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A Simple Model for the Arterial System

Hans-Uno Bengtsson and Patrik Edén,

Department of Theoretical Physics, Lund University,
Sölvegatan 14A,
S-223 62 Lund, Sweden

Abstract
We present a simple model for the arterial part of the cardiovascular system, based on Poiseuille flow constrained by the power dissipated into the cells lining the vessels. This, together with the assumption of a volume-filling network, leads to correct predictions for the evolution of vessel radii, vessel lengths and blood pressure in the human arterial system. The model can also be used to find exponents for allometric scaling, and gives good agreement with data on mammals.

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Introduction

In all but miniscule living organisms, branching networks are responsible for the transport of gases and nutrition. Extra interest in nutrition networks emerged from the suggestion (Dawson, 1991; West *et al.*, 1997) that the quasi-fractal structure could be the key to understanding allometric scaling, *i.e.,* the observation that a biological quantity Q of an organism scales as the body mass M to some power b, $Q \propto M^b$. As the mass is related to volume, all quantities related to length, area or volume are expected to scale as the mass to an integer number of thirds. Frequently, however, data exhibit significant deviations from these exponents (Calder, 1984; Schmidt-Nielsen, 1984; Peters, 1983). For example, capillary density scales with an exponent between -0.2 and 0 (Schmidt-Nielsen & Pennycuick, 1961; Hoppeler *et al.*, 1981), and though the question is not fully settled (Dodds *et al.*, 2001), it appears that rest metabolism scales with an exponent close to 3/4, rather than the expected 2/3.

Though Dodds *et al.* (2001) present severe criticism of the West *et al.* model, which they find to rely on partly incorrect math, the central hypothesis - that fractal nutrition transport networks affect allometric scaling exponents - remains intriguing and worth further investigation. Inspired by the approach of West *et al.* (1997), we present a simple model which correctly predicts the development of radii, lengths and pressure in the human arterial system. Related allometric scalings for aorta properties are in agreement with data, as is the scaling of capillary density.

The Model

The Network

The arterial network begins with one vessel (the aorta) and splits into many smaller vessels through subsequent branchings. We say that a vessel belongs to level k if there are k branches between the vessel and aorta. (Thus, aorta itself is the only vessel at level 0.)

In our model, each vessel is characterized by four relevant parameters: a radius R, a length L, an average speed of flow U, and a pressure difference ΔP between its ends. The pulsatile nature of the flow necessarily implies that U, ΔP and to some extent R, vary with time. However, as will be seen later, our results do not depend on the detailed properties of the oscillations. In the following, we will therefore let U, ΔP and R refer to time-averaged quantities.

The distributions of vessel radii, vessel lengths, etc., at different levels k and in the arterial system as a whole, is a field of research in its own right (Zamir, 2001), which can be approached using computer simulations of stochastic processes with physiological constraints (Schreiner & Buxbaum, 1996). In this paper we focus, however, on average properties of the network, and for our purposes, a simple and more transparent model will suffice. We therefore assume that each level k is characterized by a typical radius R_k, a typical length L_k, a typical speed of flow U_k, and a typical pressure fall ΔP_k, and assign these values to all vessels at level k. The four

typical values, together with N_k, the number of vessels at level k, fully characterize the level. Finally, we assume that there is a typical number of branches, c, from the aorta to a capillary, and assign all capillaries to a terminal level c.

For the calculations below, it is convenient to introduce the branching ratio

$$n_k = \frac{N_k}{N_{k-1}}, \tag{1}$$

and the branchings parameters

$$u_k = \frac{U_k}{U_{k-1}}, \quad r_k = \frac{R_k}{R_{k-1}}, \quad l_k = \frac{L_k}{L_{k-1}}, \quad \delta_k = \frac{\Delta P_k}{\Delta P_{k-1}}. \tag{2}$$

It is not uncommon to set all $n_k = 2$, *i.e.,* to assume all branchings to be bifurcations (Zamir, 2001; Schreiner & Buxbaum, 1996), and let trifurcations be represented by two bifurcations with a very short connecting segment. We present our model with general numbers n_k, as this will demonstrate that few of the results depend on their value.

Branching Parameter Relations

Making four assumptions, we can express the branching parameters in terms of the branching ratio. The first three assumptions deal exclusively with the flow itself, and are independent of the actual structure of the network; furthermore, they are all local assumptions. Only the forth assumption addresses the structure of the network, a global assumption.

