Network model of pulsatile hemodynamics in the microcirculation of the rabbit omentum

JOSEPH F. GROSS, MARCOS INTAGLIETTA, AND BENJAMIN W. ZWEIFACH
Department of Chemical Engineering, University of Arizona, Tucson, Arizona 85721; and Physical Sciences Department, Rand Corporation, Santa Monica, 90406, and Ames-Bioengineering, University of California, San Diego, La Jolla, California 92037

With the development of the pressure servo-nulling technique by Wiederhielm, Woodbury, and Rushmer (22), the methodology of Landis could be applied to monitor dynamic events. They first utilized this technique in the microcirculation of the frog mesentery and showed that pressures are pulsatile in arteriolar capillaries with diameters as small as 26 μm.

Pulsatile flow was observed in the cat mesentery by Bloch (2), Wayland and Johnson (21), and Gachtings, Meiselman, and Wayland (5), and in the omentum by Intaglietta et al. (7–9). Rosenblum (20) observed pulsatile flow in arterioles and venules of subarachnoid space. Gachtings (4) measured venous pulsatile pressure in small mesenteric vena. Although these investigations provide evidence for significant pulsatile conditions in the microvasculature, most studies assume the opposite. Furthermore, in most microcirculatory studies, the site of the measurement is identified solely in terms of arterioles, capillaries, and venules without specification of the location within the branching order.

Given these considerations, we have systematically determined experimentally the nature of pulsatile flow in the microcirculation of the rabbit omentum in terms of the level of branching within the network of blood vessels and have formulated an analytical network model that comprises the structural properties at each level of branching.

EXPERIMENTAL METHODS

The experimental aspect of this investigation is aimed at characterizing pulsatile conditions in the microvasculature of the rabbit omentum as a function of the level of branching where the measurements are made. Toward this end, pressure and red cell velocity were determined throughout the microcirculation of the omentum of anesthetized rabbits.

The methodology for the study of circulation at the arteriole, capillary, and venule levels in the rabbit omentum has been described in a previous publication by Zweifach and Intaglietta (23). In brief, rabbits of 1.5–2.4 kg weight are anesthetized with phenobarbital (30 mg/kg body wt), and the omentum is exposed through a lateral incision in the abdominal wall. The animal is supported in a cradle attached to the stage of an intravital microscope, and the omentum is draped over a glass pedestal consisting of a flat circular glass disc 1 cm in diameter and 1 mm thick with a beveled edge. The edges of the tissue are held down by small wedges of wet cotton that allow the tissue to assume...
its natural unstretched dimensions. The preparation is kept moist with a 37°C drip of buffered Ringer solution with 1.5% gelatin. The tissue is transilluminated with a high-pressure 100-W mercury arc. Systolic pressure is recorded in the abdominal aorta by a catheter inserted from the femoral artery.

Pressure in selected microvessels was measured directly with the servo-nulling system of Wiederhielm, Woodbury, and Rushmer (22), which was built according to the description given by Intaglietta, Pawula, and Tompkins (8).

Measurements were performed in arterioles, capillaries, and venules using glass micropipettes with sharp beveled openings in the 1- to 4-μm range.

Velocities of red blood cells were determined on line by the method of Intaglietta, Richardson, and Tompkins (9), which is based on the dual-slit technique described by Wayland and Johnson (21). The method consists of on-line measurement of the time delay in the maximum cross-correlation between the time-varying voltages generated by the images of the streaming red blood cells projected onto two adjacent photodetectors aligned in the direction of the flow.

The data-reduction system has a frequency response of 1 Hz. Therefore, in order to recover the pulsatile components due to the action of the heart, the output of the photodetectors and the central blood pressure were recorded on analog magnetic tape at 60 inches/s and played back at a slower speed in such a fashion that the components of at least double the heart rate of the experimental animal (120–180 beats/min) could be processed by the system.

Pulsatile velocity wave forms buried in noise were recovered with a second data-reduction process (9) performed on the on-line computed velocity that was recorded on magnetic tape simultaneously with the systemic arterial pressure. The arterial pressure recording was then used to generate a series of pulses synchronous with the action of the heart. Cross-correlation of this pulse train with the computed velocity eliminates frequency components not present in the systemic pressure and provides a noise-free output. This output is further filtered in such a fashion that only the fundamental of the reference signal is preserved. The absolute value of this phase is not significant, but the relative change measured as the microcirculation is examined at each level and is directly related to the mechanical properties of the system.

