Flow and pressure distributions in vascular networks consisting of distensible vessels

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Departments of 1Mathematics, Statistics, and Computer Science and 2Biomedical Engineering, Marquette University, Milwaukee 53201-1881; 3Research Service, Zablocki Veterans Affairs Medical Center, Milwaukee 53295; and 4Department of Physiology, Medical College of Wisconsin, Milwaukee, Wisconsin 53226

Submitted 30 August 2002; accepted in final form 20 January 2003

Krenz, Gary S., and Christopher A. Dawson. Flow and pressure distributions in vascular networks consisting of distensible vessels. Am J Physiol Heart Circ Physiol 284: H2192–H2203, 2003. First published January 30, 2003; 10.1152/ajpheart.00762.2002.—We examine the influence of vessel distensibility on the fraction of the total network flow passing through each vessel of a model vascular network. An exact computational methodology is developed yielding an analytic proof. For a class of structurally heterogeneous asymmetric vascular networks, if all the individual vessels share a common distensibility relation when the total network flow is changed, this methodology proves that each vessel will continue to receive the same fraction of the total network flow. This constant flow partitioning occurs despite a redistribution of pressures, which may result in a decrease in the diameter of one and an increase in the diameter of the other of two vessels having a common diameter at a common pressure. This theoretical observation, taken along with published experimental observations on pulmonary vessel distensibilities, suggests that vessel diameter-independent distensibility in the pulmonary vasculature may be an evolutionary adaptation for preserving the spatial distribution of pulmonary blood flow in the face of large variations in cardiac output. 

Flow partitioning; heterogeneity; mathematical models; nonlinear; pulmonary circulation

PULMONARY CAPILLARY PERFUSION and alveolar ventilation are adequately matched for efficient gas exchange over a wide range of cardiac output from rest to heavy exercise. Normally, this matching is achieved in a largely passive manner, despite the fact that the heterogeneous and asymmetric vascular geometry (10) results in a wide distribution of local flows (16, 17, 22). The pulmonary arteries are also quite distensible, as required to provide the appropriate impedance for the right ventricle output. Even though the pulmonary arterial wall structure varies considerably from the main pulmonary artery to the precapillary terminal arteries (8, 44), the distensibility, defined as the fractional change in vessel diameter per unit change in pressure, is essentially constant and independent of vessel diameter and vessel wall composition (2, 8, 28).

The same is true for the veins (1). We have observed that, in model arterial (diverging flow) or venous (converging flow) treelike structures having one common outflow or inflow pressure, respectively, common distensibility results in the fraction of the total flow passing through each vessel segment of the heterogeneous asymmetric tree being constant, regardless of the total flow or the pressure at the inlet(s). This is true, despite the fact that in such a tree the distending pressures and, therefore, the diameters of individual vessels of identical unstressed diameter may diverge substantially when the total flow or inflow pressure is changed. Depending on the functional relation between pressure and diameter, given two identical vessels located in different parts of the tree, the diameter of one may increase while the diameter of the other decreases in response to a given change in total flow; yet the ratio of flows passing through the two vessels will remain the same. Thus the flow distribution, normalized to total flow, will be the same as if the vessel walls were rigid. This perhaps counterintuitive observation led us to the conclusion that the vessel diameter-independent distensibility of the pulmonary blood vessels may be an adaptation that helps fix the pulmonary flow distribution in the face of the large variations in total pulmonary blood flow (30). This conclusion was met with some skepticism, at least in part because of the stipulation of a common terminal outlet pressure (for an arterial tree) or inlet pressure (for a venous tree). Thus, because the capillary inlet pressure distribution is not known, it is not clear to what extent this stipulation might affect the degree to which the idealized model might reflect the behavior of the real system. In the present study, we extend the theoretical analysis to the more general case of an entire vascular network diverging from a single inlet and then converging to a single outlet, with some additional observations on multiple inlet-outlet networks.