Assumption one is that the total volume flow is conserved, *i.e.,* the same on any level;

$$N_k \cdot \pi R_k^2 U_k = \text{constant}, \tag{3}$$

or

$$n_k r_k^2 u_k = 1. \tag{4}$$

In other words, we consider the body under reasonably invariant conditions, during wich no part of the cardiovascular network systematically accumulates blood for a long time. Thus, we consider time-scales such that oscillations of blood volumes are irrelevant.

Assumption two is that the flow through a vessel on any level is given by Hagen–Poiseuille's law,

$$\pi R_k^2 U_k \propto \frac{\Delta P_k R_k^4}{L_k}, \tag{5}$$

or

$$\frac{\delta_k r_k^2}{l_k u_k} = 1. \tag{6}$$

This assumption is based on treating blood as a simple, incompressible fluid. Furthermore, we expect possible local turbulence, in the vicinity of branchings, to have negligible effect on the pressure fall over a whole vessel, and we neglect possible variations in viscosity. We note that, for time-averaged quantities U_k and ΔP_k, Hagen–Poiseuille's law applies, also when the full flow is pulsatile (Womersley, 1955*a,b*).

Assumption three is that the power dissipated per unit mantle area is a constant,

$$\frac{N_k \cdot \pi R_k^2 U_k \Delta P_k}{N_k \cdot 2\pi R_k L_k} = \text{constant}, \tag{7}$$

or

$$\frac{\delta_k r_k u_k}{l_k} = 1. \tag{8}$$

A quick order-of-magnitude estimate shows why the power dissipated per unit mantle area is an important quantity. The human rest metabolism of 100 W corresponds to a specific power of roughly 1 W/kg. Assuming this specific power not to be drastically different in the body cells and the body fluids, we find, using a typical cell size of 10^{-5} m and a typical density of 10^3 kg/m^3, a load of 10^{-12} W or 1 pW per cell, which can be taken as a typical value. A flow causing a considerably higher load on the cells lining the mantle is presumably not very well adapted to the workings of the body. (Of course, during strenuous exercise, the power, and thus the load, can increase by a factor 10 or so.) On the other hand, there is no reason for the flow to be restricted in order to give a load appreciably lower than this.

Now, the number of capillaries in the human body is of the order of 10^{10}, the average speed of flow through the capillaries is of the order of 10^{-3} m/s and the radius of the capillaries is of the order of 10^{-5} m. The pressure drop over the capillaries is roughly 20 mm Hg or $3{\cdot}10^3$ N/m^2, giving a power of the order of 10 W. The length of a capillary being of the order of 10^{-3} m, this power is dissipated over a mantle area of the order of 10^3 m^2, representing in all approximately 10^{13} cells; thus the power dissipated per unit mantle area translates into a load on each cell of the order of 1 pW, the typical value of the load.

From the above three assumptions we immediately find

$$u_k = n_k^{-1/5}, \tag{9}$$

$$r_k = n_k^{-2/5}, \tag{10}$$

and

$$\frac{\delta_k}{l_k} = n_k^{3/5}, \tag{11}$$

irrespective of the structure of the network.

Assumption four, pertaining to the overall structure of the network, allows some leeway. A common and reasonable assumption is that the network is volume-filling, *i.e.,* $N_k \cdot L_k^3 =$ constant. This assumption gives $l_k = n_k^{-1/3}$ and $\delta_k = n_k^{4/15}$. On the other hand, our discussion above of power per cell as a limiting factor shows the mantle area to be of importance; an alternative assumption might then be conservation of mantle area, *i.e.,* $N_k{\cdot}2\pi R_k L_k =$ constant, leading to $l_k = n_k^{-3/5}$ and $\delta_k = 1$. Obviously, neither alternative is true for the first few levels; however, both of them are *a priori* reasonable descriptions of possible network structures on the deeper levels, *i.e.,* for large k.