The method of actively filtering data virtually eliminates random noise and periodic components not related to the quality reference signal, such as respiration and vasomotion. Information on these phenomena could be obtained by providing the correlation computer with a reference signal at the appropriate characteristic frequency.

ANALYTICAL METHOD

The analytical approach that is used is based on the network model of Intaglietta, Richardson, and Tompkins (9) and is extended to include the unsteady fluid mechanics in the microcirculation and the geometrical data at each level of branching characteristic of this microvascular network.

In this model, the microvasculature is postulated to consist of a number of levels connected in series. Each level contains a set of tubes of equal diameter connected in parallel. The model is shown in Fig. 1.

The fluid mechanics of the microcirculation have been described in detail in a review by Gross and Arroyo (6). Flow in the microvessels is characterized by low values for both the Reynolds number and the Womersley frequency parameter. The Reynolds number is the ratio of the inertial forces in the fluid to the viscous forces and is defined as \( Re = \frac{r_0 U}{\nu} \), where \( U \) is the velocity of the flow (cm/s), \( r_0 \) is the characteristic dimension of the vessel, i.e., the internal radius (cm), and \( \nu \) is the kinematic viscosity of the fluid (cm²/s). When \( Re \ll 1 \), inertial forces are negligible and viscous forces predominate. The Womersley frequency parameter is given by \( \alpha = r_0 \sqrt{\omega/\nu} \), where \( \omega \) is the characteristic frequency of the system. The following assumptions are made in this model: 1) Blood is an incompressible, Newtonian fluid. 2) Equations of motion are linearized. 3) Vessel wall is cylindrical, uniform, impermeable, and infinitely long. 4) Vessel wall is elastic, thick walled, and displaces in radial direction only. 5) Static forces balance on vessel wall. 6) Effects due to bifurcations are assumed negligible. On the basis of these assumptions, an equation for the flow in a tube can be obtained by balancing the pressure gradient along the vessel with the shear forces and momentum changes in the blood:

\[
\frac{\partial P}{\partial x} = -RQ - \rho \frac{\partial Q}{\partial t}
\]

Pressure Shear Momentum gradient change

where

\[
R = \frac{8 \mu}{\pi r_0^4}
\]

\( P = \) intravascular hydrostatic pressure, dynes/cm²
\( \mu = \) viscosity of blood, poise
\( \rho = \) density of blood, g/cm³
\( r_0 = \) radius of vessel, cm
\( Q = \) volume flow of blood, cm³/s
\( x = \) axial dimension, cm
\( t = \) time, s

In this study the viscosity of blood is assumed to be 2 cP. This value is somewhat lower than that measured by whole blood in viscometers in order to account for the reduced

![FIG. 1. Network model of microcirculation.](https://aiiteg.org/)

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forces. Hematocrits usually seen in the microcirculation. (See also the review by Gross and Aroesty (6).)

It is useful to compare the structure of the governing flow equations in terms of the Womersley frequency parameter that was defined earlier. If inertial forces dominate the system, as they do in flow in the large arteries where \( r_0 \) is large and therefore \( \alpha \gg 1 \), then the pressure gradient is balanced by the momentum change of the blood. In this case, the coefficient \( \rho/\pi r_0^2 \) characterizes the dynamic behavior of the system. On the other hand, in the microcirculation, where radii are small, \( \alpha \ll 1 \), viscous forces are predominant, and the pressure gradient is balanced by shear forces.

The dependence of the volumetric flow rate \( Q \) on the axial distance, \( x \), is determined by relating the change of flow rate along the tube to the velocity of the fluid, \( v_r \), normal to the vessel wall:

\[
\frac{\partial Q}{\partial x} = -2\pi r_0 v_r
\]

The \( v_r \) can result from two effects: 1) movement of the vessel wall due to the elastic properties of the wall, and 2) actual fluid movement across the vessel wall due to fluid exchange. If \( v_r \) is related to the movement of the vessel wall \( (\text{assumption 3}) \), then Patel et al. (16) have shown that:

\[
\frac{\partial Q}{\partial x} = \frac{C}{\omega} \frac{\partial P}{\partial t}
\]

for which Pollack, Reddy, and Noordergraaf (17) defined the compliance \( C \) as

\[
C = \frac{3S(a + 1)^2}{E(2a + 1)} l
\]

where

- \( S \) = cross-sectional area of the vessel,
- \( E \) = Young's modulus of the vessel,
- \( a \) = ratio of vessel radius to wall thickness,
- \( l \) = length of vessel.