Glossary

\[ \alpha \] Distensibility parameter defined by

\[ \frac{D}{D_0} = f(P) = 1 + \alpha P \] (see Fig. 7)

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FLOW AND PRESSURE DISTRIBUTIONS IN NETWORKS

\[ L \quad \text{Length variable} \]

\[ P \quad \text{Transmural and vascular pressures, which, for simplicity, are taken as equal} \]

\[ P_i \quad \text{Pressure at node } i \]

\[ P_{in} \quad \text{Pressure at the inlet of a vessel segment} \]

\[ P_{out} \quad \text{Pressure at the outlet of a vessel segment} \]

\[ \Delta P \quad \text{Upstream-downstream pressure drop across a vessel segment: } \Delta P = P_{in} - P_{out} \]

\[ \psi(P) \quad \text{Nonlinear transformation of } P, \text{ e.g., for Poiseuille flow and } f(P) = 1 + \alpha P, \]

\[ \psi(P) = f(f(P))^4 \quad \text{dP} = f(1 + \alpha P)^4 \quad \text{dP} = \quad (1 + \alpha P)^4/(5\alpha), \quad \psi \text{ enables use of an equivalent Ohm's law for distensible vessels where upstream-downstream pressure drop for a single vessel } \]

\[ [\psi(P_{in}) - \psi(P_{out})] \text{ is directly proportional to flow through the vessel segment; constant of proportionality is resistance if vessel diameter is fixed at zero pressure} \]

\[ \psi_{n_j} \quad \text{Nonlinear transformed pressure: } \psi_{n_j} = \psi(P_{n_j}) \text{ at node } n_j \]

\[ \Delta \psi \quad \text{Nonlinear transformed upstream-downstream pressure drop for distensible vessels: } \Delta \psi = \psi(P_{in}) - \psi(P_{out}) \]

MODEL VASCULAR NETWORK

The first model vascular networks we consider are those with a single arterial inlet and a single venous outlet. To simplify the notation, intravascular pressure at a point in the network will be taken to be the same as transmural pressure at the point, which we will refer to jointly as the pressure (P). We employ the standard development for relating pressure to flow within distensible vessels (5, 9, 13, 28, 30, 36, 53).

First, consider a single distensible vessel subjected to constant, nonpulsatile flow. For ease of exposition, assume Poiseuille flow within the vessel (13). If a vessel segment is modeled as a distensible right circular cylinder and entrance effects are ignored, the local frictional pressure drop per unit length from inlet to outlet of a single vessel is represented as

\[ \frac{dP}{dL} = \frac{128\mu}{\pi D^4} F \]  \hspace{1cm} (1)

where \( P \) denotes pressure, \( L \) is vessel length, \( \mu \) is blood viscosity, \( F \) is vascular flow, and \( D \) is vessel diameter (13). The model vessels share a common diameter-pressure relation given by

\[ \frac{D}{D_0} = f(P) \]  \hspace{1cm} (2)

We refer to Eq. 2 as the vessel distensibility relation. For example, \( f(P) \), which has been discussed in the pulmonary circulation literature, includes \( f(P) = 1 + \alpha P \) and \( f(P) = b + (1 - b)e^{-\beta P} \) (1, 2, 13, 53).

Throughout the analysis, we assume that \( f(P) \) is sufficiently smooth, so when \( D = D_0f(P) \) is used in Eq.
1, the differential equation has a unique solution, 2) \( f(0) = 1 \) and \( f(P) > 0 \) for all \( P \), and 3) distensibility is constant throughout the vascular network; i.e., \( f(P) \) in Eq. 2 applies to each vessel within the vascular network. No additional restrictions are placed on \( f(P) \). Under the above assumptions (13, 30, 36) we can conclude the following.

**Lemma 1.** If \( \Psi(P) \) denotes an antiderivative of the fourth power of \( f(P) \), then

\[
\Psi(P_{in}) - \Psi(P_{out}) = r_0 F
\]

where \( P_{in} \) and \( P_{out} \) are the inlet and outlet pressures of the vessel segment, respectively, and \( r_0 \) is the vascular resistance at the zero pressure diameter \( (D_0) \).

**Remark 2.** In *Lemma 1*, individual vessel blood viscosity is part of \( r_0 \), rather than part of \( \Psi \). Thus \( \mu \) appears as a multiplicative constant separated from \( \Psi \). This separation allows us to view \( \mu \) as possibly being different from vessel to vessel, and \( \mu \) affects only the vessel’s \( r_0 \).

