Pressure pulse velocity is related to the longitudinal elastic properties of the artery

2004 Physiol. Meas. 25 1397

(http://iopscience.iop.org/0967-3334/25/6/005)

INSTITUTE OF PHYSICS PUBLISHING PHYSIOLOGICAL MEASUREMENT

Physiol. Meas. **25** (2004) 1397–1403 PII: S0967-3334(04)81656-3

Pressure pulse velocity is related to the longitudinal elastic properties of the artery

Yuh-Ying Lin Wang[1,2], Ming-Yie Jan[2], Gin-Chung Wang[3,4], Jian-Guo Bau[2] and Wei-Kung Wang[2,4]

[1] Department of Physics, National Taiwan Normal University, Taipei, Taiwan, Republic of China
[2] Biophysics Laboratory, Institute of Physics, Academia Sinica, Taipei, Taiwan, Republic of China
[3] Department of Electrical Engineering, National Taiwan University, Taipei, Taiwan, Republic of China
[4] Department of Electrical Engineering, U. C. Berkeley, USA

E-mail: yuhying@phy03.phy.ntnu.edu.tw

Received 2 June 2004, accepted for publication 9 July 2004
Published 7 September 2004
Online at stacks.iop.org/PM/25/1397
doi:10.1088/0967-3334/25/6/005

Abstract

It is known that arteries in their natural position are always subject to a longitudinal stress. However, the effect of this strong longitudinal tension has seldom been addressed. In this paper, we point out that the traditional pulse wave velocity formulae considering only the circumferential elasticity fail to include all the important energies. We present a vigorous derivation of a pressure wave equation, *the pressure wave equation with total energy*, which considers all the important energies of the whole arterial system by treating the arterial wall and the blood as one system. Our model proposes that the energy transport in the main arterial system is primarily via the transverse vibration motion of the elastic wall. The final equation indicates that the longitudinal stress is essential and the high frequency phase velocity is related to the longitudinal tension along the arterial wall and its Young's shearing modulus. By applying this equation, we suggest that longitudinal elastic property is an important factor in hemodynamics and in the treatment of cardiovascular diseases.

Keywords: pulse wave velocity (PWV), hemodynamics, compliance, longitudinal tension

1. Introduction

Pulse wave velocity (PWV) is an index of arterial stiffness (Blacher *et al* 1999) and arterial stiffness is a predictor of cardiovascular mortality in hypertensive patients (Laurent *et al* 2001). O'Rourke *et al* (2002) stated that the most hallowed measure of arterial stiffness is pulse wave velocity. The Moens–Kortweg's formula for the PWV has been widely used since1878.

Starting from Euler (1755), hemodynamic theories have been expressed as blood (fluid) flowing through vessels (pipe) (Noordergraaf 1978, Milnor 1989, Fung 1996, Nichols and O'Rourke 1998, Li 2000). Based on this model, the Navier–Stokes equation is widely used to describe this fluidic system. However, Milnor *et al* (1966) pointed out that the kinetic energy contributes merely 2 to 7% of the total ventricular output under basal resting conditions. This suggests that the traditional model where the blood is adopted as the primary system and the elastic wall as its boundary is far from complete.

Bergel (1961) and McDonald (1974) found that the natural length of most of the arteries is never reached and a considerable amount of force is needed to pull it to its *in vivo* length. The *in situ* longitudinal strain was studied by Han and Fung (1995). Arteries are subject to a longitudinal tension in natural position. What is the effect of this arterial structure?

Recently, we pointed out that the area gradient is automatically accompanied with the pressure gradient for a compliant artery. We further quantitatively estimated that the contribution of the area gradient force is in comparable order to the pressure gradient force for the axial blood flow (Jan *et al* 2004). Most studies in PWV, including the derivation of the Moens–Kortweg's formula, considered the pressure gradient force only and omitted the area gradient force without giving any justification. The main reason is that retention of the area gradient force term will cause the derivation of a linear wave equation to be inaccessible. Some researchers tried to take the oscillating wall as a correction factor, which resulted in complicated final equations and parameters, such as the work of Womersley (1955).

