2.14 ± 0.17</td>
</tr>
<tr>
<td>Average hydraulic input power, PA:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>As energy of pressure (Wₚ) (mw)</td>
<td>146.2 ± 18.7</td>
<td>131.6 ± 12.1</td>
</tr>
<tr>
<td>As kinetic energy (Kᵧ) (mw)</td>
<td>11.0 ± 2.8</td>
<td>12.3 ± 1.7</td>
</tr>
<tr>
<td>Wₚ + Kᵧ (mw)</td>
<td>157.2 ± 14.6</td>
<td>143.9 ± 12.5</td>
</tr>
<tr>
<td>(Wₚ + Kᵧ)/body weight (mw/kg)</td>
<td>7.66 ± 0.66</td>
<td>10.34 ± 1.84</td>
</tr>
</tbody>
</table>

RV = right ventricle; PA = pulmonary artery; LA = left atrium; mw = milliwatts.

FIGURE 1

Pulmonary vascular input impedance in anesthetized, open-chest dogs. The points represent mean values and

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The average pulmonary vascular input impedance measurements in the anesthetized dogs are shown in Figure 1. These do not differ significantly from those we reported earlier, except for the values at frequencies below 2 cycles/sec. The present experiments give more accurate data in this region than were available before, and show a slight convexity of the impedance modulus toward the ordinate. They also suggest a turning of the impedance phase angle back toward zero at low frequencies, as the transmission-line model of vascular beds predicts.

Consecutive measurements of impedance in a given animal at different heart rates confirmed our earlier conclusion that the impedance modulus and phase angle are sensitive to changes in heart rate. The bars ± 1 standard error of the mean, in 29 animals (see Methods, groups 1 and 2). The frequencies plotted on the abscissa refer to the frequency of sinusoidal waves derived from pressure and flow waves by harmonic analysis. Ordinates are impedance modulus (above) and impedance phase angle (below).
pedance spectrum (the total pattern of impedance moduli and phases versus frequency, as shown in Fig. 1) is independent of heart rate under these conditions. The impedance at any given frequency, in other words, remains the same whether the heart rate is fast or slow. As heart rate changes, so does the frequency of the fundamental harmonic of the pressure and flow pulsations, and the impedance encountered at that fundamental frequency, but the impedance spectrum itself remains constant. It should be noted that the abscissae of Figures 1 and 2 represent the frequency of sinusoidal components of the pulse, not heart rate.

In each of the unanesthetized dogs, input impedance was not significantly different from those in the anesthetized animals. As the averages in Figures 1 and 2 show, impedance magnitude in both groups reached a minimum in the neighborhood of 3 cycles/sec, then oscillated with frequency about a level of approximately 3,000 dyne sec cm⁻² kg. The zero-phase crossing was at a slightly higher frequency in the unanesthetized group.

**HYDRAULIC POWER**

The mean values for average hydraulic input power in the anesthetized and unanesthetized animals are compared in Table 1. The combined pressure and kinetic power \(W_p + K_p\) per unit body weight was higher in the unanesthetized group, with their slower heart rate, even though the mean pulmonary blood flows per unit body weight were not significantly different in the two groups. Various subdivisions of the hydraulic power associated with pulmonary blood flow in anesthetized animals are shown in Table 2. The data for output power in this table were derived from 5 dogs in which both input and output power were measured, and the data for input power from this group plus the 25 animals in which input alone was determined. Mean cardiac output and body weight were similar in both groups. The averages given in Table 2 are shown graphically in Figure 3, which indicates the average power at the input to the pulmonary bed (pulmonary artery) the outflow from the bed (pulmonary veins), and the difference between input and output, or power dissipated in moving blood through the bed. The power associated with pulsations of flow in the pulmonary artery

### Table 2

<table>
<thead>
<tr>
<th>Hydraulic Power Associated with Pulmonary Blood Flow in Anesthetized Dogs</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Pressure power</strong></td>
</tr>
<tr>
<td>Mean terms</td>
</tr>
<tr>
<td>Pulsatile</td>
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<tr>
<td>Combined</td>
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<td><strong>Kinetic power</strong></td>
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<tr>
<td>Mean terms</td>
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<tr>
<td>Pulsatile</td>
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<tr>
<td>Combined</td>
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<tr>
<td><strong>Total power</strong></td>
</tr>
<tr>
<td>Mean terms</td>
</tr>
<tr>
<td>Pulsatile</td>
</tr>
<tr>
<td>Combined</td>
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</tbody>
</table>

