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# Analysis of transverse wave as a propagation mode for the pressure pulse in large arteries

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A general axial momentum equation for fluid inside a transverse vibrating elastic tube is derived here. In a system with high pressure and low elastic modulus, transverse motion of the wall contributes important nonlinear forces, and the longitudinal fluid wave which assumes that flow is governed by the Navier-Stokes equation cannot describe the pulse wave completely. By comparing the transverse elastic wave with the longitudinal fluid wave, we conclude that transverse wave is a significant wave mode in large arteries. The longitudinal stress force, commonly ignored in the literature, can be considered as a significant factor that influences the propagation of the arterial pulse. © 2007 American Institute of Physics.

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## I. INTRODUCTION

Pulse waves in arteries play important roles in current studies for the diagnosis of cardiovascular diseases. It has been found that pulse wave velocity is likely to be a marker of arterial diseases and of prognosis.<sup>1</sup>

Researchers have proposed that noninvasive recordings of the blood pressure pulse waveform from the radial artery can be used to obtain valuable diagnostic information<sup>2</sup> and have been trying to construct “transfer functions” to relate the noninvasive measurement of the peripheral blood pressure waves with the central aortic pressure wave.<sup>3,4</sup>

Theoretical studies of the pulse wave mechanism started in the early nineteenth century. A mathematical formula was developed initially by Young in 1808. Resal<sup>5</sup> later combined the axial equation of motion of fluid as well as the equation of continuity for fluid in a distensible vessel to obtain a wave equation, giving the pulse wave velocity as

$$\text{PWV} = \sqrt{\frac{Eh_w}{2r\rho_b}}, \quad (1)$$

where  $E$  is Young’s modulus of elasticity of the wall material,  $h_w$  is the thickness of vessel wall,  $r$  is the inner radius of vessel, and  $\rho_b$  is the density of blood. This equation is now known as the Moens-Korteweg formula.

Since then, hemodynamical theories are almost always expressed in the form of a model of flow  $Q$  driven by the gradient of the pressure  $P$ . Most of the basic equations can be traced back to the hydraulic Navier-Stokes equation. Thus, the force produced by the vibrating arterial wall in the axial blood motion cannot be taken into account directly. We will call all these models the longitudinal  $PQ$  wave model. It is widely believed that the longitudinal  $PQ$  wave is the only one of physiological importance,<sup>6</sup> and the original Moens-Korteweg formula for pulse wave velocity has been generally accepted to be true as a first order of approximation. A considerable fraction of the literature has been devoted to the modification of the  $PQ$  wave by considering the omission of some nonlinear correction terms.<sup>7-9</sup>

Milnor *et al.*<sup>10</sup> pointed out that axial fluid kinetic power takes less than 7% of the total ventricular output. Bergel<sup>11</sup> and McDonald<sup>12</sup> found that all arteries *in vivo* are under large longitudinal tension. In earlier works,<sup>13,14</sup> we derived a pressure wave equation with total energy and described the periodic changing of the cross-sectional area  $S$  in the arterial tube as a transverse wave with large longitudinal tension. In this mechanism, the pulse wave velocity is related to the longitudinal elasticity property of the artery; and the wall is the major medium for the wave propagation.<sup>14</sup> We will call this mode the transverse  $PS$  wave.

In this study, a general axial momentum equation for fluid inside a transverse vibrating elastic tube is derived. We show that the distributed transverse motion of the wall contributes important forces in a system with high pressure and

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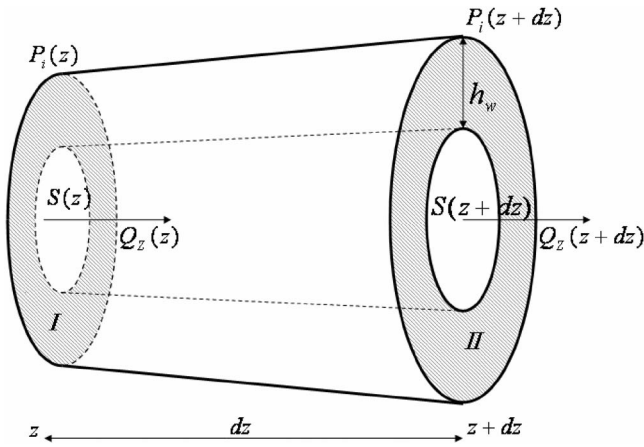


FIG. 1. A segment of a fluid-vessel system with internal fluid pressure  $P_i$ , axial fluid flux  $Q$ , luminal cross-sectional area  $S$ , and wall thickness  $h_w$  embedded in a surrounding of pressure  $P_e$ .

low elastic modulus. We apply the results to the arterial system and calculate the error caused when these terms are omitted. We examine the radial momentum equation of the artery and point out that the inappropriate elimination of some force terms has caused the transverse wave mode to be ignored historically. Comparisons of the transverse elastic  $PS$  wave and the longitudinal fluid  $PQ$  wave are listed. We also give reasons why the transverse  $PS$  wave is a significant wave mode in large arteries.