To overcome this difficulty, we construct a model that takes the blood and the vessel together as one system so that the interaction forces between them become the internal forces. By observing the large longitudinal stretch, we construct a pressure wave model that is similar to a transverse vibration wave in a stretched elastic string. In this new model, energies associated with both the axial motion and the transverse motion of the blood and the elastic vessel are included. Furthermore, the linearity of the final wave equation is still attainable.

By analyzing the new model, we find that the longitudinal stress is essential for energy transportation in the circulatory system. In addition, we deduce that the PWV is related to the longitudinal elastic properties of the artery.

2. Theory: a vigorous derivation of the pressure wave equation with total energy

The arterial wall and the blood were treated as two separate systems in most of the hemodynamics study. For example, in the derivations of the Moens–Korteweg's equation (Moens 1878) and the Womersley equation (Womersley 1955), the blood was taken as the main system and the arterial wall was taken as its boundary. However, there is high energy transfer between the blood and the elastic wall. The interaction forces between the blood and the vessel are too complicated to be expressed precisely in mathematical form. Nevertheless, the forces are so important that we cannot either neglect them or take them as a correction term without giving a logical reason.

We tackle this problem by constructing a model that combines the blood and the vessel together as one system so that the interaction forces between them become the internal forces, avoiding the difficulties in managing the boundary between the blood and the vessel.

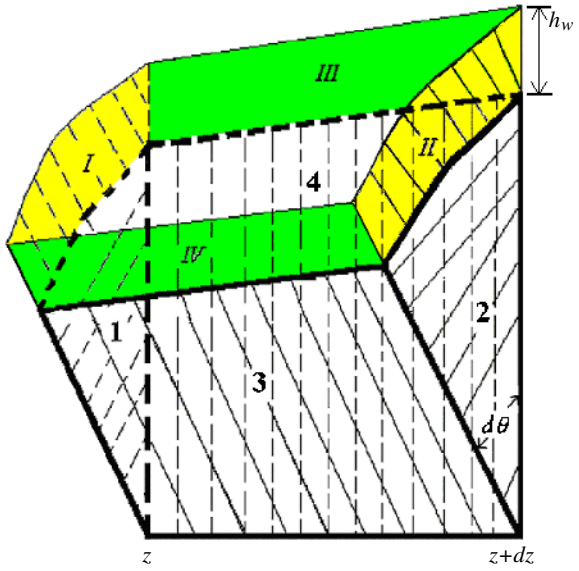

Figure 1. A small element of azimuth angle $d\theta$ of the arterial segment is the basic system to be studied. It is composed of a thin layer of vessel with thickness h_w and a wedge-shaped fluid. The element is in contact with the surrounding via five surfaces at the vessel and four surfaces at the fluid.

(This figure is in colour only in the electronic version)

We assume the cross section of the artery is circular with inner radius $r(z, t)$, where z is the axial position and t is the time. The radial equation of motion is studied by taking a small element of azimuth angle $d\theta$ from the cylindrical segment of axial length dz. The element is composed of a thin layer of vessel of width h_w and a wedge-shaped fluid (figure 1). It is in contact with the surroundings via five surfaces at the vessel and four surfaces at the fluid. The Newton's equation of motion of this element is determined by the forces acting on these nine surfaces. The complicated forces acting on the adjacent surface between the fluid and the elastic vessel are internal forces and will cancel each other due to Newton's third law.

The pressure forces $A_1\bar{P}_1$ and $A_2\bar{P}_2$ act on surfaces 1 and 2 by the outside adjacent fluids in opposite axial directions, and the total effect is an area gradient force accompanied with a pressure gradient force. Both forces are in the axial direction and would not contribute to our equation in radial direction.