Differentiating Eq 1 with respect to \( x \) for the case of the microcirculation \( (\text{negligible inertial forces}) \) and combining with Eq 4, we obtain for each level of branching:

\[
\frac{\partial^2 P}{\partial x^2} = R_n C_n \frac{\partial P}{\partial t}
\]

which is the "diffusion" equation for pulsatile pressure in the microcirculation. According to this formulation, pressure at each level is governed by diffusion, and the "diffusion coefficient" \( 1/R_n C_n \) is determined by the properties of the system given by Eq 2 and Eq 5.

The solution of this equation is in the form of

\[
P = P_0 e^{i\omega(t-x/\omega)} e^{-kx}
\]

where

- \( c \) = phase velocity or displacement speed of the wave,
- \( x \) = physical separation of the waves,
- \( k \) = attenuation constant,
- \( P_0 \) = initial amplitude.

The wave speed is a function of the frequency:

\[
c = \sqrt{\frac{4 \mu C_n}{\pi r_0^5 \omega}} = \sqrt{\frac{R_n C_n}{2\omega}}
\]

The attenuation constant is given by:

\[
k = \sqrt{\frac{2\omega R_n C_n}{2}}
\]

where \( C_n \) = wall compliance, which can be comparatively large. Hence, pulsatile flow in the microvessels is highly attenuated and dispersive, which has been shown in in vivo measurements (9).

The pulsatile characteristics of flow in the microcirculation were determined by assuming that the microvasculature can be compared to the hydraulic network shown in Fig. 1, where the behavior of the pressure at each level is given by Eq 6. The procedure is carried out by computer and consists of solving Eq 6 for the first level using \( R_1 \) and \( C_1 \), which are calculated from the known geometrical data (12). The values for \( E \) utilized in this study were \( 10^5 \) dynes/cm² for the midcapillaries, \( 5 \times 10^6 \) dynes/cm² for the intermediate levels, and \( 10^6 \) dynes/cm² for arteries and veins (see Fung (3)). The exit values of the pressure \( P \) and flow \( Q \) are used as inputs to the channels of the next level. These inputs are then solved using \( R_n \) and \( C_n \), and the process is repeated until the last level is reached. In this way it is possible to obtain values of \( P \) and \( Q \) at each level of the microcirculation and to compare them with experimental data. It is assumed that flows are equally distributed at each bifurcation. All of the calculations are based on an initial phase angle of zero for the pressure at the entrance to the omentum. The initial phase angles for the flow are determined by the solutions to the equations defining the network model.

The rheological behavior of blood in the microcirculation is very complex and ranges from a two-phase flow in the capillaries to a Casson behavior in the largest arterioles. Capillary flows in the microcirculation have been described in detail in the review by Gross and Aroesty (6). Since in vivo data are not available, the present study assumes that flows are Newtonian.

**EXPERIMENTAL RESULTS**

Capillary pressures and flows in the rabbit omentum were found to be pulsatile throughout the microvasculature of this tissue. The frequency of the periodic fluctuations was of the order of 1 Hz, which corresponds to the heart rate of these experimental animals. Periodic fluctuations due to the cardiac cycle were more pronounced on the arterial end of the system, whereas those due to respiration were primarily in evidence at the venous end.

Pressure and flow measurements in the rabbit omentum are given in Table 1. They are grouped according to the order of branching classification proposed by Intaglietta and Zweifach (12) for the rabbit omentum and are expanded to include small arteries of the order of 50-μm diam and venules in the 75-μm range. Measurements were obtained from 12 capillary beds. Pulsatile effects in comparatively large microvessels (arteries and arterioles from 60 to 12 μm diam, and venules and veins) were primarily deter-
TABLE 1. Pressure and velocity measurements in microvasculature of the rabbit omentum