**Remark 3.** As pointed out elsewhere (30), *Lemma 1* is not the most general result possible, because any resistance per unit length formula that allows the separation of \( D_0 \) and \( f(P) \) could be employed, giving rise to an appropriate \( \Psi \). An example of a separable local resistance per unit length relation that describes how \( P \) changes with \( L \) would be as follows: \( dP/dL = -C\mu /D_0 f \), where \( F \) is flow in a particular vessel segment, \( D \) is vessel segment diameter, \( \gamma > 0 \) is fixed, and \( C \) and \( \mu > 0 \) are constant within an individual vessel but might change from vessel to vessel; however, they do not change with diameter or flow. With a distensibility relation, \( f(P) \), such that

\[
\frac{dP}{dL} = -\frac{C\mu}{[D_0 f(P)]^4} F
\]

has a unique solution: \( P = \phi(L) \); then, as shown elsewhere (30)

\[
\phi'(L) = -\frac{C\mu}{[D_0 f(\phi(L))]^4} F
\]

or

\[
\int_0^{L_0} [f(\phi(L))]^{-\phi'(L)} dL = \int_0^{L_0} \frac{C\mu}{D_0} F dL
\]

where \( P_{in} \) and \( P_{out} \) are the inlet and outlet pressures of the vessel segment, respectively, and \( \Psi \) and \( r_0 \) are modified from their Poiseuille flow-derived formula.

In what follows, the key observation is not dependent on whether one uses a Poiseuille flow assumption to model vascular resistance or selects an affine vs. an exponential relation between diameter and pressure to model vessel distensibility. The key observation is, instead, that \( \Psi \) should be viewed as the abstraction of pressure in a distensible vessel, rather than actual pressure \( (P) \). With this observation in mind, Eq. 3 can be viewed as a hemodynamic equivalent of Ohm’s law, which accommodates distensibility of vessel segments, relating resistance and flow to a pressure drop

\[
\Delta \Psi = \Psi(P_{in}) - \Psi(P_{out}) = r_0 F
\]

where \( r_0 \) is a fixed vessel resistance, \( F \) is flow in the vessel, and \( \Delta \Psi \) represents the nonlinear transformed pressure drop across the vessel segment. Rather than the usual vessel segment pressure drop, \( \Delta P = P_{in} - P_{out} \), this “Ohm’s law” relates the drop in nonlinear transformed pressure \( (\Delta \Psi) \) from inlet to outlet of the vessel segment to a fixed reference resistance (i.e., the resistance at the zero pressure diameter) and actual vessel flow. By ascending a single inlet-single outlet vascular network using the standard electrical circuit analogy and Eq. 6, one can write equations that calculate flow fractions and nonlinear pressures for a vascular network containing distensible vessels. Then nonlinear pressures \( (\Psi) \) can be inverted to obtain actual pressures \( (P) \).

To illustrate the computational methodology proposed above, consider the vascular network in Fig. 1. First, we examine the reference case, where reference inlet flow is 1, reference outlet pressure is 0, and reference resistances are \( r_1, \ldots, r_{22} \). For this reference setting, vessel segment \( i \) would have flow \( f_i \), \( i = 1, \ldots, 22 \).
22), and the vascular network would experience pressures \( p_2, \ldots, p_{18} \); e.g., \( p_2 \) is the pressure at node 2 and \( p_{18} \) is the pressure at node 18. Then, in the reference case, conservation of flow at node 18 in Fig. 1 would imply \( f_1 = 1 \), whereas at node 17, conservation of flow would yield \( f_1 = f_2 + f_3 \), or, equivalently, \(-f_1 + f_2 + f_3 = 0\). Similarly, at node 5, \( f_{12} + f_{13} = f_{18} \), which one should write as \( f_{12} + f_{13} - f_{18} = 0 \). At node 7, \(-f_6 + f_{12} = 0\). The upstream pressure at node 2 is \( p_2 = f_{22}r_{22} \), or \( f_{22}r_{22} = p_2 \). The two distinct pathways to node 15 would yield \( f_{10}r_{10} + p_{11} - p_{15} = 0 \), as well as, e.g., \( f_{11}r_{11} + p_{12} - p_{15} = 0 \). Overall, the 17 (conservation of) flow equations and 22 upstream pressure equations in the reference setting result in a system

\[
Ax = b
\]

where \( x = [f_1, f_2, \ldots, f_{22}, p_2, p_3, \ldots, p_{18}]' \), \( b = [1, 0, 0, \ldots, 0]' \), the symbol ‘ denotes vector transpose, and the matrix \( A \) (see APPENDIX A) captures the left-hand sides of the equations corresponding to conservation of flow and pressure calculations.