The pressure forces acting normally on surface 3 and 4 by the adjacent fluid will contribute a net force F_p in the radial direction:

$$F_p = (A_3\bar{P} + A_4\bar{P})\sin(d\theta/2) = \bar{P}r\,dz\,d\theta. \tag{1}$$

The elastic vessel is subjected to a longitudinal tension T_w per unit circumferential length. Longitudinal stretching forces $T_w r_1\,d\theta$ and $T_w r_2\,d\theta$ are acting outwards tangentially by outside vessels contacting the surfaces I and II. The local slopes $(\partial r/\partial z)$ at z and $z + dz$ are small but different, there will be an effective tension force F_T in the radial direction, and

$$F_T = \left(T_w r_2\,d\theta\frac{\partial r}{\partial z}\right)_{\mathrm{II}} - \left(T_w r_1\,d\theta\frac{\partial r}{\partial z}\right)_{\mathrm{I}} = T_w r\,d\theta\frac{\partial^2 r}{\partial z^2}\,dz. \tag{2}$$

This force is quite similar to the transverse force term arising from the longitudinal tension in the case of transverse wave in an elastic string (Kreyszig 1999).

We further assume the elastic vessel obeys the Hooke's rule, so that the outside elastic vessel elements adjacent to surface I, and surface II will act restoring shearing stresses on the element system, the resulting shearing force F_s is in the radial direction and

$$F_s = \left(E_{rZ} h_w r_2 \, \mathrm{d}\theta \frac{\partial r}{\partial z} \right)_{\mathrm{II}} - \left(E_{rz} h_w r_1 \, \mathrm{d}\theta \frac{\partial r}{\partial z} \right)_{\mathrm{I}} = E_{rz} \, h_w r \, \mathrm{d}\theta \frac{\partial^2 r}{\partial z^2} \, \mathrm{d}z. \tag{3}$$

Here E_{rz} is the shearing modulus of elasticity of the vessel.

Similarly, assuming the wall has circumferential elastic modulus E_θ, thus on surfaces III and IV, there are restoring circumferential stresses acting by the outside adjacent vessels. The vector sum of these two forces is called the restoring circumferential force F_c. It is in the negative radial direction and

$$F_c = -2E_\theta \frac{\Delta r}{r} h_w \, \mathrm{d}z \sin(\mathrm{d}\theta/2) = -E_\theta h_w \frac{\Delta r}{r} \, \mathrm{d}\theta \, \mathrm{d}z \tag{4}$$

where $\Delta r = r - r_0$ and r_0 is the static radius.

On the upper surface, there is local external pressure $P_0(z)$, it will contribute a force F_{P_0}, which is in the negative radial direction, and

$$F_{P_0} = -P_0 r \, \mathrm{d}\theta \, \mathrm{d}z. \tag{5}$$

Next, we consider the resistance force F_f in the radial direction acting on the element system that is conducting a transverse vibration motion. This resistance force is due to the viscosity of the blood, the viscoelasticity of the wall as well as the conditions of the surrounding medium. For simplicity, we assume that F_f is proportional to the radial velocity $\mathrm{d}r/\mathrm{d}t$, and

$$F_f = -R \frac{\mathrm{d}r}{\mathrm{d}t} r \, \mathrm{d}\theta \, \mathrm{d}z \tag{6}$$

where R is the resistance constant. Here we will apply the Newton's equation of motion of the basic element system only in the radial direction.

Let P_r be the momentum of the element in the radial direction. If we assume there is a thin layer of thickness h_b and density ρ_b of blood moving transversely with the elastic vessel that is of thickness h_w, density ρ_w and velocity $\partial r/\partial t$, then

$$P_r = \mu \partial r/\partial t \, r \mathrm{d}\theta \, \mathrm{d}z = \mu \frac{\partial r^2}{\partial t} \, \mathrm{d}\theta \frac{\mathrm{d}z}{2}. \tag{7}$$

Here $\mu = \rho_w h_w + \rho_b h_b$.