<table>
<thead>
<tr>
<th>Vessel</th>
<th>Level</th>
<th>No. of Measurements*</th>
<th>Diameter, μm</th>
<th>Pressure, cm H2O</th>
<th>Pressure Amplitude, cm H2O</th>
<th>Pressure Phase, deg</th>
<th>RBC Velocity, cm/s</th>
<th>Velocity Amplitude, cm/s</th>
<th>Velocity Phase, deg</th>
</tr>
</thead>
<tbody>
<tr>
<td>Arteries</td>
<td>1</td>
<td>7P-2V</td>
<td>52.</td>
<td>5.8</td>
<td>93.</td>
<td>11.</td>
<td>14.0</td>
<td>5.4</td>
<td>68</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>3.9</td>
<td>0</td>
<td>0.15</td>
</tr>
<tr>
<td>Arterioles</td>
<td>2</td>
<td>5P-2V</td>
<td>20.</td>
<td>4.6</td>
<td>44.</td>
<td>7.4</td>
<td>8.4</td>
<td>4.0</td>
<td>81</td>
</tr>
<tr>
<td>Art. cap. I</td>
<td>3</td>
<td>8P-3V</td>
<td>13.6</td>
<td>2.3</td>
<td>38.</td>
<td>6.9</td>
<td>3.5</td>
<td>3.0</td>
<td>92</td>
</tr>
<tr>
<td>Art. cap. II</td>
<td>4</td>
<td>2P-3V</td>
<td>11.2</td>
<td>2.8</td>
<td>28.</td>
<td>1.2</td>
<td>90</td>
<td>0.28</td>
<td>0.06</td>
</tr>
<tr>
<td>Mid. cap.</td>
<td>5</td>
<td>4P-3V</td>
<td>9.5</td>
<td>0.4</td>
<td>25.</td>
<td>4.0</td>
<td>1.2</td>
<td>1.0</td>
<td>90</td>
</tr>
<tr>
<td>Ven. cap. I</td>
<td>6</td>
<td>IV</td>
<td>8.0</td>
<td></td>
<td></td>
<td></td>
<td>0.35</td>
<td>0.16</td>
<td>0.02</td>
</tr>
<tr>
<td>Ven. cap. II</td>
<td>7</td>
<td>4P 5V</td>
<td>11.1</td>
<td>1.1</td>
<td>20.</td>
<td>4.0</td>
<td>1.0</td>
<td>1.0</td>
<td>90</td>
</tr>
<tr>
<td>Venule</td>
<td>8</td>
<td>1P-7V</td>
<td>23.5</td>
<td>4.2</td>
<td>18.</td>
<td>2.0†</td>
<td>100</td>
<td>0.67</td>
<td>0.28</td>
</tr>
<tr>
<td>Vein</td>
<td>9</td>
<td>5P-IV</td>
<td>72.0</td>
<td>12.6</td>
<td>16.</td>
<td>3.1</td>
<td>0.4</td>
<td>0.5</td>
<td>143</td>
</tr>
</tbody>
</table>

* Designation P and V refers to number of pressure and velocity measurements performed. † This value is not statistically significant.
§ Phase of maximum amplitude in recorded waveform relative to maximum amplitude in systemic arterial pressure. Note that pressure and flow phase angles change in parallel. ¶ Phase of fundamental component.

discussed by direct pressure measurements. Conversely, pulsatile effects in capillaries were determined by velocity measurements.

The comparatively large standard deviation in phase angle relative to the fundamental in the recording of pressure in the abdominal aorta is probably due to the variability in heart rate between experimental animals and to the uncertainty of the exact location of the catheter tip relative to the mesenteric artery.

DISCUSSION

Relation between pulsatile flow and microvascular properties. The persistence of pulsatile conditions provides a tool for the study of the effects of the compliance, resistance, and heart rate on the nonsteady flow parameters in the microcirculation.

The dependence of the pressure amplitude and the pressure and flow phase angles on the heart rate were calculated for multiples and submultiples of the fundamental frequency (the heart rate). The results are summarized in Figs. 2–4. Figure 2 shows that the attenuation of the pressure wave through the bed is not strongly dependent on the frequency. However, a distinct trend exists and as frequency increases attenuation or filtering in the arterial portion of the network is comparatively greater than that at the venous end. This condition explains in part why the experimental data show a substantial amplitude drop in the first three levels of branching. Considering that the arterial pressure pulse contains as many as six harmonics, the arterial microcirculation, acting as a low-pass filter, attenuates high frequencies, and the resulting waveform is uniformly attenuated by the rest of the network.