We now turn to the distensible vessel flow and pressure calculations for the vascular network depicted in Fig. 1. Lowercase subscripted variables refer to the corresponding uppercase variables denote the distensible vessel value. 

Remark 4. Because antiderivatives differ by at most an additive constant, we may select \( \Psi \) such that \( \Psi(P_{n_i}) = 0 \) at pressure \( P_{n_i} \). This is equivalent to selecting the nonlinear outlet pressure to be the zero baseline, simplifying notation and computations without affecting the generality of the results. We do not require that \( P_{n_i} = 0 \).

For any nonzero total inlet flow \( (F) \), the flow in the \( i \)th distensible vessel segment will be denoted \( F_i \). Suppose the \( i \)th distensible vessel segment is between nodes \( n_a \) and \( n_b \). We will show that 1) \( F_i = F_i(P) \) and 2) \( P_{n_i} \) is obtained from the nonlinear equation

\[
\Psi_{n_i} = \Psi(P_{n_i}) = FP_{n_i}
\]

The same can be shown for \( P_{n_j} \).

To see this, Eq. 6 is used as an equivalent Ohm’s law. The matrix equation arising from conservation of flow and nonlinear transformed pressure drop is

\[
Ay = Fb
\]

where \( y = [F_1, F_2, \ldots, F_{22}, \Psi_2, \Psi_3, \ldots, \Psi_{18}]' \). The matrix \( A \) is exactly the matrix obtained in the reference calculation. Uniqueness of the solution of Eq. 7 implies that the system of equations that determines the flows \( (F_i) \) and nonlinear pressures \( (\Psi_{n_i}) \) is equivalent to

\[
A(Fx) = (Fb)
\]

where \( y = Fx \) are related through \( F_i = F_i(P) \) (i = 1, \ldots, 22) and \( \Psi_{nj} \) (\( n_j = 2, \ldots, 18 \)) are given by Eq. 8.

With this example of calculation methodology in mind, we can now state the main result. Suppose each vessel segment in a vascular network is assigned a unique number \( i \).

**Theorem 5.** Suppose that, in an arbitrary single inlet-single outlet vascular network, 1) every vessel segment has the same distensibility relation, \( D/D_0 = f(P) \) (each vessel segment, however, may have a different \( D_0 \)), 2) \( \mu \), although it may be different in each vessel segment, remains constant within a vessel segment as flow or diameter changes, and 3) up to a multiplicative constant, every vessel segment has the same separable local resistance per unit length relation. Then, for each vessel segment \( i \) in the vascular network, there exists a unique constant \( f_i \), independent of \( F \), such that

\[
\frac{F_i}{F} = f_i
\]

relating the flow in the vessel \( (F_i) \) to the nonzero total inlet flow \( (F) \).

This result follows directly from the calculation methodology using conservation of flow and nonlinear pressure \( (\Psi) \) and relating the reference calculations to the distensible vessel calculations.

**Corollary 6.** Under the same suppositions as for theorem 5, if \( F_a \) denotes the flow in one daughter vessel at a bifurcation and \( F_L \) denotes the flow in the other daughter vessel, then \( F_a/F_L \) is the same for every nonzero total inlet flow \( (F) \) through the vascular network.

To provide a concrete numerical example, we employ an \( f(P) \) that exhibits autoregulatory-like behavior to the vascular network in Fig. 1. This example was chosen, despite the focus of the introduction on the pulmonary circulation in which passive mechanics dominates, because the example tends to be a rather severe challenge to one’s intuition. In a network experiencing autoregulatory behavior, it is easy to see that, for two vessels having the same \( D_0 \), a change in total flow can result in an increase in diameter of one and a decrease in diameter of the other, but perhaps not so obvious is that the fraction of flow through each vessel will remain the same. One such autoregulatory distensibility relation is \( f(P) = a_1 + b_1P^{c_1}e^{e_1P} + (1 - a_1)e^{-e_1P} \), where \( a_1 = 0.9, b_1 = 0.25, b_2 = 1, c_1 = 0.25 \), and \( c_2 = 0.25 \) (30). A graph of \( f(P) \) vs. \( P \) is given in Fig. 2. This \( f(P) \) provides a maximum diameter of 131% \( D_0 \) but falls to only 90% \( D_0 \) as pressure increases.