## II. THEORY

### A. Derivation of a general axial momentum equation for fluid in an elastic tube

We consider a long circular, thin wall elastic tube which is filled with incompressible fluid and is embedded in a surrounding with external pressure  $P_e$ . Figure 1 illustrates a small segment of the tube with length  $dz$ , luminal cross-sectional area  $S$ , inner radius  $r$ , and mean inner radius  $R$ . The density of the fluid is  $\rho_b$  and the thickness of the wall is  $h_w$ .  $Q$  is the total fluid flux at axial position  $z$ . It is also called the volume rate of flow and is the integration of the velocity across the lumen of the tube.<sup>6,15,16</sup> The area  $S$  is a function of the axial position  $z$  because of the distributed transverse vibration of the wall.

The interaction forces between the fluid and the elastic wall are very complicated and depends greatly on the velocity profile of the fluid. In order to avoid dealing with the complicated boundary condition between the elastic tube and fluid, we will take them together as one system. We assume a uniform internal pressure  $P_i$  over the luminal cross section. The momentum equation of the combined system along the axial direction  $z$  follows Newton's second law. The total axial momentum change of the whole system is equal to the sum of forces acting on surfaces adjacent to its neighbors and the viscous force due to the flow.

Pressure forces  $P_i(z)S(z)$  and  $-P_i(z+dz)S(z+dz)$  are acting on the two ends of the segment (Fig. 1) by two fluid neighbors. The sum of these two forces can be decomposed into a pressure gradient force  $F_P$  and an area gradient force associated with the neighboring fluid,  $F_{Ab}$ , where

$$F_P = -S \frac{\partial P_i}{\partial z} dz = -S \frac{\partial P}{\partial z} dz, \quad (1')$$

$$F_{Ab} = -P_i \frac{\partial S}{\partial z} dz. \quad (2)$$

From the external surrounding, the wall is subject to a component  $F_e$  in the  $z$  direction, and

$$F_e = 2\pi r dz P_e \frac{\partial r}{\partial z} = P_e \frac{\partial S}{\partial z} dz. \quad (3)$$

Combining the external force in Eq. (3) with the area gradient force associated with the neighboring fluid  $F_{Ab}$  in Eq. (2), we have a total area gradient force  $F_A$ ,

$$F_A = -(P_i - P_e) \frac{\partial S}{\partial z} dz = -P \frac{\partial S}{\partial z} dz, \quad (4)$$

where  $P$  is the transmural pressure given by  $P = P_i - P_e$ .

We further assume that the elastic vessel obeys Hooke's rule, so that the outside elastic vessel elements adjacent to surfaces I and II (Fig. 1) of the wall will act, restoring longitudinal stresses  $S_{zz}$  on the system, giving

$$\begin{aligned} S_{zz} &= E_{zz} \frac{\Delta l_z}{l_z} = E_{zz} \frac{[1 + (\partial r / \partial z)^2]^{1/2} dz - dz}{dz} \\ &\cong E_{zz} (\partial r / \partial z)^2 / 2. \end{aligned} \quad (5)$$

Let  $F_{zz}$  be the resulting longitudinal stress force in the axial direction, thus

$$\begin{aligned} F_{zz} &= E_{zz} h_w \pi r \left( \frac{\partial r}{\partial z} \right)^2 \Big|_{\text{II}} - E_{zz} h_w \pi r \left( \frac{\partial r}{\partial z} \right)^2 \Big|_{\text{I}} \\ &= E_{zz} h_w \pi \frac{\partial}{\partial z} \left[ r \left( \frac{\partial r}{\partial z} \right)^2 \right] dz. \end{aligned} \quad (6)$$

Here  $E_{zz}$  is the longitudinal Young's modulus of elasticity of the vessel.