*Figure 2*

Average pulmonary vascular input impedance in 5 resting unanesthetised dogs.
Average hydraulic power at the inlet (pulmonary artery) and outlet (pulmonary vein near left atrium) of the pulmonary bed in anesthetized, open-chest dogs. (See Table 2.) The difference between the two is the power dissipated (Diss.) by blood flow through the pulmonary vessels. Power associated with mean pressure and flow (mean terms), and that associated with pulsations of pressure and flow (oscillatory terms) are indicated separately, as are the pressure-energies (pressure × flow) and kinetic energies per unit time. Mean terms are equivalent to the power that would exist if flow were constant, without pulsations, at the observed mean flow and pressures (see Table 1). The oscillatory terms represent the extra power inherent in pulsations.

averaged 31% of the total input power in the anesthetized dogs, and 48% in the unanesthetized group.

The pulsations are so attenuated by passage through the pulmonary bed that very little of the original pressure and flow oscillations remains at the terminus of the pulmonary veins, and as a consequence the greater part of the 'pulsatile power' is dissipated in the pulmonary bed. The use of mean pressure and flow to calculate power would therefore underestimate the true values by a considerable amount, as others have pointed out.\textsuperscript{6,7,13}

In the absence of pulsatile flow measurements, investigators have often substituted mean systolic pressure, flow, and velocity in equations 4 and 8 to arrive at an approximation of the external work of the heart.\textsuperscript{8,12} In the present experiments, such calculations would underestimate the pressure power in the pulmonary artery by 10%, on the average, with individual variations ranging from -21 to +10%. Calculation of the kinetic power in the pulmonary artery from mean systolic velocity would give values ranging from 79 to 17% less than those given by integration, the average underestimate being 48%.

Kinetic power amounted to 7% of the total input power, which is approximately the same proportion as estimated by Prec and his colleagues in human subjects.\textsuperscript{12} Our assumption of equal cross section for the pulmonary artery and pulmonary veins was equivalent to assuming that none of the 'steady-flow' kinetic power ($K_M$) was dissipated. The continuously changing blood velocity and total vascular cross section as blood moves through the pulmonary bed necessarily leads to interconversion of pressure and kinetic energies, but no information about these can be gained from the present studies at the inlet and outlet of the bed.

'Reactive power'\textsuperscript{9} was calculated in 3 of the anesthetized dogs and averaged 10% of the total input energy during systole; by definition, mean reactive power is zero, and the systolic portion was exactly balanced by a negative reactive power in diastole. If the pulmonary bed were equivalent to a simple electrical circuit, this would mean that 10% of the total stroke work had been expended in distending the walls of the pulmonary artery, and the same amount of energy returned to the circulation as these walls recoiled during diastole. This simple analogy does not apply, however, to the pulmonary vascular bed, where the phase relations of pressure and flow depend on reflected waves and pulse wave velocities,\textsuperscript{1,2,4,14} and the physiological significance of this reactive power, if any, cannot be interpreted at present.

**Relation Between Heart Rate and Flow Harmonics**

The duration of ventricular systole and flow into the pulmonary artery depend principally on heart rate. The amplitude of the flow pulse, on the other hand, is a function of both stroke volume and rate; if rate is constant, the pulse amplitude varies directly with stroke volume.
Changes in harmonic proportions of flow pulsations (ratio of harmonic moduli to mean flow) with heart rate, for the first 5 harmonics, in anesthetized dogs. Vertical bars indicate ±1 standard error of the mean, and n the harmonic number. This graph, together with the simultaneous changes in phase angles (not shown) gives a quantitative description of the observed changes in the shape of the flow pulse with changes in heart rate.

and hence with cardiac output. This relation between the shape of the flow pulse and the heart rate led us to look for equivalent relationships in the Fourier expressions for flow, and to express them quantitatively. At any given rate, the ratio of each harmonic amplitude to mean flow \(\frac{Q_n}{Q_o}\), was found to be fairly consistent (Fig. 4). These ratios, which we will call 'harmonic proportions,' describe the normal shape of the flow pulse at a given heart rate, and appear to be independent of the absolute value of \(Q_o\).

The average harmonic proportions at different heart rates in our experiments on anesthetized animals are shown in Figure 4, and the proportions in the unanesthetized dogs were similar. The data for this graph were derived by subdividing the 51 observations made at different rates on 29 dogs into 6 groups, according to heart rate (0 to 0.99, 1.00 to 1.99, 2.00 to 2.49, 2.50 to 2.99, 3.00 to 3.49, 3.50 to 4.49 beats/sec). Group limits were chosen so that from 7 to 13 observations were included in each group, except for the group centered at 400 beats/sec, which contained only 4 observations.