The corresponding numerical results obtained for the pressure amplitude at each level of branching are plotted in Fig. 2 by normalizing relative to the value at the first order or branching. It can be seen that qualitative agreements exist with the experimental results. It should be noted, however, that perfect agreement cannot be expected because the experimentally measured pulse shape is the additive result of the fundamental and higher harmonics.

The pressure phase angle (see Fig. 3) shows a strong dependence on the heart rate. Direct pressure phase comparison with experimental data is not made because the phase corresponding to the pressure fundamental was not available. The flow phase lag (see Fig. 4), which is directly proportional to ω, exhibits a comparatively large change for the higher harmonics. The flow phase dependence on frequency shows most of the phase shift occurring in the small artery and in the postcapillary section of the bed. Almost no phase shift occurs at the capillary level. Although the model predicts a comparatively different dependence of the pressure and flow angles as a function of heart rate throughout the network, it should be noted that these parameters are almost identical at the venous end of the bed (Figs. 3 and 4).

The flow phase angle for the first order of branching of the experimental data was determined by estimating the difference in phase between the recorded velocity wave forms and the time derivative of the pressure wave forms. This form of comparison is appropriate since, according to Eq 1, the flow is proportional to the pressure gradient ∂P/∂x, and this is related to the pressure-time derivative:

\[ \frac{\partial P}{\partial x} \cdot \frac{1}{c} \frac{dx}{dt} \cdot \frac{\partial P}{\partial x} \cdot \frac{1}{c} \frac{dt}{d} \]

(10)
PULSATILE MICROHEMODYNAMICS

where \( c \) — phase velocity defined in Eq 8. The initial angle thus estimated was determined to be approximately 45°.

The behavior of an omental microcirculatory bed as a function of different structural properties of the bed was calculated for the following four cases:

i) The length and, hence, the resistance and compliance of the vein are increased by 102. This makes it possible to assess the importance of the venous resistance distal to the bed.

ii) The stiffness of the capillaries is increased by an order of magnitude in order to establish which flow property varies most with the compliance of the capillaries.

iii) The effect of a vasodilator is simulated by increasing the diameter of all vessels (except the capillaries) by 20%. This simulates the effect of a vasodilator.

iv) The effect of a vasoconstrictor is simulated by decreasing the diameter of all vessels (except the capillaries).

The capillary diameter is assumed not to be affected by pharmacological elements. These are gross oversimplifications of the actual in vivo effects of these agents, since it is well known that they do not act uniformly throughout the microvasculature.

by 20%. Each of these cases is always referred back to the "normal" case discussed previously.

The variations of the pressure amplitude through the bed for the above cases are given in Fig. 5. It is clear that the maximum variation for any case from the normal is about 5%, and this occurs in the arterial side. No noticeable change occurs in the venous end of the bed. The increase of distal venous resistance and decrease of capillary compliance have essentially no effect on the attenuation of the pressure amplitude through the bed.

The vasodilation effect reduces arterial resistance and increases compliance; hence, there is less attenuation of the pressure amplitude in the arterial portion of the bed than in the normal case. However, as pointed out previously, the maximum deviation from the normal case is not large (5%). Vasoconstriction yields an increased resistance over that for the normal case, as would be expected.

The differences in the four cases for the pressure phase shift are significantly greater than for the pressure amplitude (see Fig. 6). Lengthening the vein (case i) has a strong effect on the pressure phase shift, which results from the fact that the transit distance of the pressure wave is increased, and consequently more phase shift can develop. This could be a relatively sensitive measurement for establishing the role of distal venous properties in the microcirculatory bed.

Changing the compliance of the capillaries has almost no measurable effect on the distribution of pressure phase shift. Substantial changes in pressure phase shift occur for cases iii and iv and in the directions that would be expected. Vasodilation reduces the pressure phase shift by about 8° at the end of the bed. Conversely, vasoconstriction increases phase shift by 16°.