In the following, we will actually assume a more general network structure defined by

$$N_k \cdot R_k^{D_R} L_k^{D_L} = \text{constant}, \tag{12}$$

or

$$n_k r_k^{D_R} l_k^{D_L} = 1, \tag{13}$$

giving

$$l_k = n_k^{-\frac{1-\frac{2}{5}D_R}{D_L}} \equiv n_k^{-\Gamma} \tag{14}$$

and

$$\delta_k = n_k^{\frac{3}{5}-\Gamma}, \tag{15}$$

and express our results in terms of the parameter Γ.*

Metabolism Preview

Before we continue, let us have a quick look at the consequences for the metabolism assuming a self-similar network, *i.e.,* constant branching ratio and branching parameters. The total arterial blood volume V is given by

$$V = \sum_{k=0}^{c} n^k \cdot \pi R_k^2 L_k = \pi R_0^2 L_0 \cdot \sum_{k=0}^{c} (nr^2 l)^k = (r^2 l)^{-c} \cdot \pi R_c^2 L_c \cdot \frac{1-(nr^2 l)^{c+1}}{1-nr^2 l}. \tag{16}$$

Now, from the calculated scaling properties of the network, $nr^2 l = n^{\frac{1}{5}-\Gamma}$, which means that for $\Gamma > 1/5$, the term $(nr^2 l)^{c+1}$ disappears in the large c limit. Making the further assumption that properties on the deepest (capillary) level, c, are independent of body mass M, and using the observation that total blood volume V is proportional to body mass, we find

$$M \propto V \propto \frac{1}{1-n^{\frac{1}{5}-\Gamma}} n^{c(\frac{4}{5}+\Gamma)}, \tag{17}$$

or

$$N_c = n^c \propto M^{\frac{1}{\frac{4}{5}+\Gamma}}. \tag{18}$$

Finally, the assumption that metabolic rate B is proportional to the volume flow (which is constant on all levels) yields

$$B \propto \pi R_0^2 U_0 = N_c \cdot \pi R_c^2 U_c \propto M^{\frac{1}{\frac{4}{5}+\Gamma}}. \tag{19}$$

*One possibility to determine the parameter Γ would be to demand that the total impedance for the network, $\propto \sum_k L_k/N_k R_k^4$, be minimal, subject to the constraint of a fixed total arterial blood volume, $\propto \sum_k N_k R_k^2 L_k$ (West *et al.*, 1997). Substituting for N_k from equation (12) and using the method of Lagrange multipliers, we are then to minimize the function $F(\{R_k\},\{L_k\}) = \sum_k (R_k^{-4+D_R} L_k^{1+D_L} + \lambda R_k^{2-D_R} L_k^{1-D_L})$. However, the set of equations $0 = \partial F/\partial R_k$ and $0 = \partial F/\partial L_k$ have solutions only if $D_R + D_L = 3$. Thus, impedance minimization is not robust against even small fluctuations of the total dimensionality, which are to be expected for a real, biological system. We will therefore not add impedance minimization to our list of assumptions.

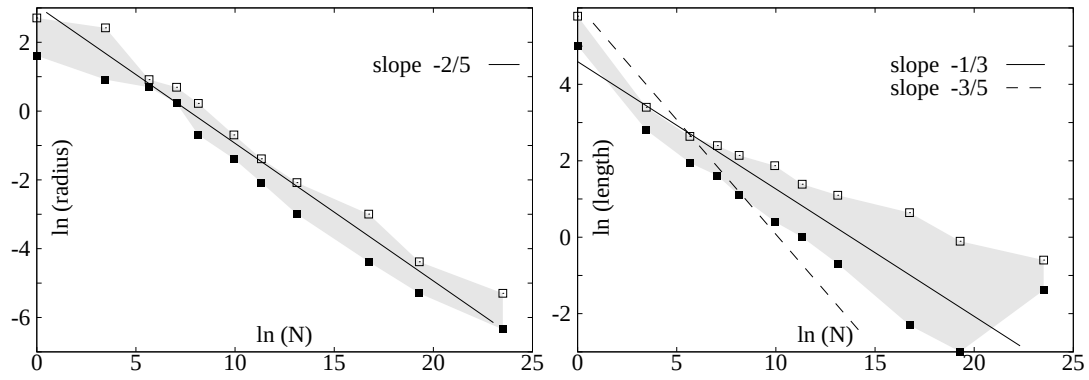

Figure 1: *Logarithm of radius and length, respectively, as a function of the logarithm of number of vessels, at various levels in the human arterial system. The shaded areas cover the regions between lower range values (filled boxes) and upper range values (open boxes), as given in Schneck (2000). The straight lines represent model predictions.*

The case $\Gamma = 1/3$ (volume-filling network) gives a metabolism proportional to $M^{15/17}$; the case $\Gamma = 3/5$ (mantle area-preserving network) gives a metabolism proportional to $M^{5/7}$. The exponent 5/7 lies comfortably within the reported range for rest metabolism; 15/17 is definitely on the high side. However, in the next section we show that $\Gamma = 1/3$ is supported by data on the human arterial system. Furthermore, 15/17 is a good exponent for the scaling of the number of capillaries, N_c. The different scaling exponents for N_c and B are further discussed below.