With increasing heart rate, the ratio \(\frac{Q_n}{Q_o}\) became smaller for all harmonics; this change is the harmonic equivalent of the decrease in stroke volume that must occur if cardiac output remains constant while heart rate increases. The decrease was greatest in the higher harmonics, so that a larger and larger proportion of the flow pulse was represented by the first, or fundamental, harmonic. As heart rate increases, in other words, and diastole shortens to a greater degree than systole, the flow pulse comes more and more to resemble a simple sinusoidal wave at the frequency of the heart rate. At slow rates, on the other hand, the relatively long diastole with flow at zero represents additional harmonics. Higher harmonics also represent the steeper parts of the systolic flow curve, but this is a less important factor than the length of diastole in determining the size of such harmonics, as the small amplitude of the higher harmonics at fast rates demonstrates.

The phase of each flow harmonic also changed in a consistent way with heart rate. As rate increased, phase became more negative, so that pressure waves tended to lag behind flow by an increasing angle.

**RELATION OF HYDRAULIC POWER TO HEART RATE**

If harmonic proportions of flow depend principally on rate under these experimental conditions, as these results suggest, then:

\[
\frac{Q_n}{Q_o} = C_n Q_o
\]

where \(C_n\) is a function of heart rate and can be evaluated from the data illustrated in Figure 4. (In Fig. 4 and other illustrations heart rate is given in cycles/sec, \(f\), rather than radians/sec, \(\omega\); \(\omega = 2\pi f\).) Substitution of equation 11 in equation 1 then gives:

\[
Q(t) = Q_o + \sum_{n=1}^{N} C_n Q_o \sin (n\omega t + \phi_n)
\]

and a similar substitution in equation 5 gives:

\[
W_o = \frac{1}{2} \sum_{n=1}^{N} (C_n Q_o)^2 Z_n \cos \theta_n
\]

or, on rearrangement,
All the terms summed at the right on equation 14 are functions of frequency, and can be evaluated from the data in figures 1 and 4, so that we may define a 'pressure power coefficient,' $G(f)$, as a function of frequency such that:

$$G(f) = \sum_{n=1}^{N} C_n Z_n \cos \theta_n$$

(15)

substituting this in equation 14 gives:

$$W_o = \frac{1}{2} Q_o^2 G(f)$$

(16)

At any heart rate, $f$, in other words, the pulsatile component of pressure power, is proportional to the square of the mean flow. By using equations 16 and 4 to compute $W_o$ and $W_M$, and equation 12 to calculate values for $Q$, so that $K_o$ may be determined by equation 9, total hydraulic power at the input of the pulmonary bed under the conditions of these experiments can be predicted from mean flow and the frequency of the heart beat. The graphs in Figures 5 and 7 are the results of such calculations.

Figure 5 represents estimates of the power dissipated in moving blood through the pulmonary bed at different heart rates, assuming the impedance spectrum shown in Figure 1, the harmonic proportions in Figure 4, and a mean blood flow of 42.0 cm³/sec. Hydraulic power remaining at the outlet of the system was assumed to remain constant at the levels indicated in Table 2. This assumption that output power is independent of heart rate is consistent with our observations, though further experiments would be required to rule out small variations in $W_o$ and $K_o$ with rate. Since our assumption of equal cross-sectional areas
at the inlet and outlet of the bed implies no net loss in the 'steady-flow' component of kinetic power, $K_M$ does not appear in Figure 5. The marked decrease in oscillatory power ($W_o$ and $K_o$) with increasing rate up to about 165 beats/min is a reflection of the changes in harmonic proportions ($C_n$) and impedances ($Z_n \cos \theta_n$) with rate. Since constant output power has been assumed, the relation between input power and rate has the same shape as the relation between dissipated power and rate. The net effect is that input power and power dissipated for a given mean flow are both reduced by approximately one-half when heart rate increases from 60 to 180 beats/min, and remain almost constant as rate increases beyond this limit.

The inverse relation between power and heart rate within this range was found in each experiment where heart rate changed while other conditions remained constant. An example is illustrated in Figure 6, which shows records from a dog with complete heart block, paced at two rates. When rate was increased from 32 to 100 beats/min, the total pulmonary arterial input power did not change significantly, although mean pulmonary blood flow increased from 38.4 to 51.6 cm$^3$/sec.

