Med Biol Eng Comput (2009) 47:143–151
DOI 10.1007/s11517-008-0413-0

SPECIAL ISSUE - ORIGINAL ARTICLE

Input impedance of distributed arterial structures as used in investigations of underlying concepts in arterial haemodynamics

Alberto Avolio

Received: 21 May 2008 / Accepted: 1 October 2008 / Published online: 24 October 2008

Abstract By definition, vascular impedance is described in the frequency domain as the ratio of sinusoidal functions of pressure and flow, yielding spectral values of impedance modulus and phase. The impedance spectrum is determined by the structure and physical properties of the vascular system, such that for a given system the relation between pressure and flow can be modified by alteration of the geometric or mechanical properties of the vascular segments. Whereas input impedance of an arterial system can be readily determined by simultaneous measurement of just two time varying signals of blood pressure and flow, the production of the same impedance spectrum from the physical properties of the system would require information of inordinate complexity and magnitude. Hence, arterial models with a tractable number of parameters or explicit mathematical description are used to approximate the physiological impedance of a vascular structure, which in all animal species consists of distributed branching arterial networks. Although models are a necessary approximation, the strong similarity between the impedance spectra of models and physiological arterial systems enables investigations of fundamental concepts. This is illustrated by examining the effect of the branching structure on the decoupling of the high peripheral resistance from the ejecting ventricles and how physical parameters derived from the impedance spectrum can be used to investigate concepts of optimal design and features related to body size across a broad range of animal species.

A. Avolio (✉)
Australian School of Advanced Medicine, Macquarie University, North Ryde, Sydney, NSW 2109, Australia
e-mail: alberto.avolio@mq.edu.au

Keywords Vascular impedance · Multibranched models · Arterial haemodynamics · Allometric relations

1 Introduction

Arterial impedance, determined as the ratio of the frequency components of blood pressure and flow, essentially considers the arterial bed as a single entity (as in the 'black box' analogy). However, interpretation of the impedance frequency spectrum is based on the conceptualization of the system being investigated, which can take the form of basic lumped parameter models to complex networks of branching structures of elastic tubes structures [23, 26]. Since the early descriptions of relations of blood pressure and flow in the circulation, investigations involved the use of models for characterization of physiological parameters. The most simple, a purely resistive model, can provide useful information on the effect of physiological changes in the microcirculation affecting total peripheral resistance by mean arterial pressure and cardiac output. The classic windkessel model includes total arterial compliance defined by the elastic properties of arteries and the inclusion of an induction term accounts for the blood inertia [26]. None of these lumped parameter models gives a full description of the physiological impedance as determined by pressure and flow. However, each describes a certain part of the spectrum; the resistive model is the zero frequency value and the Windkessel describes the falling impedance modulus at low frequencies [22, 30, 31]. The limitation of lumped parameter models is in the absence of spatial parameters such as arterial length so as to include the finite speed of wave propagation. In some configurations of hydraulic arterial models as used in loading ejecting ventricles [46], the Windkessel is modified to include an impedance term which

represents characteristic impedance, being essentially the constant impedance at high frequencies of the impedance spectrum. The inclusion of length allows modeling of distributed systems. This is done by using transmission line analogies by assigning equivalent resistance, capacitance and inductance values per unit length, and arterial segments can be described as a series and parallel network of uniform tubes [3, 35, 43, 44, 47].

There have been numerous distributed models that have been described involving both linear and nonlinear representations of arterial properties [3, 6, 13, 35, 36, 38–44], all showing overall patterns of impedance which resemble physiological systems. However, what is of interest is not merely a reconstruction of the physiological system using models, either by physical components or in terms of mathematical relationships, but how the specific models can be used to uncover fundamental aspects of arterial haemodynamics that are not apparent by simple analysis of pressure and flow signals [30, 31]. Models that take into account nonlinearities in formulations of vascular impedance show that nonlinear effects are relatively small [40] and so linear models are generally a good representation.

This paper examines how the characterization of the arterial system in terms of input impedance of distributed arterial models is used to study fundamental phenomena in arterial haemodynamics. These include the effect of the branching structure of elastic tubes on the decoupling of the high peripheral resistance from the ejecting ventricles and also how physical parameters derived from the impedance spectrum can be used to investigate concepts of optimal design and features related to body size across a broad range of animal species.

2 Vascular impedance

Arterial vascular beds can be characterized either by determination of a global parameter, such as input impedance, describing the opposition to flow, and so defined by the relationship of arterial blood pressure and flow, or by defining the sub-units of the system, such as arterial segments, in terms of their physical properties so that the impedance of each sub-unit can be described by equations derived from the solution of fluid flow in closed boundaries. In general terms, the first method is used for calculation of impedance from physiological measurements and the second relates to construction of arterial models.

2.1 Impedance calculated from pressure and flow waves

Impedance is defined in the frequency domain and relates oscillatory components of the time-varying signals of pressure $P(t)$ and flow $Q(t)$. Assuming that $P(t)$ and $Q(t)$ are periodic functions and the system is stationary and in steady state oscillation, the harmonic components, $P_k(\omega)$ and $Q_k(\omega)$ are obtained by Fourier decomposition of $P(t)$ and $Q(t)$ and the impedance spectrum is calculated as

$$Z_k(\omega) = \frac{P_k(\omega)}{Q_k(\omega)}; \quad (k = 1, 2, \ldots, N) \tag{1}$$

This is a complex quantity with modulus ($|Z|_k(\omega)$) and phase ($\mathrm{Zphs}_k(\omega)$) plotted as function of frequency for the N harmonics. For physiological signals, the impedance spectrum can be completely specified for $N = 10$. However, since impedance is a ratio, small magnitudes of $P_k(\omega)$ and $Q_k(\omega)$ can give erroneous results of both modulus and phase. Hence, to eliminate the effect of noise, a threshold would need to be set to not consider those frequency components below the assigned value. This is equivalent to a filtering procedure and eliminates spurious impedance calculations. In practice, a number of cardiac cycles are analysed to obtain a mean value for both modulus and phase.