For a system with tube strongly constrained in the axial direction, such as in the real arterial system, the axial motion of the wall is negligibly small.<sup>6</sup> The total axial momentum change of the whole system is approximately equal to the axial momentum change of the fluid only. Thus, the axial momentum equation of the combined system can be written as

$$dz \cdot \rho_b dQ/dt = F_P + F_A + F_{zz} + F_v. \quad (7)$$

$F_P$ , defined in Eq. (1), is the force due to the pressure gradient. Area gradient force  $F_A$  and longitudinal elastic restoring force  $F_{zz}$ , expressed in Eqs. (4) and (6), respectively, are the forces associated with the local slope which is induced by the distributed pulsatile movement of the elastic wall.  $F_v$  is the viscous force, which depends on the velocity profile of the axial fluid flow.

The area gradient force  $F_A$  can be related to the pressure gradient force by the chain rule,

$$F_A = -P \frac{\partial S}{\partial P} \frac{\partial P}{\partial z} dz = -\frac{PS}{K} \frac{\partial P}{\partial z} dz = \frac{2P}{E_P} F_P, \quad (8)$$

where  $K = S \partial P / \partial S$  is the elastic modulus of luminal volume change per unit length of tube, and  $E_P = R \partial P / \partial r$  is Peterson's modulus. Gosling and Budge have shown that  $K = E_P / 2$ .<sup>17</sup>

The circumferential incremental Young's modulus  $E_{inc}$  is related to Peterson's modulus as  $E_{inc} = E_P R / h_w$ .<sup>17</sup> Thus, by the chain rule, the longitudinal elastic restoring force  $F_{zz}$  in Eq. (6) is related to the pressure gradient force by

$$F_{zz} \cong -\frac{E_{zz}}{E_{inc}} \left[ \frac{R^2}{E_P^2} \left( \frac{\partial P}{\partial z} \right)^2 - \frac{2R^2}{E_P} \frac{\partial^2 P}{\partial z^2} \right] F_P. \quad (9)$$

$F_{zz}$  might affect waves with short wavelength in higher harmonic modes of the pressure pulse.

From the above, we may now prove that the longitudinal  $PQ$  wave can be deduced only in a stiff tube. In cases that Peterson's elastic modulus is much larger than the transmural pressure of the fluid, the local slope or the local luminal cross-sectional area gradient due to the small transverse oscillation is negligibly small. Then, without including the viscous effect of the fluid, Eq. (7) may be approximated as

$$\rho_b \frac{\partial Q}{\partial t} = -S \frac{\partial P}{\partial z}. \quad (10)$$

If the fluid is noncompressible and there is no leakage through lateral branches, the equation of continuity of the fluid can be expressed as<sup>6,15,16</sup>

$$\frac{\partial Q}{\partial z} = -\frac{\partial S}{\partial t} = -\frac{S}{K} \frac{\partial P}{\partial t}, \quad (11)$$

where  $K$  is the elastic modulus of luminal volume change per unit length of the tube.

Mathematically, Eqs. (10) and (11) are very similar to the two basic equations used to derive the one dimensional electromagnetic wave. Pressure  $P$  and axial flux  $Q$  play the same roles as the  $E$  field and the  $B$  field, respectively. Historically, several equations with more or less similar forms as these two equations have been derived.<sup>6,15,16</sup> Combining these two equations leads to a linear blood pressure wave equation. The famous Moens-Korteweg formula for pulse wave velocity as a function of elastic modulus and the density of fluid  $\rho_b$  was then obtained.<sup>6,15,16</sup>

Recent studies on blood wave propagation show that the nonlinearity of the hoop stress strain relation modifies the pulse wave speed.<sup>8,9</sup> In dealing with the axial momentum equation, most of the works started from the Navier-Stokes equation. Thus, the effect of the transverse motion of the elastic wall on the fluid axial motion was also unable to be considered in the first step.

The one dimensional longitudinal hydraulic  $PQ$  wave deduced from Eqs. (10) and (11) propagates along a fixed direction with the fluid as the medium. If there is a net direct fluid flow, the viscous force due to this flow may cause further attenuation in the longitudinal flow wave.

When Peterson's elastic modulus of the tube is of comparable order to the transmural pressure of the fluid, from Eq. (8) the area gradient force will contribute a significant

term. Thus, the flow equation [Eq. (10)] is no longer a good approximation to describe the real axial flow.