Thus, the Newton's equation of motion becomes

$$\frac{\partial P_r}{\partial t} = F_p + F_{P_0} + F_f + F_c + F_T + F_S. \tag{8}$$

We may substitute equations (1) to (7) into equation (8) and integrate over θ from zero to 2π. By defining $S = \pi r^2$, the cross section of the local vessel, we have

$$\mu \frac{\partial^2 S}{\partial t^2} + R \frac{\partial S}{\partial t} + 2\pi [(E_\theta h_w \Delta r/r) - r(\bar{p} - P_0)] = \tau \frac{\partial^2 S}{\partial z^2} + F_{\mathrm{ext}}. \tag{9}$$

Here, $\tau = E_{rZ} h_w + T_w$. F_{ext} is any other additional external force per unit axial length acting on the arterial system. For example, if the heart gives an input force $F(t)$ at $z = \xi$, we may write $F_{\mathrm{ext}}(z, t) = F(t)\delta(z - \xi)$, where $\delta(z - \xi)$ is a delta function. The area compliance C_A is defined as $C_A = \frac{\mathrm{d}S}{\mathrm{d}P}$, and the gauge pressure P is defined as the difference

between the internal fluid pressure $\bar{P}(z,t)$ and the surrounding external pressure $P_0(z)$. That is $P(z,t) = \bar{P}(z,t) - P_0(z)$.

Thus equation (9) becomes

$$\mu\frac{\partial^2 P}{\partial t^2} + R\frac{\partial P}{\partial t} + \kappa P = \tau\frac{\partial^2 P}{\partial z^2} + \frac{1}{C_A}F_{\text{ext}}. \tag{10}$$

Here

$$\kappa \cong \frac{E_\theta h_w}{r^2} - \frac{2\pi r}{C_A}. \tag{11}$$

Equation (10) can be further expressed as

$$\frac{\partial^2 P}{\partial t^2} + b\frac{\partial P}{\partial t} + \omega_0^2 P = V_\infty^2\frac{\partial^2 P}{\partial z^2} + \frac{2\pi r_0}{LC_A}F_{\text{ext}}. \tag{12}$$

Here $\omega_0^2 = \kappa/\mu$, and the high frequency phase velocity $V_\infty = \sqrt{\frac{\tau}{\mu}}$.

Or we may express it as

$$V_\infty = \sqrt{\frac{T + S_w E_{rz}}{L}}. \tag{13}$$

Here $T = 2\pi r_0 T_w$ is the longitudinal tension along the elastic vessel, $S_w = 2\pi r_0 h_w$ is the cross sectional area of the elastic wall, $L = 2\pi r_0\mu = 2\pi r_0(\rho_w h_w + \rho_b h_b)$ and is the mass per unit axial length of the vessel and the adherent blood that conducts the transverse vibration.

We name equation (10) or equation (12) as *the pressure wave equation with total energy*. The pressure wave equation derived here is similar to the wave equation on a stretched elastic string. Both of them can be applied to systems of comparable or higher dimension wavelength. Both are based on the same assumption that the slope due to the wave vibration is small, i.e., the amplitude of the radial oscillation is smaller than the wavelength of the pressure wave, which is a perfect fit for the real physiological condition of any arterial system.

3. Result and discussion

The high frequency phase velocity (equation (13)) in the pressure wave equation with total energy is similar to the Moens–Koteweg's pulse wave velocity formula, which depends on the geometry and mass density of the arterial system and on the stiffness of the arterial wall. However, in Moens–Korteweg's formula or many other modified formulae, the stiffness is counted through the Young's modulus of elasticity related to the circumferential stretching, while our formula indicates that PWV is mainly associated with the Young's shearing modulus and the longitudinal tension of the blood vessel.

Traditionally, the usual justification for focusing on the longitudinal equation of motion of the blood is that the longitudinal velocity component of the blood is much larger than the radial one. However, the kinetic power is only 2% to 7% of the total ventricular output under basal resting conditions (Milnor *et al* 1966). Exchanging the roles of the perturbation and the main Hamiltonian leads to inaccurate equations.