Results

In our model for blood transport networks, each quantity Q has local, level-specific values Q_k and branching parameters $q_k = Q_k/Q_{k-1} = n_k^{\Upsilon}$, where n_k is the branching ratio at level k and Υ is an exponent specific for Q. In this section we confront the model with data, both on the human arterial system and on allometric scaling.

Human Arterial System

The exponent Υ of the branching parameter is given by the slope of $\ln(Q_k)$ as a function of $\ln(N_k)$;

$$\Upsilon = \frac{\ln(q_k)}{\ln(n_k)} = \frac{\ln(Q_k) - \ln(Q_{k-1})}{\ln(N_k) - \ln(N_{k-1})}. \tag{20}$$

In Schneck (2000), the typical number of vessels are tabulated, together with ranges of radii and lengths, for several levels of the human vascular system. The data on the arterial system are illustrated in Fig. 1. Straight lines with slopes predicted by our model are inserted, to guide the eye.

The model prediction $r_k = n_k^{-2/5}$ agrees remarkably well with data, even close to the heart. This suggests that our neglect of the dampening of pulsatile flow does not introduce any appreciable errors. This is also supported by some simple phenomenology. Over the human arterial system, the amplitude of pressure oscillations drops from 20 mm Hg to almost nothing, while the average pressure drops from 100 mm Hg to 40 mm Hg, a considerably larger reduction.

The data on vessel lengths determine the parameter Γ. It should be noted that data on aorta length (at $k = 0$) refer to total aortic length, not average uninterrupted distance between branch origins, as is the case for the other levels. Thus, the apparent fit to the slope $-3/5$ at early levels is just an artefact of mixing different vessel length definitions, and we can conclude that the volume-filling network (slope $-1/3$) is favoured by data on vessel lengths. Furthermore, as discussed below in connection to allometric scaling, data on pressure fall support $\delta_k = n_k^{4/15}$, related to $\Gamma = 1/3$, and disfavour $\delta_k = 1$, related to $\Gamma = 3/5$.

Allometric Scaling

To be able to make predictions on allometric scaling, we add the assumption that capillary properties are fixed by cell properties, and hence are body mass independent. This implies that all scalings can be parameterized in terms of the number of capillaries, N_c.

The relation for quantity Q between level 0 (aorta) and level c (capillaries), is simply

$$Q_c = \left[\prod_{k=1}^{c} q_k\right] Q_0 = N_c^{\Upsilon} Q_0. \tag{21}$$

For some quantities, *e.g.,* pressure and total blood volume, the global, cumulative quantity $Q_\Sigma = \sum_k Q_k$ is of interest. The expression for Q_Σ greatly simplifies if we assume self-similarity, $n_k = n$. We then get

$$Q_\Sigma = Q_0 \sum_{k=0}^{c} q^k = Q_0 \frac{1 - q^{c+1}}{1 - q} = \frac{Q_0 - q Q_c}{1 - q}. \tag{22}$$

In general, there are enough levels c for $q \neq 1$ to imply that one of the terms Q_0 or qQ_c dominates. Thus, Q_Σ scales either as its aorta value or as its capillary value. (If $q_k = 1$, as for flow, we have $Q_\Sigma = Q_0(c+1) = Q_c(c+1)$ and we can let convenience govern our choice of associating Q_Σ with aorta or capillary level.)

Cumulative variables Q_Σ can be associated with aorta or capillary level even if the self-similarity assumption is relaxed. If $q_k < 1$ and $n_k = \bar{n}$ for sufficiently many initial levels $k \leq \bar{k}$, we get $Q_\Sigma = (Q_0 - \bar{q} Q_{\bar{k}})/(1 - \bar{q}) \sim Q_0$. Similarly, if $q_k > 1$ and sufficiently many final levels $k \geq \tilde{k}$ have $n_k = \tilde{n}$, we get $Q_\Sigma \sim Q_c$.