2.2 Impedance calculated from arterial properties

Womersley's solution of the fluid motion equations in cylindrical conduits resulted in closed form equations describing the derivation of oscillatory flow velocity from pressure gradient and dimensions of the tube and properties of the fluid [49]. This facilitated both the calculation of flow velocity profiles along the cross-section of the tube and produced a closed form expression for the longitudinal impedance (i.e. impedance per unit length Z_L) as a function of the oscillatory non-dimensional parameter (α) and a series of Bessel functions. For a propagating system, where the real wave speed (c_0) is determined by the properties of the elastic tube (elastic modulus E, radius R, wall thickness h, and fluid density ρ; $c_0^2 = Eh/\rho 2R$) from the electrical transmission line analogy, the relationships between wave velocity, frequency (ω) and Z_L result is a closed form expression for characteristic impedance (Z_0) as [23]

$$Z_0 = \frac{\rho \cdot c_0}{(1-\sigma^2)^{1/2}} \frac{1}{(M'_{10})^{1/2}} \exp(-\mathrm{i}\varepsilon_{10}^2) \tag{2}$$

where according to Womerley's nomenclature, $\alpha = \sqrt{(R\omega/\nu)}$; ν is the kinematic viscosity of blood; $M'_{10}\exp(-\mathrm{i}\varepsilon_{10}) = 1 - F_{10}$ and $F_{10} = 2J1(\alpha \mathrm{i}^{3/2})/\alpha \mathrm{i}^{3/2} J0(\alpha \mathrm{i}^{3/2})$; where $J0$ and $J1$ are Bessel functions of order 0 and 1, respectively, and $\mathrm{i} = \sqrt{-1}$; ω is the frequency. σ is the Poisson's ratio of the artery wall.

Similarly, a propagation coefficient (γ) is defined as

$$\gamma = \mathrm{i}\omega \frac{(1 - F_{10})^{-1/2}}{c_0} \tag{3}$$

From Eqs. 2 and 3, an expression is obtained for input impedance (Z_{in}) of a uniform tube of length L as

$$Z_{\mathrm{in}} = \frac{Z_0(1 + \Gamma \exp(-2\gamma L))}{(1 - \Gamma \exp(-2\gamma L))} \tag{4}$$

where the reflection coefficient (Γ) is defined in terms of Z_0 and the terminal impedance (Z_{T}) as

$$\Gamma = \frac{(Z_0 - Z_{\mathrm{T}})}{(Z_0 + Z_{\mathrm{T}})} \tag{5}$$

Equations 2–5 describe the formulation of the impedance for a segment of artery with known dimensions, wall elasticity and filled with blood of a known density. Thus, if a complete closed form set of equations can be used for a single tube, then similar calculations are done for tubes connected in parallel or in series. Branching structures make use of this mathematical formulation to calculate input impedances at all branch points to arrive at a value of input impedance for the entire tree [3, 13, 14, 17, 41, 42]. Impedance and propagation coefficients are calculated as complex quantities and input impedance is expressed as modulus and phase as a function of frequency, similar to the quantity derived from measurement of pressure and flow as described by Eq. 1.

3 Effect of branching and distributed arterial structures

One of the salient features of arterial beds is the tree-like structure [15]. Vessels branch with change in diameter at bifurcation points, resulting in a general reduction of diameters of branches with successive branching, but a progressive increase in total cross-sectional area. Studies in bifurcating arterial structures indicate that the area ratio is optimized such that there is minimal energy loss at junctions [15, 32, 33]. This has the effect of increasing the efficiency of transmission of pulsations to the periphery with reflections emanating predominantly from the increase in resistance of peripheral beds. Womersley [50] showed that minimal reflection coefficient is a function of the non-dimensional parameter α, and so the optimal area ratio also becomes a function of α, such that as α decreases a larger area ratio of parent to daughter branches is required for minimal reflection [23, 50]. When simplified to single tube models [23, 28], the concept of effective length to determine the distance to the functional reflecting site is determined from the frequency of the minimum of impedance modulus [7, 13, 21, 23]. This arises from the resonance concept in tubes [21, 23] where impedance minimum corresponds with a frequency of phase zero crossing, or approaching close to zero. However, these frequencies do not always coincide in vascular systems, and when the discrepancy is large it may present a source of error in the calculation of distance to a theoretical discrete reflecting site in terms of the equivalent quarter wavelength of the tube. The presence of wave reflection from peripheral beds at different distances from the input of an arterial tree has the effect of canceling the effect of positive pressure augmentation of frequency components due to the phase difference. In the arterial system, the presence of reflection is seen in the form of the impedance spectrum, where it is high at low frequencies but much lower at high frequencies and the modulus oscillates around the value of characteristic impedance [23]. This type of impedance pattern is interpreted as a device for decoupling the high peripheral impedance from the pulsating ventricles [8, 41, 42].