In contrast, our derivation of the equation of motion takes all the important energies including pressure energy, kinetic energy and elastic potential energies into consideration. The circumferential elastic energy is related to the area compliance C_A, while the longitudinal elastic energy is related to the shearing modulus of elasticity E_{rz} of the vessel and longitudinal tension T_w per unit circumferential length. The effect of the circumferential elasticity on the wave velocity for different frequencies could be linked via ω_0^2 or κ in equation (11).

The average percentage of the relative shortening of artery in terms of *in vivo* length ranges 21% to 42% (Fuchs 1900, Bergel 1961, McDonald 1974). This stretching structure facilitates the propagation of the transverse vibration wave, which is analogous to pulling an elastic string tightly to make the transverse vibration possible.

The circumferential elastic property does help transforming the local blood pressure energy into local axial kinetic energy of the blood. However, if we neglect the local slope of the elastic vessel, as most of the hemodynamic studies did, the pressure wave cannot propagate long due to the viscosity of the blood. Neglecting the local slope cannot be justified by arguing that the variation of the tube radius is small as compared with either the static radius of the artery or the wavelength of the pressure wave.

The blood vessel can be described as a bundle of oscillating strings with longitudinal stretching. The restoring shearing stress and the radial component of the tension arising from the local slope of the string make the pressure wave traveling from one Windkessel unit to the adjacent Windkessel unit as long as the longitudinal tension is high enough. This phenomenon can be understood by watching small slope transverse vibration wave in a string; the wave can exist so long as the string is under high tension.

Recently, we (Lin Wang *et al* 2004) gave a short communication to propose some mechanism of the circulatory system. Avolio and Kerkhof (2004) commented that many of the concepts need further investigation and *in vivo* experimental validation. We stated that the ultimate purpose of the circulation is to facilitate the whole arterial system maintaining a steady transverse vibration, that is, to keep a large area of oscillation for all the blood vessels. The amplitude of the local area wave is proportional not only to the local pressure, but also to the local distensibility. Therefore, a compliant arterial wall is more efficient than a stiff one under the same pressure pulse.

It has been studied that the circumferential elastic modulus varies with the pressure (Bergel 1961); here we propose that the effect of the longitudinal stretch on the wall's compliance is also an important factor that needs to be studied physiologically. A string with longitudinal stress is easier to oscillate than a loose one; however, if it is too tight, the string's structure will be distorted and its natural frequencies are changed. Similarly, in the arterial system, there is an optimal condition in the longitudinal stress that makes the circulation reach its highest efficiency in energy transportation. Finding ways to tune the arterial longitudinal elastic properties to meet the optimized energy consumed is an important issue in the healing of many diseases.

Bank and Kaiser (1998) demonstrated that the effect of smooth muscle relaxation decreases PWV without altering the arterial distensibility in normal human subjects, which implies that the PWV not merely depends on the circumferential elasticity. The smooth muscle relaxation might change the longitudinal elastic properties and therefore change the PWV as proposed by equation (13).

We thus propose that future studies for the treatment of cardiovascular diseases should take the effects of the longitudinal elastic properties into account.

In deriving *the pressure wave equation with total energy*, only the following assumptions are made: (1) the cross section of the artery is circular; (2) the elastic vessel obeys the Hooke's rule; and (3) the slope due to the wave vibration is small, i.e., the amplitude of the radial oscillation is smaller than the wavelength of the pressure wave. These reasonable assumptions justify the use of this equation in many conditions in the main arterial system. For example, a geometric tapering does not necessarily induce reflection if the tube adjusts its elastic properties so that there is no phase velocity mismatch. At the bifurcation point, we can solve the equation by applying suitable boundary conditions; such as the single value of the pressure wave and the continuity of the blood flow. Our work provides a starting equation for

hemodynamics research. All parameters are clearly defined in terms of measurable quantities. Those parameters and the detailed solution of various cases can only be obtained through many further *in vivo* and simulation studies.