The autoregulatory \( f(P) \) was applied to the 22-vessel vascular network in Fig. 1, where vessels in the network are numbered \( i = 1, \ldots, 22 \) and nodes are denoted \( n_1, \ldots, n_{18} \), and vascular network outlet pressure \( (P_{n_i}) \) was set to zero. Employing the vessel-numbering scheme, \( r_i \) denotes the reference resistance that would result at the diameter corresponding to zero pressure in vessel segment \( i \). With the assumption of Poiseuille flow, \( \Psi(P) \) would be an antiderivative of the fourth power of \( f(P) \). Figure 3 is one such antiderivative, with the appropriate additive constant such that \( \Psi(0) = 0 \). For the vascular network in Fig. 1, we set

\[
AJP-Heart Circ Physiol • VOL 284 • JUNE 2003 • www.ajpheart.org
\]
r_1 = 1/8, r_2 = r_21 = 1/2, r_3 = r_4 = r_5 = r_18 = r_19 = r_20 = 1, r_6 = r_8 = r_10 = r_12 = r_13 = r_15 = r_16 = r_17 = 2, r_7 = r_9 = r_11 = r_14 = 3, and r_22 = 1/16. Particular reference resistances would correspond to a known vessel geometry at zero pressure. However, because flow division depends solely on the reference resistances, it is sufficient to select the resistances.

Solving the reference case Eq. 7 and numerically inverting the nonlinear expression Eq. 8 provide the pressures \( P_n \) at the nodes of Fig. 1. The pressures, as a function of total vascular network flow, are given in Fig. 4. At any fixed total vascular network flow, a vertical line would cut through the various pressure curves, which, along with the zero outlet pressure, would depict the complete nodal pressure distribution in the network. At a total network flow of 20, the pressure at node 3 is \( P_3 = 4.90 \), at node 4 the pressure is \( P_4 = 2.82 \), and, in regard to further increases in flow and, therefore, pressures, vessel 1 has become a rigid tube with diameter \( \sim 90\% \) of \( D_0 \). In Fig. 5, the pressure drop across each individual vessel in Fig. 1 is plotted as a function of individual vessel flow. If the vessels were rigid, each plot would be a straight line. Although each vessel has the same distensibility relation, the nonlinear interaction of distensibility, pressure, and vascular network connective structure is readily apparent in the nonlinear appearance of the curves and, more importantly, in the differences in concavities of the curves, and yet every vessel segment in the vascular network is receiving a constant fraction of the total vascular network flow over the entire range of total network flow.

To demonstrate the alternative condition, i.e., the effect of changing total flow on individual vessel flows in networks with varying vessel distensibility, we carried out the simulations depicted in Fig. 6. The col-
Fig. 6. Hemodynamic calculations for network topology in Fig. 1 using distensibility relation $D/D_0 = 1 + \alpha P$. Each column represents a different choice of $\alpha$: $\alpha$ increases with decreasing vessel size (left), $\alpha$ is the same for all vessels (middle), and $\alpha$ decreases with decreasing vessel size (right). From top to bottom: pressure at 18 nodes as a function of total network flow, vessel segment flow as a function of total network flow, fraction of total flow in a vessel segment as a function of total network flow, and pressure drop across a vessel segment as a function of vessel segment flow. Negative values indicate retrograde flow.

DISCUSSION

The primary observation of this study is that if a heterogeneous asymmetric vascular network (having the stated properties) consists of blood vessels, each of which has the same distensibility relation, despite potentially wide variations in pressures within vessels that have a common diameter at a given pressure, the
flow distribution within the network will be unaffected by changes in total network flow and the accompanying redistribution of pressures. A similar observation was made previously for diverging (arterial) and converging (venous) trees, with the restriction that there was a common outlet or inlet pressure, respectively. Because there is little available experimental information on capillary pressure distributions (24), it is unknown how damaging this restriction might be to the relevance of the theorem to any real vascular system. Extension to the single inlet-single outlet network helps address this question to the extent that an arterial (or venous) tree can now be considered a part of a network for which there is somewhere downstream (or upstream) a common pressure and in which distributed arterial outlet or venous inlet pressure would be the normal condition for a heterogeneous asymmetric network.