One of the remarkable observations in input impedance spectra of physiological arterial systems and that of models is their basic similarity (Fig. 1). Similar spectral patterns are seen in both modulus and phase where modulus decreases rapidly with frequency from a high peripheral resistance to settle around the value of characteristic impedance, and phase is initially negative and then approaches zero with increasing frequency. This general pattern is seen in any branching structure, regardless of the number of branches or terminations [3, 13, 24, 27, 29–31, 35, 41, 42]. This is an important feature, as the input impedance can therefore be used not only as a means to characterize the relationship between pulsatile pressure and flow, but also as a means to use tractable models to describe the component parameters and to evaluate their role in the production of pressure patterns throughout the arterial system.

The use of input impedance as described or measured at the aortic root is significant since it determines aortic root pressure for a given ventricular ejection, and it is this pressure which is then propagated throughout the arterial tree [14, 23, 42]. Input impedance is therefore a complete characterization of the whole arterial system. The pressure or flow signals generated by the heart are only forcing signals to excite the system and so similar impedance values would be obtained with any type of pressure or flow waveform, with the assumption that specific frequency components of pressure and flow are linearly related.

3.1 Distributed models of randomly branching systems

Many of the early distributed arterial models were constructed using physical components of resistors, capacitors and inductors to construct electrical transmission line analogs of arterial segments. The models were used

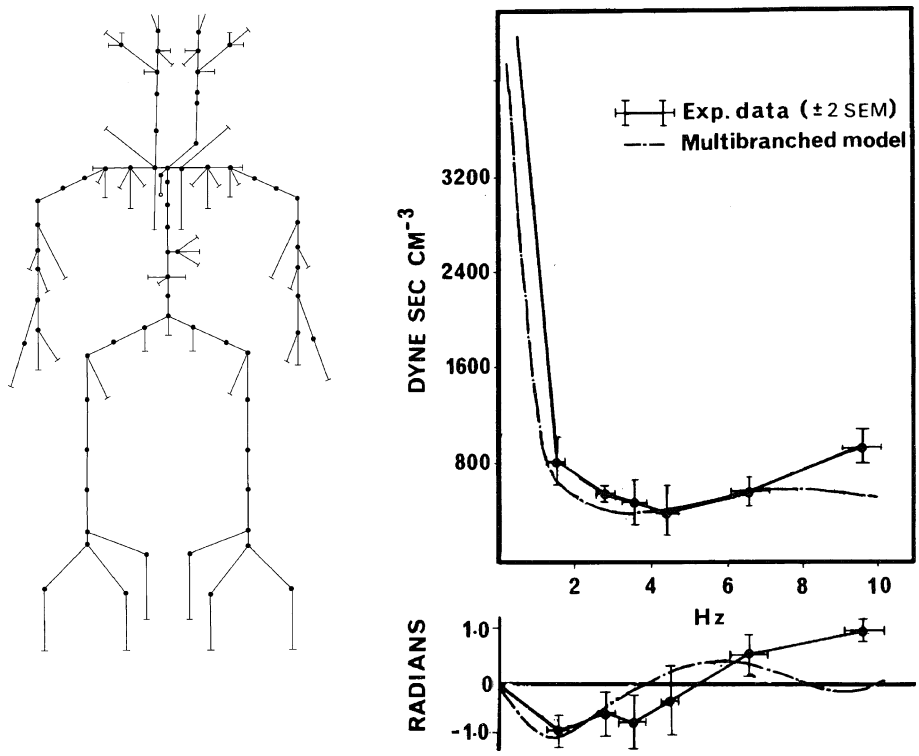

Fig. 1 *Left panel* multibranched model of the human arterial tree where each segment is represented by a uniform elastic tube using the electrical transmission line analogy and with realistic dimensions. The model contains 128 segments. *Right panel* input impedance of the model shown on the left determined from the summation of impedances of individual segments and input impedance of human subjects determined from blood pressure and flow signals at the ascending aorta. Data from [3, 27]

essentially to study ballistocardiography [47]. These were followed by mathematical representations of arterial segments such as Taylor's early classic work on arterial distributed models [41, 42]. This has resulted in an extensive body of work on arterial models, much of which is contained in texts on haemodynamics [23, 26]. The significant aspect of Taylor's models is that the branching structure was non-physiological. The branching configuration was generated by a mathematical random number algorithm. It did not consider any aspects of anatomical structure, and yet the model showed the characteristic input impedance patterns [3, 27, 41]. This implied that the important "decoupling" effect of the high peripheral resistance from the ejecting ventricle could be obtained with *any* branching pattern; the extent of the variation was determined by the specific type of branching generated (Fig. 2). Taylor showed that with a dichotomous branching structure containing branches of random lengths, the input impedance could be used to quantify the effects of anatomical parameters, such as branch dimensions, and also arterial wall properties such as viscoelasticity and wave propagation effects [41, 42]. Other later studies used the input impedance spectra in distributed models of the arterial system to include the effect of nonlinearity [35, 36, 39], confirming the early assumptions of linearity for generation of impedance patterns and pressure waveshape in the ascending aorta [23]. These studies highlighted the difference in model computation of nonlinearities related to geometric tapering or convective or elastic nonlinearities [35, 36].

3.2 Teleonomical investigations by fractal arterial models

The concept of self-similarity lends itself to spatial quantification of vascular branching structures [51]. The mathematical formulation allows conceptual investigation related to effect of branching and spatial variation of vascular and parenchymal components [5, 6, 9, 15, 16, 49]. Input impedance of a 3-dimensional fractal model of the pulmonary arterial system was used by Lefevre to investigate teleonomical aspects of arterial design [17]. The term 'teleonomy' refers to purposefulness or goal directed functions in living systems. This involves consideration of principles of adequate design and relationship between physiological structure and function [15, 32, 33]. Since the purpose of the arterial system is to distribute blood to the peripheral vascular beds, teleonomical considerations involve obtaining a specific set of arterial parameters which minimizes an energy cost function related to arterial structure and impedance to blood flow.