The flow phase shift shown in Fig. 7 is more difficult to interpret. Very little quantitative change occurs for the four cases on the arterial side of the bed. On the venous side, the final phase angles are similar to those for the pressure phase shift. It appears, then, that the pressure phase shift is a comparatively more sensitive measurement parameter for the determination of the behavior and properties of the microcirculatory bed.
on the existence of a velocity component that is lateral (radial) to the motion of blood through the vasculature. In specifying the nature of this component in Eq 3, it was stated that this could also be due to exchange normal to the vessel wall. This possibility has been suggested by Intaglietta, Richardson, and Tompkins (9) in order to explain the comparatively large phase shifts measured in the omentum of the cat, which imply a comparatively compliant microcirculation. If this were the case, visible diameter changes with the heart rate should be directly noticeable by intravital microscopy. These changes were not observed by visual inspection and were found by Intaglietta and Tompkins (10) by means of data processing of television microscopy recordings of microvessel diameters to be in the submicron range, which corresponds to a relatively stiff . . . microcirculation.

LEVEL IN MICROCIRCULATORY BED

FIG. 6. Variation of intraluminal pressure phase angle for the different physiological conditions given in Fig. 5.

FIG. 7. Variation of flow phase angle for different physiological conditions in Fig. 5.

The changes shown by the model are comparatively small as a consequence of the diffusionlike behavior of the time-dependent effects in this model.

From a theoretical viewpoint, this type of approach is justified because of the substantial disparity between viscous and momentum effects. From experimental evidence, it is seen that pressure amplitudes follow the theoretical prediction, thus lending support to a diffusionlike effect in this system. Whether the comparative insensitivity to parameter variation found in the model will be reflected in similar experimental data remains to be seen. It is possible, however, that since microcirculatory networks appear to be designed to maintain a specific steady state related to the nutritional and homeostatic needs of the tissue, even in the presence of substantial input and output changes, the AC or pulsatile properties of the model also reflect this situation.

Compliance due to exchange. The analysis leading to the formulation of a diffusionlike equation for the propagation of periodic pressure waves in the microcirculation is based

\[ v_r^* = \frac{K[P(t) - p]}{\omega R c^3} \]  

(11)

where \( K \) = Landis-Zweifach (23) capillary filtration coefficient and \( p \) = term that combines all colloid osmotic and tissue hydrostatic pressures and is assumed here to be a constant.

Differentiating Eq 1 with respect to \( x \) for the case of negligible momentum change and combining with Eq 3 and Eq 11, we obtain

\[ \frac{\partial^2 P}{\partial x^2} + \frac{\pi_L K R p}{\pi_v K R p} - \frac{\pi_L K R p}{\pi_v K R p} = 0 \]  

(12)

which describes pressure diffusion in the microcirculation due to the exchange across the semipermeable vessels of the microcirculation.

The comparative importance of the two causes for the diffusionlike behavior of pulsatile effects in the microcirculation can be determined by evaluating the magnitude of the "diffusion coefficients" that characterize the time-dependent effects. This special form of the diffusion coefficient for the case of predominance of mechanical compliance effects \( D_{\text{MECH}} \) is taken from ref. 1:

\[ D_{\text{MECH}} = \frac{1}{\omega R c^3} \]  

(13)

where \( \omega \) appears in taking the time derivative that is necessary in order to obtain a form equivalent to Eq 7.

Combining Eq 13 with Eq 2 and Eq 5, where the term \((a + 1)^2/(2a + 1)\) is assumed to equal 2, we obtain:

\[ D_{\text{MECH}} = \frac{r_v^2 E}{48 \mu K l} \]  

(14)

Conversely, the diffusion coefficient for the case of predominance of the exchange effects, \( D_{\text{EXCH}} \), from Eq 12 and Eq 2 is

\[ D_{\text{EXCH}} = \frac{r_o^3}{16 \mu K l} \]  

(15)

and the ratio between the coefficients becomes

\[ \frac{D_{\text{MECH}}}{D_{\text{EXCH}}} = \frac{3E K}{\omega c} \]  

(16)
Estimates on the values of $E$ and $K$ for the capillaries of the rabbit omentum are $E = 3 \times 10^7$ dynes/cm$^2$ (3) and $K = 10^{-9}$ cm$^3$/dynes (23). The value of $\omega$ is assumed to be of the order of $2/s$ and $r_0 = 5 \times 10^{-4}$ cm. The value of the ratio expressed in Eq. 16 is of the order of 1 for this microvascular network, which represents the scaling between pressure diffusion and exchange diffusion of pulsatile effects.

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REFERENCES