As indicated in the introduction, this study was motivated by an attempt to understand the significance of observations indicating that the distensibility of the pulmonary arteries (Fig. 7) and veins (1) is virtually independent of vessel size. The network model does not completely resolve the question, because the specific assumption invoked is that the arteries, capillaries, and veins have the same distensibility relation. Over the physiological range of pulmonary pressures, that assumption appears to be reasonable for the pulmonary arteries and veins (1, 2, 26). Whether the same can be said for the capillaries is not so clear; in part, it depends on whether the capillaries are viewed as cylinders (21, 47), which distend with a uniform increase in diameter, or as a punctuated sheet, wherein distension is only orthogonal to the alveolar surface (13, 14, 47), or somewhere between these extremes. For a cylindrical capillary, the geometric component of the resistance would involve the fifth power of the diameter, as in the cylindrical arteries and veins, whereas the sheet resistance involves the fourth power of the dimension orthogonal to the alveolar surface (13). On the other hand, the available data suggest that the capillary distensibility is at least within the same order of magnitude as the arteries and veins, with values of capillary distensibility (defined as the fractional change in the vessel dimension orthogonal to the alveolar surface per unit change in pressure), obtainable from the literature, ranging from ~0.023/mmHg for the dog lung (40) to ~0.07/mmHg for cat and dog lungs (13, 15).

Cox (8) was apparently the first to point out that the mechanical properties of the pulmonary arterial vessel walls are essentially independent of vessel diameter and the composition of the individual vessel walls defined by relative amounts of connective tissue and smooth muscle. This is reiterated by the compilation of data in Fig. 7, updated from the findings reported by al-Tinawi et al. (2), wherein the vessel diameter independence of the distensibility coefficient $\alpha$ is reflected by the fact that the data from several studies obtained using various methods can be correlated by a virtually constant value of $\alpha$ over several orders of magnitude in $D_0$ from the main pulmonary artery to terminal arterioles and represent many more orders of magnitude in individual vessel segment resistances. Despite the diameter independence, there is, in fact, variability in the individual values within a given diameter range, even between studies on the same species. The reasons for this are not clear but may reflect sensitivity to some aspect(s) of study conditions that has not been systematically identified. Thus it seems probable that the variability within a given diameter range in Fig. 7 is greater than would be expected within any particular diameter range.
individual lung. However, the objective of the analysis is not to provide an argument that the distensibility is constant. Rather, it points out that limits on the distribution of individual vessel distensibilities would be a logical result of evolutionary pressure to maintain gas exchange efficiency (i.e., the ventilation-perfusion distribution) over a wide range of cardiac output.

Determination of the impact of the various obvious differences between the model and the real system (e.g., pulsatile flow and gravity effects) will probably require numerical simulations beyond the scope of present study. Thus, even having generalized the model to encompass an entire network, it remains idealized. This allows for the analytic approach to understanding the model behavior, and we believe that the observations provide a reference point for understanding the implications of vascular network design in a sense similar to other idealizations, including “Poiseuille’s law,” “sheet flow,” the “fifth power law” (13, 53), “Murray’s law” (32), and others (31, 36, 43, 49).

It may also be useful to reiterate that the theorem presented here is not dependent on the assumption of Poiseuille flow. Rather, in the deviation of Eq. 9, the existence of a $\frac{\partial}{\partial \theta}$ and the reference $r_0$ is what is needed, where the reference $r_0$ might be thought of as the resistance that would exist if the vessel diameters were fixed at their zero pressure values. This is accomplished if, up to a multiplicative constant, each vessel in the vascular network has the same local normalized resistance per unit length expression. Although the results allow for blood viscosity being different in each vessel, the restriction of constant viscosity within a vessel may be viewed as more limiting. However, changes in viscosity within any single vessel segment due to physiologically reasonable flow or diameter changes are small (29).