The cost function used by Lefevre [17] was related to efficiency and adaptivity as defined by Taylor [43] according to the following criteria:

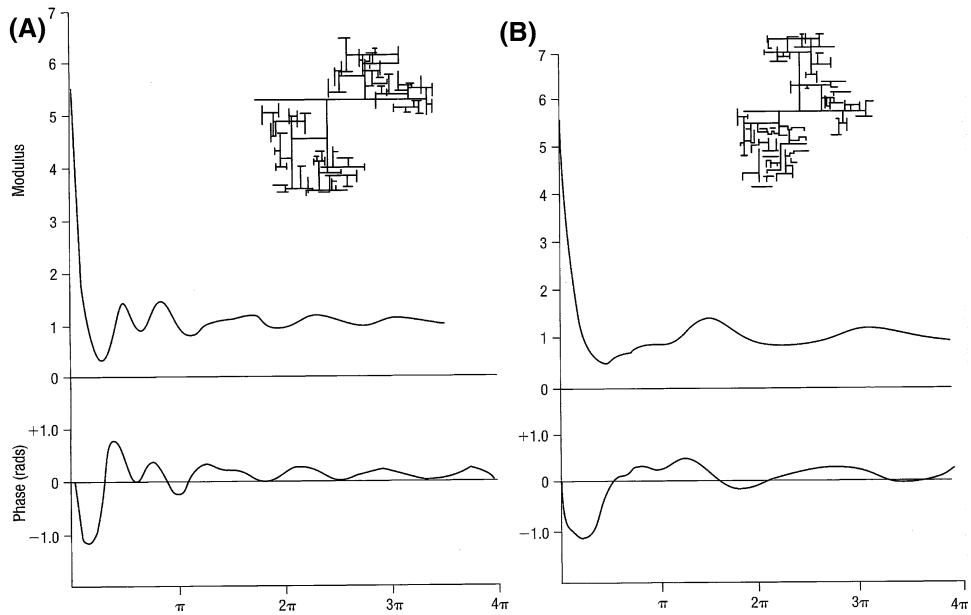

Fig. 2 Input impedance of a system of randomly bifurcating elastic tubes. Different assemblies **A** and **B** show similar patterns of impedance modulus and phase for low frequencies. However, the oscillations of the spectrum at higher frequencies is different, indicating the effect of the different branching structure. From [39]

(1) *Efficiency* For efficient operation the vascular structure should contain narrow but long vessels so as to maximize the parenchymal lung volume (mainly air). Vessels must be short and of a sufficiently large caliber to minimize fluid resistance and be compliant to minimize impedance. For efficient construction and repair, vessels would need to be as narrow as possible to minimize the amount of blood and wall material [33].

(2) *Adaptivity* Increased compliance would reduce the magnitude of the impedance spectrum for the harmonic components of heart rate, but would maximize arterial blood volume, which would compromise capacity to adapt to rapid changes in blood volume and pressure. Hence arteries would need to be stiff for adequate response to fast changes in blood pressure (as in baroreceptors) to avoid instabilities in distribution of blood volume.

The cost function ($F(P)$) was described with specific weighting given to a number of apparently conflicting requirements:

$$F(P) = w_1\left[V_p\right] + w_2[V_v] + w_3[Z_i] + w_4[C_i]$$

where P is a parameter space related to lung lobar geometry, lobar elasticity and large vessel caliber; V_p is a function related to parenchymal volumes; V_v describes lobar and large artery volumes; Z_i is an impedance vector at different branching generations ($i = 1, 2, \ldots, N$) described by features of the impedance modulus shown in Fig. 3b. The specific impedance values used in the cost function were the value at 0 Hz (i.e. peripheral resistance), characteristic impedance obtained by the average of impedance modulus between 2 and 10 Hz (corresponding to the range of 2–10 harmonics as in the calculations for the different animal species with different body size and heart rates, Table 1) and impedance modulus value of the secondary peak. C_i is the total compliance at each generation ($i = 1, 2, \ldots, N$); w_1, w_2, w_3, w_4 are weights determined from optimization algorithms.

Using this technique of vascular impedance as a central parameter for optimization criteria based on optimality principles of functional design, Lefevre [17] estimated optimal values of lobar and haemodynamic parameters related to steady and pulsatile arterial function such as vessel caliber and pulse wave velocity for each generation of the pulmonary vascular tree. The theoretically derived values based on optimization of the cost function showed close agreement with physiological values of pressure, flow and impedance, and vascular and lobar dimensions [12, 19, 20, 48]. Although Lefevre [17] applied this technique to the pulmonary circulation, it could similarly be applied to the systemic circulation. Furthermore, the cost function was able to be optimized using the oscillatory impedance components corresponding to a relatively small fraction of the total external stroke work (of the order of 10 and 25% for left and right ventricle, respectively [23]). Other explicit optimizing indices such as minimization of reflected energy would entail high level of arterial compliance or low pressures, which would not be entirely physiologically feasible, although these were included as constraints to the model [17].