References

Avolio A P and Kerkhof P L 2004 On tubes, strings, and resonance in the arterial system—what makes the beat go on? *IEEE Trans. Biomed. Eng.* **51** 196–7

Bank A J and Kaiser D R 1998 Smooth muscle relaxation: effects on arterial compliance, distensibility, elastic modulus, and pulse wave velocity *Hypertension* **32** 356–9

Bergel D H 1961 The static elastic properties of the arterial wall *J. Physiol.* **156** 445–57

Blacher J, Asmar R, Djane S, London G M and Safar M E 1999 Aortic pulse wave velocity as a marker of cardiovascular risk in hypertensive patients *Hypertension* **33** 111–1117

Euler L 1755 Principes généraux du mouvement des fluides *Mémoires de l'Académie Royale des Sciences et des Belle-Lettres de Berlin* **11** 274–315

Fuchs R F 1900 Zur Physiologie und Wach-stummsmechanik des Blutgefassystems *Arch. Ges. Physiol.* **28** 7

Fung Y C 1996 *Biodynamics: Circulation* 2nd edn (New York: Springer)

Han H C and Fung Y C 1995 Longitudinal strain in canine and porcine aortas *J. Biomech.* **28** 637–42

Jan M Y, Hsu T L, Hsin H, Wang W K and Lin Wang Y Y 2003 The physical conditions of different organs are reflected in the pressure pulse spectrum of the peripheral artery specifically *Cardiovasc. Eng.: An Int. J.* **3** 21–9

Jan M Y, Hsin H, Bau J G and Lin Wang Y Y 2004 One important area gradient force has been omitted in the Moens and Korteweg's equation *Proc. 26th Annual Int. Conf. of the IEEE EMBS (San Francisco, 1–5 September 2004)* at press

Kreyszig E 1999 *Advanced Engineering Mathematics* 8th edn (New York: Wiley) pp 585–6

Laurent S, Boutouyrie P, Asmar R, Gautier I, Laloux B, Guize L, Ducimetiere P and Benetos A 2001 Aortic stiffness is an independent predictor of all-cause and cardiovascular mortality in hypertensive patients *Hypertension* **37** 1236–41

Li J K-J 2000 *The Arterial Circulation: Physical Principles and Clinical Applications* (Totowa NJ: Humana Press)

Lin Wang Y Y, Chang C C, Chen J C, Hsiu H and Wang W K 1997 Pressure wave propagation in a distensible tube arterial model with radial dilation *IEEE Eng. Med. Biol. Mag.* **16** 51–6

Lin Wang Y Y, Lai W C, Hsiu H, Jan M Y and Wang W K 2000 The effect of length on the fundamental resonance frequency of the arterial models with radial dilatation *IEEE Trans. Biomed. Eng.* **47** 313–6

Lin Wang Y Y, Jan M Y, Hsiu H and Wang W K 2002 Hemodynamics with total energy *Conf. Proc. of the 2nd Joint EMBS-BMES Conf. (Houston, October 2002)* pp 1240–1

Lin Wang Y Y, Jan M Y, Shyu C S, Chiang C A and Wang W K 2004 The natural frequencies of the arterial system and their relation to the heart rate *IEEE Trans. Biomed. Eng.* **51** 193–5

McDonald D A 1974 *Blood Flow in Arteries* 2nd edn (London: Edward Arnold)

Milnor W R, Bergel D H and Bargainer J D 1966 Hydraulic power associated with pulmonary blood flow and its relation to heart rate *Circ. Res.* **19** 467–80

Milnor W R 1989 *Hemodynamics* 2nd edn (Baltimore MD: Williams & Wilkins)

Moens A I 1878 *Die Pulskurve* (Leiden)

Nichols W W and O'Rourke M F 1998 *McDonald's Blood Flow in Arteries* 4th edn (London: Edward Arnold)

O'Rourke M F, Staessen J A, Vlachopoulos C, Duprez D and Plante G E 2002 Clinical applications of arterial stiffness; definitions and reference values *Am. J. Hypertens.* **15** 426–44

Noordergraaf A 1978 *Circulatory System Dynamics* (New York: Academic)

Womersley J R 1955 Method for the calculation of velocity, rate of flow and viscous drag in arteries when the pressure gradient is known *J. Physiol.* **127** 553–63