Although our primary goal was to examine the potential for fixed flow partitioning within a heterogeneous asymmetric vascular network, the methodology can be employed to determine flows within multiple inlet-multiple outlet vascular networks where each vessel experiences the same distensibility relation (see APPENDIX B). It is further clear that reference flow distribution calculations apply to the distensible vessel case under any of the following conditions: 1) a multiple inlet-single outlet vascular network, where the inlet flows may increase or decrease, but inlet flows are delivered in a fixed ratio, 2) a single inlet-multiple outlet vascular network, where all outlet pressures are fixed at the same value, and 3) a multiple inlet-multiple outlet vascular network, where the inlet flows may increase or decrease but the inlet flows are delivered in a fixed ratio and all outlet pressures are fixed at the same value. In each case, the individual vessel segment flows throughout the network would follow the constant partitioning results described above.

The observation that distensibility of the pulmonary arteries and veins is diameter independent over several orders of magnitude in vessel diameter may reflect a design feature that takes advantage of the observations described above. A structure with vessels sharing a common distensibility should result in a stabilizing effect on the impact of changing cardiac output on the pulmonary capillary flow distribution without requiring an elaborate controlling mechanism (28). When the distensibility is not constant throughout the network, in particular, when it is diameter dependent, the fraction of total flow within any one branch of the network may diverge from the initial flow distribution, and even reversal of flow in some segments is possible (Fig. 6). Some observations of the effects of changing cardiac output on the pulmonary flow distribution (4, 23, 46) appear to be generally consistent with a nearly constant flow distribution.

These observations may have implications for the function of diseased lungs that are somewhat analogous to the effect of the distribution of airway mechanics on the breathing frequency dependence of the distribution of ventilation. That is, in diseased lungs having an abnormally broad distribution of time constants among respiratory units, the increase in breathing frequency generally accompanying an increase in total ventilation results in a redistribution of the fraction of the total ventilation received by a given respiratory unit (41). Likewise, increasing cardiac output in a lung with a disease extended distribution in individual vessel distensibilities and would tend to result in a redistribution of blood flow. Little information is available regarding any changes in the longitudinal or parallel distributions of distensibilities of vessels that might occur as the result of pulmonary vascular remodeling in pulmonary diseases.

APPENDIX A

The connective structure of the single inlet-single outlet vascular network and the explicit choice of the separable local resistance per unit length relation determines the matrix $A$ in Eq. 7. For the example vascular network in Fig. 1, $A$ is given by

$$A = \begin{bmatrix} A_{1,1} & A_{1,2} \\ A_{2,1} & A_{2,2} \end{bmatrix}$$

where
APPENDIX B

Provided that sufficient and appropriate boundary conditions are given so that they uniquely specify the reference calculation, a linear system of equations can be created to determine the resulting flows and pressure throughout the vascular network that contains distensible vessels. For example, consider the vascular network in Fig. 8, where each vessel has the same distensibility relation \( f(P) \) and, up to a multiplicative constant, each vessel has the same separable local resistance per unit length relation, which gives rise to some matrices.

\[
\begin{pmatrix}
A_{2,1} = \begin{bmatrix}
 f_1 & f_2 & f_3 & f_4 & f_5 & f_6 & f_7 & f_8 & f_9 & f_{10} & f_{11} & f_{12} & f_{13} & f_{14} & f_{15} & f_{16} & f_{17} & f_{18} & f_{19} & f_{20} \\
 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & r_{18} & 0 & 0 \\
 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & r_{19} & 0 & 0 \\
 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & r_{14} & 0 & 0 \\
 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & r_{17} & 0 & 0 \\
 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & r_{12} & 0 & 0 \\
 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & r_{13} & 0 & 0 \\
 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & r_{15} & 0 & 0 \\
 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & r_{16} & 0 & 0 \\
 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & r_6 \\
 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & r_7 \\
 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & r_8 \\
 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & r_9 \\
 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & r_{10} \\
 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & r_{11} \\
 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & r_4 \\
 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & r_5 \\
 0 & r_2 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
 r_1 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
\end{bmatrix}
\end{pmatrix}
\]