4 Input impedance of arterial systems at different scales—effect of animal body size

The association of animal size and physiological parameters is considered in terms of allometric relations where a parameter (x) scales with body weight (BW) to a certain

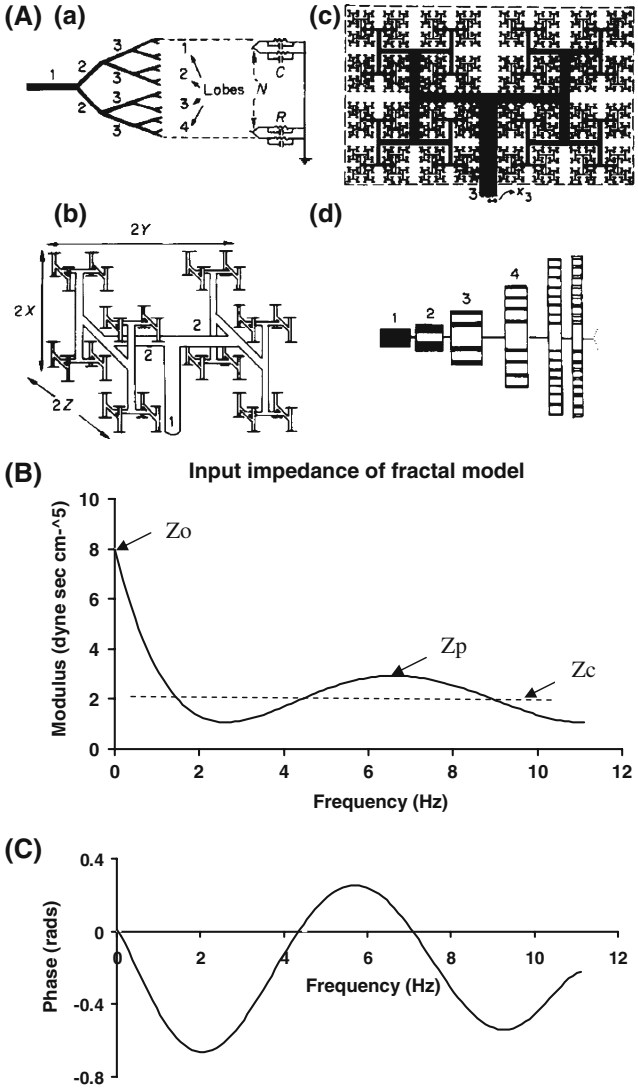

Fig. 3 **A** The dichotomous branching structure used by Lefevre [17] for construction of the fractal model for optimization of the energy cost function. *a* Self similar branching structure up to 20 generations; *b* three-dimensional orthogonal branching structure in the *X–Y–Z* planes; *c* two-dimensional projection (*X–Z* plane) of a lobe of the model, with vessels in *black* and, the cube representing parenchymal volume; *d* equivalent tube structure in series with similar cross-sectional area in each generation of branching as that shown in *a*. **B** Impedance modulus of model. Values shows as Z0, Zp, Zc were used in the cost function for parameter optimization. **C** Impedance phase of model

power (k), $x = a\mathrm{BW}^k$, where a is a constant [34]. While these relations apply to some fundamental circulatory parameters such as heart size, heart rate, cardiac output, lung volumes, parameters such as arterial pressure, pulse wave velocity, blood flow velocity, sarcomere length for mammalian muscle, do not depend on body size. Such parameters are essentially physical constants that can be thought of as imposing absolute constraints on design criteria [10, 34]. Hence, for the heart muscle, as for skeletal muscle, the structure of the contractile machinery being independent of body size means that the maximum force per unit cross-sectional area of muscle would be essentially similar for a mouse and for an elephant. That is, for a contracting heart, the scaling parameters and the physical constants would result in the appropriate level of force developed to expel the blood from the ventricles. Hence, for different stroke volumes and aortic dimensions, both of which scale to body size, the aortic velocity of the ejecting blood is essentially independent of body size [18, 21, 25]. This is an important consideration when comparing input impedance obtained from animals (or models) of different body size. Magnitudes can be readily compared when scaled for cross-sectional area of the aortic root [1].

It has been shown that the low frequency portion of the impedance spectrum can be modeled by a single tube and that the frequency of minimum impedance modulus ($f_{\min}$) and the pulse wave velocity (c) can be used to estimate the effective length (L_{eff}) by equating it to a quarter wavelength ($L_{\mathrm{eff}} = c/4f_{\min}$) [23, 26]. Data shown in Table 1 were derived from a number of our previous studies conducted in different animal species with a large range of body size [1–4, 24]. The impedance spectra for each species were used to determine $f_{\min}$ and measured values of wave velocities were used to determine L_{eff}. In addition, the impedance plots were normalized with respect to characteristic impedance (determined by averaging between 2 and 8 harmonic) and the frequency axis was normalized with respect to heart rate (Fig. 4). This process of normalization allows comparison across the range of species and shows comparable values at the frequency corresponding to heart rate. The species that stand out, however are the snake and the human. This suggests that at the normal heart rate, these are less well matched as the relative impedance is more than three times that of the other species.

The allometric relationship shown in Fig. 5 has been determined using the data only for the mammalian species (ie the snake has been omitted). The relation between body weight and L_{eff} shows a strong correlation indicating that L_{eff} can be considered to belong to the set of parameters related to design criteria. The body weight exponent is 0.35. This is similar to the value of 0.32 reported in other studies which relate indices of arterial length and body weight [18, 19]. This is entirely consistent with the relationship of length to volume to the power of 1/3 assuming a constant density. A similar range of exponent for body weight (0.33–0.38) was obtained for diastolic time (T_{d}) and diastolic time constant (τ) for aortic pressure in other studies which used normalized impedance to demonstrate that T_{d}/τ is independent of body size [45]. These results have been applied to the evolutionary basis of ventricular/vascular coupling. Notwithstanding the low number of species, the large range of body weight produced a high

Table 1 Data from [1–4, 24]