Applying the expressions for Q_0 and Q_Σ to our model, together with the reasonable constraint $1/5 < \Gamma < 3/5$, implies

$$\text{Aorta radius, } R_0 \quad \propto \quad N_c^{2/5}, \tag{23}$$

$$\text{Aorta length, } L_0 \quad \propto \quad N_c^{\Gamma}, \tag{24}$$

$$\text{Arterial blood volume, } V \;\propto\; N_c^{\Gamma+4/5}, \tag{25}$$
$$\text{Aorta mean pressure, } P_0 \;=\; \text{constant}, \tag{26}$$

and

$$\text{Volume flow, } \Phi \;\propto\; N_c. \tag{27}$$

Here we have omitted all factors of capillary properties Q_c, assumed to be body mass invariant. We have chosen to present results for observables that can be independently measured. The performance of the model on these observables can be easily transferred to indirectly measured quantities, *e.g.,* average circulation time, $\propto V/\Phi$, and aorta mean blood velocity, $U_0 \propto \Phi/R_0^2$. Some frequently discussed observables, *e.g.,* heart frequency and oxygen affinity, are beyond the scope of our simple model.

We note that the time-averaged aorta pressure, P_0, is predicted constant by any model with $\delta_k \geq 1$. More discriminating is the relation between the total arterial pressure fall, ΔP_Σ, and the pressure fall over the capillaries, ΔP_c. For humans, the average aorta pressure is about 100 mm Hg and the pressure in the capillary ends are roughly 40 mm Hg and 20 mm Hg, respectively. Thus, $\Delta P_\Sigma/\Delta P_c \approx$ (80 mm Hg)/(20 mm Hg) = 4. This disfavours models where $\delta_k \approx 1$, since they predict $\Delta P_\Sigma/\Delta P_c \approx c = \ln(N_c)/\ln(c)$, which is ≈ 35 for $n = 2$ and ≈ 20 for $n = 3$. Instead, we find support for self-similar networks with $\delta \approx 4/3$, since these models give $\Delta P_\Sigma/\Delta P_c \approx \delta/(\delta - 1)$. Our model with $\delta = n^{4/15}$ gives $\delta \approx 1.20$ for $n = 2$, and $\delta \approx 1.34$ for $n = 3$, resulting in the predictions $\Delta P_\Sigma/\Delta P_c \approx 6$ and 4, respectively. Both these numbers are in reasonbale agreement with data, considering the rough estimates involved in the calculation. This is the only result here presented that depends on the actual value of the branching number n.

Data show that the total blood volume is proportional to the body mass (Stahl, 1967; Prothero 1980). If this holds also for the arterial blood volume, our model predicts allometric scaling exponents as in Table 1. These agree well with data, except for cardiac output Φ. However, as pointed out by Dodds *et al.*, in a resting body, blood does not flow through all capillaries. The total number of capillaries is probably better associated with maximal blood flow, which is indeed thought to scale with an exponent closer to unity (Bishop, 1999).

Thus, blood flow in a resting body, and the rest metabolism, are beyond the scope of simple models of this kind. However, we conclude that our model gives correct answers to the questions it actually addresses. We find good agreement with data on capillary density and with data on vessel radii, vessel lengths and blood pressure in the human arterial system. The allometric scaling of corresponding aorta quantities also agrees well with data, suggesting that the model is applicable to all mammals.

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Variable	Model ($\Gamma = \frac{1}{3}$)	Data
Blood volume V	$\equiv 1$	1.02 (Stahl, 1967), 0.99 (Prothero 1980)
Capillary density N_c/M	$-\frac{2}{17} = -0.12$	-0.14 (Schmidt-N. & Pennycuick, 1961), -0.21 to -0.07 (Hoppeler *et al.*, 1981)
Aorta radius R_0	$\frac{6}{17} = 0.35$	0.41 (Clark, 1927), 0.36 (Holt *et al.*, 1981)
Aorta length L_0	$\frac{5}{17} = 0.29$	0.32 (Holt *et al.*, 1981)
Aorta pressure P_0	0	≈ 0 (Schmidt-Nielsen, 1984; Calder, 1984)
Volume flow Φ	$\frac{15}{17} = 0.88$	0.81 (Stahl, 1967), 0.79 (Holt *et al.*, 1968), 0.75 (White *et al.*, 1986)

Table 1: *Comparison of model predictions and data on allometric scaling exponents. The exponent for blood volume is fixed to 1, and the other exponents are model predictions. Data are from the compilations in Calder (1984) and Schmidt-Nielsen (1984).*

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