and

\[
\begin{pmatrix}
A_{2,2} = \begin{bmatrix}
f_{21} & f_{22} & p_2 & p_3 & p_4 & p_5 & p_6 & p_7 & p_8 & p_9 & p_{10} & p_{11} & p_{12} & p_{13} & p_{14} & p_{15} & p_{16} & p_{17} & p_{18} \\
 0 & 0 & 0 & 1 & 0 & 0 & 0 & -1 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
 0 & 0 & 0 & 1 & 0 & 0 & 0 & -1 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
 0 & 0 & 0 & 1 & 0 & 0 & 0 & -1 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
 0 & 0 & 0 & 1 & 0 & 0 & 0 & -1 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
 0 & 0 & 0 & 0 & 1 & 0 & 0 & 0 & -1 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
 0 & 0 & 0 & 0 & 0 & 1 & 0 & 0 & 0 & -1 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
 0 & 0 & 0 & 0 & 0 & 0 & 1 & 0 & 0 & 0 & -1 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
 0 & 0 & 0 & 0 & 0 & 0 & 0 & 1 & 0 & 0 & 0 & -1 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 1 & 0 & 0 & 0 & -1 & 0 & 0 & 0 & 0 & 0 & 0 \\
 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 1 & 0 & 0 & 0 & -1 & 0 & 0 & 0 & 0 & 0 \\
 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 1 & 0 & 0 & 0 & -1 & 0 & 0 & 0 & 0 \\
 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 1 & 0 & 0 & 0 & -1 & 0 & 0 & 0 \\
 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 1 & 0 & 0 & 0 & -1 & 0 & 0 \\
 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 1 & 0 & 0 & 0 & -1 & 0 \\
 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 1 & 0 & 0 & 0 & 0 \\
 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 1 & 0 & 0 & 0 \\
 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 1 & 0 & 0 \\
 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 1 & 0 \\
 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 1 \\
 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
\end{bmatrix}
\end{pmatrix}
\]
We denote the direction of individual vessel flow in Fig. 8 to be from a higher node number to a lower node number; e.g., the flow in vessel 4, F_4, is considered directed from n_3 to n_2. Furthermore, suppose we set pressures at n_1, n_4, and n_6 such that \( \psi(0) \) is the smallest of the terminal pressures. We define \( \Delta \psi_j = \psi(n_j) - \psi(n_{j-1}) \) for \( j = 1, \ldots, 6 \). Conservation of flow and calculation of nonlinear pressures, using Eq. 6, gives:

1) \( F_1 = F_2 + F_3 \)
2) \( F_4 = F_5 + F_6 \)
3) \( F_5 = F_2 + F_4 \)
4) \( \Delta \psi_1 = 0 \)
5) \( \Delta \psi_2 - \Delta \psi_3 = F_2 + F_5 \)
6) \( \Delta \psi_3 - \Delta \psi_4 = F_4 \)
7) \( \Delta \psi_4 = V_3 \)
8) \( \Delta \psi_5 - \Delta \psi_6 = F_6 \)
9) \( \Delta \psi_6 = V_2 \)
10) \( \Delta \psi_1 - \Delta \psi_6 = F_1 r_1 \)
11) \( \Delta \psi_1 - \Delta \psi_2 = V_1 \)

which can be summarized as a system of linear equations:

\[
Ay = b \quad \text{with} \quad A = \begin{bmatrix} a_1 & \cdots & a_6 \end{bmatrix}, \quad y = \begin{bmatrix} y_1 & \cdots & y_6 \end{bmatrix}, \quad b = \begin{bmatrix} 0 & 0 & 0 & 0 & 0 & 0 \end{bmatrix}^T, \quad V_1, V_2, \ldots, V_6 \]

where \( V_1 \) and \( V_2 \) denote known values from the vascular network pressure boundary conditions, and \( r_i \) denotes fixed reference resistances. Although the flows can repartition as pressures are changed, flow calculation, as well as the nonlinear pressures \( \Delta \psi_i \), is a linear problem. Nonlinearity comes into play only if one needs to invert \( \Delta \psi_i \) to obtain the actual pressures. This example also illustrates the necessary modifications if one does not select \( \psi \) to have a zero baseline.

In this setting, if, say, \( V_2 \) is always a fixed constant multiple of \( V_1 \), then each vessel in the network in Fig. 8 will experience a fixed fraction of the total network flow.

This research was supported by the National Heart, Lung, and Blood Institute Grant HL-19298 and the Department of Veterans Affairs.

REFERENCES