Species	WT (kg)	HR (Hz)	PWV (cm s^{-1})	Z mod 1	Φ (deg)	$Z \cos \Phi$	f_{min} (Hz)	L_{eff} (cm)
Rat	0.45	5.8	600	1.18	−55	0.60	14.0	10.7
Guinea pig	0.70	3.9	480	1.45	−44	1.04	14.0	10.0
Rabbit	3.0	3.5	450	1.10	−47	0.75	4.5	25
Snake	5.6	0.67	593	7.12	−59	3.36	3.0	49
Dog	5.8	2.4	590	1.49	−74	0.41	4.0	31
Kangaroo	23.3	1.4	469	1.24	−67	0.48	2.5	47
Sheep	33	2.1	520	1.06	−38	0.84	2.0	65
Human	72	1.2	600	3.53	−59	1.81	3.5	43

WT, Body weight; HR, heart rate; PWV, aortic pulse wave velocity; Φ, phase for first harmonic; $Z \cos(\Phi)$, in-phase impedance; f_{min}, frequency of impedance minimum; L_{eff}, effective length

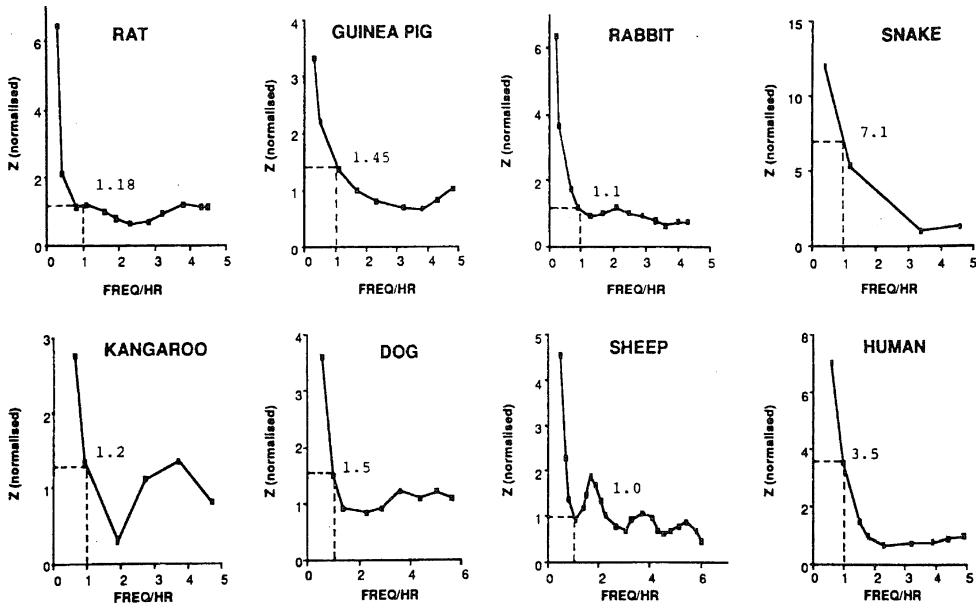

Fig. 4 Impedance modulus normalized for characteristics impedance and resting heart rate (HR) for each species. The figure indicates the normalized modulus value at the equivalent resting heart rate

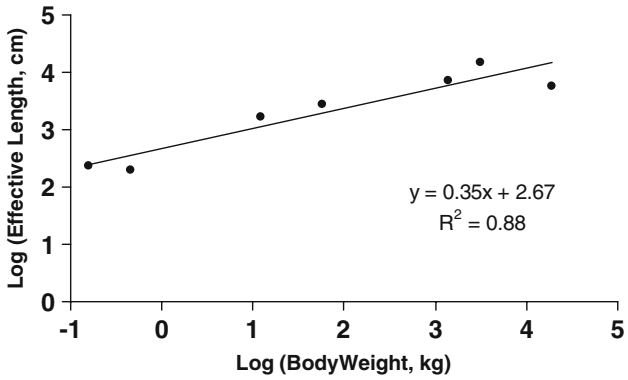

Fig. 5 The relationship between body weight and effective length calculated using the values of pulse wave velocity and frequency of minimum impedance modulus as shown in Table 1. The regression relation was calculated only for the mammalian species

correlation and so allows potentially useful application of the model. As an example, the actual L_{eff} for the human (43 cm) is 61% of the optimal value (70 cm) determined from the allometric equation. The implication of this is that the height of the adult human may be too short for optimal matching between the heart and arterial system. That is, the matching could be compromised due to a shorter effective arterial length. Since wave velocity does not scale with body weight, the return of reflected waves in humans would be relatively earlier than in other species, and so contribute to increased load on the ejecting ventricle. Whereas this observation was obtained from a range of animals of varying body size, there is recent evidence that body stature within the same species can be a significant factor. By analysis of allometric and anthropometric relations, Heymsfield et al. [11] have found that taller adult humans tend to have a lower mass-specific resting energy expenditure. Smulyan et al. [37] also showed a significant relationship between body height and parameters of oscillatory hemodynamics in a cohort of subjects with normal renal function and end stage renal disease. From these data the relation between heart rate and body weight can be estimated with a body weight exponent of 0.27, which is in close agreement with the value of 0.25 derived from allometric relationships from a large number of species of varying body size [34].