![Figure 6](http://circres.ahajournals.org/)

**Figure 6**

Pulmonary arterial flow, pressure, and power (work/sec) in an anesthetized, open-chest dog with surgically produced complete heart block, at two heart rates controlled by an artificial pacemaker through a bipolar electrode on the right ventricle. Mean flow ($Q$) and pressure ($P_{pa}$) are shown on the records, and the input powers are given below in gram-meters per second. When the rate is raised from 32/min (slightly above the spontaneous idioventricular rate in this animal) to 100/min, mean flow increases while stroke volume and the amplitude of the flow pulsations decrease. The power associated with mean pressure and flow (steady) increases along with mean flow, but the diminished pressure and flow pulsations result in a decrease in the oscillatory power components (pulsatile) so that the total input power does not change significantly. In this instance, therefore, the combination of increased rate and decreased stroke volume raised the pulmonary blood flow by 34 per cent without requiring an increase of input power.
Discussion

The measurement of hydraulic energy from pulsatile pressure and flow has long been used in studying the external work of the heart, but rarely applied to blood vessels. Estimation of the energy dissipated by blood flow through a vascular bed from measurements of hydraulic energy entering and leaving the bed has not, so far as we are aware, been reported previously, although a paper by Skalak, Wiener, Morkin and Fishman, now in press, considers the energy dissipation in the pulmonary system as a function of time during a cardiac cycle.

The energy associated with blood flow in the pulmonary artery depends not only on the characteristics of the pulmonary bed, as expressed by pulmonary impedance, but also on the characteristics of the pulse generated by the right ventricle. Our results show that these pulse characteristics, expressed as harmonic proportions, are rate dependent (Fig. 4), and are related to the pulmonary impedance spectrum in such a way that the pulmonary arterial input power with a given mean flow varies inversely with heart rate up to about 165 beats/min (Fig. 5).

This relation between hydraulic power and heart rate has its origin in the structure and physical properties of the pulmonary vascular bed on the one hand, and the characteristics of ventricular ejection on the other. Two factors are involved in this relationship: the spectrum of impedance versus frequency, and the changes in harmonic content of the flow pulse that attend changes in rate and stroke volume.

The normal pulmonary vascular impedance spectrum, which initially suggested to us the possibility of a power-rate relationship, depends on the dimensions, branching pattern, and elasticity of the pulmonary vessels. Because of the steep fall of impedance at low frequencies, which reaches a minimum at about 3 cycles/sec, the input power associated with a sinusoidal flow wave of specified amplitude must inevitably decrease as frequency increases, and therefore this inverse relationship must hold for the fundamental harmonic of any pulmonary arterial flow curve. The second and higher harmonics of flow pulsations, however, may occur in a frequency range where impedance is either rising or falling moderately (Fig. 1), and consequently the input power of the pulsations of flow and pressure found in vivo can be determined only by empiric observation of harmonic proportions as well as impedance. In these experiments, such observations showed that the fundamental harmonic and the falling impedance it encounters as rate rises outweigh the contribution of higher harmonics.

The second factor involves the shape of the flow pulse, which can be described quantitatively by a Fourier series. Normal ventricular function is such that stroke volume is controlled predominantly by changes in the amplitude of flow during systole, rather than by variations in the duration of systole. The harmonic equivalent of this fact is that the amplitude of each flow harmonic (Q in equations 1, 5, etc.) becomes smaller as stroke volume decreases. Obviously, the greater the number of strokes per unit time for a given mean flow, the smaller the stroke volume, and the smaller the absolute amplitude of each flow harmonic. This in itself tends to diminish the input power, quite apart from the simultaneous effects of impedance. Put in another way, oscillations of flow involve an 'extra' amount of work per unit time (represented by the oscillatory terms in Fig. 3, for example) beyond that required for constant flow, and the smaller the oscillations, the smaller this 'extra cost of pulsation.' Taylor has made this same point in a somewhat different theoretical context.

Since these results show that the hydraulic input power for a given mean flow declines as rate increases, it follows that less input power is required to raise pulmonary blood flow by increasing heart rate than by increasing stroke volume, within limits. Figure 7, based on the impedances and harmonic proportions of Figures 1 and 4, illustrates this point by indicating the oscillatory component of pressure power (W) at different levels of pulmonary blood flow, or cardiac output, for
Relation between mean pulmonary blood flow (abscissa) and the oscillatory component of hydraulic input power at the pulmonary artery, (ordinate) calculated from equation 16 and the data illustrated in Figures 1 and 4. Interrupted lines show the relationship when rate remains constant and flow is increased solely by increments in stroke volume; power is then proportional to the square of the mean flow. Continuous lines indicate the change in power when stroke volume remains constant and flow is raised by increasing the heart rate. Constant-rate curves are shown for 3 different rates, labeled in beats per second (f = 1.0, 2.0, 3.0), and constant-stroke curves for 3 stroke volumes, labeled in cm$^3$/stroke ($S = 10, 20, 30$). At rates above 3.0/sec, the constant-stroke curves are almost identical with the constant-rate curve for 3.0 beats/sec. Comparison of the curves shows that flow can be increased with less input power by raising heart rate than by enlarging stroke volume.