5 Conclusions

Vascular impedance is determined by the geometric and mechanical properties of the branching vascular structure. The definition of impedance in terms of the ratio of frequency components of pressure and flow assumes linearity, such that a single sinusoid of flow as input produces a sinusoid of pressure as output with identical frequency and with a phase shift. Although the mechanical behavior of arteries is known to be nonlinear, the nonlinearities are small over the normal operating range, hence making the linear approach applicable. Furthermore, impedance of a vascular structure is not dependent on the type of input waveform; that is, it does not depend on the function of the ventricle. These haemodynamic observations have made it possible to develop models where relationship of pressure and flow could be investigated in either lumped parameter models or distributed structures. The impedance spectrum becomes the central validating parameter for the models. Input impedance is a complete description of the haemodynamic load on the ejecting ventricle, and for normal operating conditions, it is a determinant of the aortic pressure pulse which is propagated throughout the arterial tree. For a range of different spectral beds, the rapid fall of impedance modulus from a peripheral resistance value to one or more local minimum value and then oscillate around a theoretical value of characteristic impedance shows a similar qualitative pattern. The particular arterial system properties would then alter specific parameters of the impedance pattern, such as the rate of fall at low frequencies, the values of characteristic impedance and the frequencies of impedance modulus minima and phase zero crossing. If all vascular beds and animal sizes obeyed a type of pure self-similarity principle [51], the normalized impedance spectra in terms of the fundamental frequency corresponding to the resting heart rate would be similar. The fact that the normalized impedances are not entirely similar (as in the input impedance of the snake and human, or in different vascular beds) can aid in identifying alterations that pertain to both geometrical factors (such as branching configurations) and arterial properties.

This paper has discussed the role of the input impedance spectrum of distributed vascular structures in investigations of haemodynamic concepts related to the branching nature of the arterial tree. The powerful attributes of the distributed models have been illustrated by systems exhibiting random branching and fractal properties. The impedance spectrum determined with such models is similar to that in anatomical systems, even though the branching configuration is different. This allows reduction of these complex models to simple tractable models such that elementary parameters of wave propagation and effective arterial lengths can be used to investigate evolutionary aspects of optimal design across a range of animal body size.

References

1. Avolio AP, O'Rourke MF, Mang K, Bason PT, Gow BS (1976) A comparative study of pulsatile arterial hemodynamics in rabbits and guinea pigs. Am J Physiol 230(4):868–875
2. Avolio AP, O'Rourke MF, Webster ME (1983) Pulse-wave propagation in the arterial system of the diamond python *Morelia spilotes*. Am J Physiol 245(6):R831–R836
3. Avolio AP (1980) Multi-branched model of the human arterial system. Med Biol Eng Comput 18(6):709–718. doi:10.1007/BF02441895
4. Avolio AP (1976) Haemodynamic studies and modeling of the mammalian arterial system. PhD Thesis, University of New South Wales, Sydney
5. Bennett SH, Goetzman BW, Milstein JM, Pannu JS (1996) Role of arterial design on pulse wave reflection in a fractal pulmonary network. J Appl Physiol 80(3):1033–1056
6. Brown DJ (1996) Input impedance and reflection coefficient in fractal-like models of asymmetrically branching compliant tubes. IEEE Trans Biomed Eng 43(7):715–722. doi:10.1109/10.503179
7. Burattini R, Di Carlo S (1988) Effective length of the arterial circulation determined in the dog by aid of a model of the systemic input impedance. IEEE Trans Biomed Eng 35(1):53–61. doi:10.1109/10.1336
8. Elzinga G, Westerhof N (1991) Matching between ventricle and arterial load. An evolutionary process. Circ Res 68(6):1495–1500
9. Grasman J, Brascamp JW, Van Leeuwen JL, Van Putten B (2003) The multifractal structure of arterial trees. J Theor Biol 220(1):75–82
10. Günther B (1975) Dimensional analysis and theory of biological similarity. Physiol Rev 55(4):659–699
11. Heymsfield SB, Childers D, Beetsch J, Allison DB, Pietrobelli A (2007) Body size and human energy requirements: reduced mass specific resting energy expenditure in tall adults. J Appl Physiol 103:1543–1550. doi:10.1152/japplphysiol.00461.2007
12. Hlastala MP, Glenny RW (1999) Vascular structure determines pulmonary blood flow distribution. News Physiol Sci 14:182–186
13. Karamanoglu M, Gallagher DE, Avolio AP, O'Rourke MF (1994) Functional origin of reflected pressure waves in a multibranched model of the human arterial system. Am J Physiol 267(5 Pt 2):H1681–H1688
14. Karamanoglu M, Gallagher DE, Avolio AP, O'Rourke MF (1995) Pressure wave propagation in a multibranched model of the human upper limb. Am J Physiol 269(4 Pt 2):H1363–H1369
15. Kassab GS (2006) Scaling laws of vascular trees: of form and function. Am J Physiol Heart Circ Physiol 290(2):H894–H903. doi:10.1152/ajpheart.00579.2005
16. Krenz GS, Linehan JH, Dawson CA (1992) A fractal continuum model of the pulmonary arterial tree. J Appl Physiol 72(6):2225–2237
17. Lefèvre J (1983) Teleonomical optimization of a fractal model of the pulmonary arterial bed. J Theor Biol 102(2):225–248. doi:10.1016/0022-5193(83)90361-2
18. Li JK, Noordergraaf A (1991) Similar pressure pulse propagation and reflection characteristics in aortas of mammals. Am J Physiol 261(3 Pt 2):R519–R521
19. Li JKJ (1996) Comparative cardiovascular dynamics of mammals. CRC Press, Boca Raton, pp 23–57