Three heart rates, stroke volume remaining constant, and for three stroke volumes, heart rate remaining constant.

If rate remains constant, power will vary as the square of the mean flow, as equation 16 implies. The interrupted lines in Figure 7, which show mean flow and power as stroke volume varies while rate remains constant, demonstrate this relationship. When rate varies while stroke volume remains constant (continuous lines in Fig. 7), the relationship is more complicated, for as rate and flow rise, the pressure power coefficient $G(f)$, equation 15 falls. The result is that power rises more slowly when increased flow is accomplished by increasing the rate at a given stroke, than by increased stroke volume at a given rate. At rates between 130 and 170 beats/min the constant-stroke curves of Figure 7 reach a plateau so that increases in mean flow of 20 to 30% occur with little change in input power. As the rate nears 180 beats/min, the constant-stroke curves approach the constant-rate curve for 180 beats/min, reflecting the relatively small variations in impedance at higher frequencies.

In the ordinary adjustments of cardiac output to metabolic demand in the normal animal, neither heart rate nor stroke volume remains constant. When our unanesthetized dogs were lying down, at rest and apparently relaxed, the heart rate would often slow to about 85 beats/min. Any signs of increased attention or alertness (lifting the head, waggling the tail, etc.) were accompanied by an increase in rate, usually to 100-120 beats/min,
and an increase in pulmonary blood flow of 20 to 35%, with a slight decrease in stroke volume. Such an increase in rate and decrease in stroke volume afforded an elevation of cardiac output with little or no increase in the hydraulic input power to the pulmonary bed, which is the result predicted by the data presented in Figure 7. Since the input power at the pulmonary artery equals the external work of the right ventricle per unit time, plus the much smaller amount of power corresponding to diastolic filling of the ventricle, this raising of cardiac output by a combination of increased rate with decreased stroke volume operates as a kind of energy-saving mechanism by which output can be increased as much as one-third above the basal level with little or no increase in external work of the right heart. As Figure 7 indicates, a heart rate of about 180 beats/min is the limit to which this mechanism can be exploited. Further elevation of mean flow by increases in rate above this limit entail approximately the same energy cost as would equivalent enlargements of stroke volume.

This mechanism presumably operates under any natural or experimental conditions where the impedance spectrum and rate dependence of harmonics are constant and similar to those found in the present experiments. Both these variables can be altered by neural or humoral stimuli that induce vasomotor responses, however, and extrapolation from these results to the exercising dog, for example, is not warranted.

Although our results show that blood can be moved through the pulmonary bed more efficiently as rate increases, in the sense that less hydraulic input power is required and less power dissipated for a given mean flow, it does not necessarily follow that the energy requirement of the right ventricle behaves in the same way. Changes in pulmonary arterial input power in our experiments presumably reflected changes in external work of the right ventricle per unit time, since these differ only by the power contributed by diastolic filling, which is small and relatively little affected by rate, but myocardial efficiency under these conditions is not known. External work is only a small part of the total energy expended by the heart, and there is no evidence to suggest that the optimal frequency of contraction for the myocardium coincides with the optimal frequency for moving blood through the pulmonary bed.

The experiments of Laurent and his colleagues on open-chest dogs show that myocardial efficiency, defined as the ratio of external work to myocardial oxygen consumption, falls as heart rate increases if external work is kept constant. The present results show only that the energy delivered to the pulmonary artery for a given mean flow diminishes as heart rate increases. Whether this 'gain in efficiency' in moving blood through the lungs is outweighed by a loss of efficiency in right ventricular function as rate increases is a question that remains to be answered.

The conclusions drawn from these measurements in the pulmonary circulation probably apply in principle to the systemic circulation as well, for the input impedance spectra of the pulmonary artery and aorta are similar in contour, though different in amplitude. Inasmuch as the ratio of pulse pressure to mean pressure is smaller in the aorta than in the pulmonary artery, the hydraulic power associated with pulsations represents a smaller fraction of the total power in the systemic than in the pulmonary circulation.

References

Hydraulic Power Associated with Pulmonary Blood Flow and its Relation to Heart Rate
WILLIAM R. MILNOR, DEREK H. BERGEL and JACK D. BARGAINER

Circ Res. 1966;19:467-480
doi: 10.1161/01.RES.19.3.467

Circulation Research is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7330. Online ISSN: 1524-4571

The online version of this article, along with updated information and services, is located on the
World Wide Web at:
http://circres.ahajournals.org/content/19/3/467

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