20. Milnor WR, Conti CR, Lewis KB, O'Rourke MF (1969) Pulmonary arterial pulse wave velocity and impedance in man. Circ Res 25(6):637–649
21. Milnor WR (1979) Aortic wavelength as a determinant of the relation between heart rate and body size in mammals. Am J Physiol 237(1):R3–R6
22. Mohiuddin MW, Laine GA, Quick CM (2007) Increase in pulse wavelength causes the systemic arterial tree to degenerate into a classical windkessel. Am J Physiol Heart Circ Physiol 293(2):H1164–H1171. doi:10.1152/ajpheart.00133.2007
23. Nichols WW, O'Rourke MF (2005) McDonald's blood flow in arteries. Arnold, London
24. Nichols WW, Avolio AP, O'Rourke MF (1986) Ascending aortic impedance patterns in the kangaroo: their explanation and relation to pressure waveforms. Circ Res 59(3):247–255
25. Noordergraaf A, Li JK, Campbell KB (1979) Mammalian hemodynamics: a new similarity principle. J Theor Biol 79(4):485–489. doi:10.1016/0022-5193(79)90239-X
26. Noordergraaf A (1978) Circulatory system dynamics. Academic Press, New York, pp 105–156
27. O'Rourke MF, Avolio AP (1980) Pulsatile flow and pressure in human systemic arteries. Studies in man and in a multibranched model of the human systemic arterial tree. Circ Res 46(3):363–372
28. O'Rourke MF (1967) Pressure and flow waves in systemic arteries and the anatomical design of the arterial system. J Appl Physiol 23(2):139–149
29. Pollack GH, Reddy RV, Noordergraaf A (1968) Input impedance, wave travel, and reflections in the human pulmonary arterial tree: studies using an electrical analog. IEEE Trans Biomed Eng 15(3):151–164. doi:10.1109/TBME.1968.4502559
30. Quick CM, Berger DS, Stewart RH, Laine GA, Hartley CJ, Noordergraaf A (2006) Resolving the hemodynamic inverse problem. IEEE Trans Biomed Eng 53(3):361–368. doi:10.1109/TBME.2005.869664
31. Quick CM, Young WL, Noordergraaf A (2001) Infinite number of solutions to the hemodynamic inverse problem. Am J Physiol Heart Circ Physiol 280(4):H1472–H1479
32. Rashevsky N (1960) Mathematical biology. Dover, New York
33. Rosen R (1967) Optimality principles in biology. Butterworths, London
34. Schmidt-Nielsen K (1984) Scaling: why is animal size so important. Cambridge University Press, Cambridge
35. Segers P, Stergiopulos N, Verdonck P, Verhoeven R (1997) Assessment of distributed arterial network models. Med Biol Eng Comput 35(6):729–736. doi:10.1007/BF02510985
36. Sheng C, Sarwal SN, Watts KC, Marble AE (1995) Computational simulation of blood flow in human systemic circulation incorporating an external force field. Med Biol Eng Comput 33(1):8–17. doi:10.1007/BF02522938
37. Smulyan H, Marchais SJ, Pannier B, Guerin AP, Safar ME, London GM (1998) Influence of body height on pulsatile arterial hemodynamic data. J Am Coll Cardiol 31(5):1103–1109. doi:10.1016/S0735-1097(98)00056-4
38. Snyder MF, Rideout VC, Hillestad RJ (1968) Computer modeling of the human systemic arterial tree. J Biomech 1(4):341–353. doi:10.1016/0021-9290(68)90029-8
39. Stergiopulos N, Meister JJ, Westerhof N (1995) Scatter in input impedance spectrum may result from the elastic nonlinearity of the arterial wall. Am J Physiol 269(4 Pt 2):H1490–H1495
40. Stergiopulos N, Young DF, Rogge TR (1992) Computer simulation of arterial flow with applications to arterial and aortic stenoses. J Biomech 25(12):1477–1488. doi:10.1016/0021-9290(92)90060-E
41. Taylor MG (1966) The input impedance of an assembly of randomly branching elastic tubes. Biophys J 6(1):29–51
42. Taylor MG (1966) Wave transmission through an assembly of randomly branching elastic tubes. Biophys J 6(6):697–716
43. Taylor MG (1969) In: Fishman AP, Hecht HH (eds) The pulmonary circulation and interstitial space. University of Chicago Press, Chicago
44. Westerhof N, Bosman F, De Vries CJ, Noordergraaf A (1969) Analog studies of the human systemic arterial tree. J Biomech 2(2):121–143. doi:10.1016/0021-9290(69)90024-4
45. Westerhof N, Elzinga G (1991) Normalized input impedance and arterial decay time over heart period are independent of animal size. Am J Physiol 261(1 Pt 2):R126–R133
46. Westerhof N, Elzinga G, Sipkema P (1971) An artificial arterial system for pumping hearts. J Appl Physiol 31(5):776–781
47. Westerhof N, Scarborough WR, Noordergraaf A (1967) Some experiments on a delay line simulating the human systemic arterial tree, with special emphasis on the ballistocardiogram. Bibl Cardiol (19):141–150
48. Wiener F, Morkin E, Skalak R, Fishman AP (1966) Wave propagation in the pulmonary circulation. Circ Res 19(4):834–850
49. Womersley JR (1957) The mathematical analysis of the arterial circulation in a state of oscillatory motion. Technical Report Wade-T. 56–614. Wright Air Development Centre, Dayton
50. Womersley JR (1958) Oscillatory flow in arteries: the reflection of the pulse at junctions and rigid inserts in the arterial system. Phys Med Biol 2:313–323. doi:10.1088/0031-9155/2/4/301
51. Zamir M (1999) On fractal properties of arterial trees. J Theor Biol 197(4):517–526. doi:10.1006/jtbi.1998.0892