The mean blood pressure is about 100 mm Hg or  $1.3 \times 10^4$  N/m<sup>2</sup> for different arterial sites, arterial sizes, and even for different species. However, the value for  $E_P$  can vary markedly. The magnitude of  $E_P$  in a large artery has been reported to be of the order of  $5 \times 10^4$  N/m<sup>2</sup> for humans,<sup>18-21</sup>  $2 \times 10^4$  N/m<sup>2</sup> for rats,<sup>22</sup> and  $1 \times 10^4$  N/m<sup>2</sup> for mice.<sup>23</sup> Thus, by Eq. (8), the ratio of the area gradient force to the pressure gradient force is more than 50% in the main artery. Therefore, omitting the forces arising from the oscillating wall will cause at least 50%, or even 100% error. From this reason, no linear longitudinal  $PQ$  wave equation can be deduced even for a first order of approximation, and the Moens-Korteweg formula cannot be deduced.

Equation (8) shows that the area gradient force  $F_A$  and the pressure gradient force are proportional and in the same direction. The high blood pressure and the low elastic modulus in the main artery cause this area gradient force to reinforce the axial blood motion greatly.

In systemic arteries, axial fluid kinetic power takes only 2%–7% of the total ventricular output under basal resting conditions.<sup>6</sup> From power transportation point of view, the radial motion cannot be counted only as a correcting factor but must be taken to play a major role.

## B. Examination of the studies about the radial equation of motion in arteries

Historically, the change of the radial momentum was taken to be affected by two terms, the forces associated with the transmural pressure and the hoop stress.<sup>15</sup> Some authors<sup>8</sup> have also pointed out the existence of the force associated with the longitudinal stress. They argued that, when using the local slope as a small expansion factor, the longitudinal stress force term is a higher order perturbation compared to the pressure force term and so can be eliminated from further consideration. By doing so, they missed the important facts that the hoop stress force is in the direction opposite to the pressure force and that it increases with the pressure. Actually, as a first order approximation, these two forces are dynamically balanced by Laplace's law.<sup>15</sup> It is inappropriate to discard any unbalanced force (the longitudinal stress force) by comparing it to one of the balanced forces (either the pressure force or the hoop stress force) even though both of which are much greater than the unbalanced force. This is the same logic used when we consider the oscillation of a vertically hanging particle; we may not discard any driving force by comparing it to the gravitational force on the particle. Indeed, if we do not discard the longitudinal stress force term, a simplified transverse wave equation can be obtained.

Lin Wang *et al.* recently derived a pressure wave equation with total energy.<sup>14</sup> The luminal cross-sectional area  $S$  is the major variable so we may call the mechanism the transverse  $PS$  wave mode.

## C. Differences between the longitudinal $PQ$ wave mode and the transverse $PS$ wave mode



- (1) For the  $PQ$  wave mode, with the blood as the medium, the axial flow equation of motion is the major momentum equation. While for the  $PS$  wave mode, with the arterial wall and the adherent blood as the medium, the radial equation of motion of the artery is the major momentum equation.
- (2) Since there is a net blood flow in the axial direction, the viscous effect of the blood is greater in the  $PQ$  wave mode. If the  $PQ$  wave mode were the major wave mechanism, the pressure wave could not propagate far without significant attenuation.
- (3) Both pressure  $P$  and luminal cross-sectional area  $S$  in the  $PS$  wave mode are scalars, actions that change axial direction of the artery will cause less impact on the propagation of the  $PS$  wave. However, since flow  $Q$  is a vector, any change in the axial direction of the artery will cause a larger effect on the flow and therefore will influence the  $PQ$  wave greatly.
- (4) By considering all of the energy involved in the wave propagation, a linear  $PS$  wave equation is obtained.<sup>14</sup> However, no linear  $PQ$  wave equation can be attained in a real arterial system even for a first order approximation.
- (5) In the  $PQ$  wave mode, the pulsatile pressure or flow is a waste of energy<sup>6,21</sup> in relation essentially to the steady perfusion of the peripheral vasculature. However, in the  $PS$  wave mode, the power transported along the artery increases with the amplitude of the pulse pressure.

### III. CONCLUSION AND DISCUSSION

After Young's work in 1808, most studies in hemodynamics started from the hydraulic Navier-Stokes equation. How the transverse vibration of arterial wall directly affects the axial flow has seldom received attention.

In this study, we demonstrate that the transverse movement of the elastic wall plays an important role in the transportation of blood and must be taken into account. Equation (7) is a general axial flow momentum equation for fluid inside an elastic cylindrical tube. Linear longitudinal  $PQ$  wave can be derived only inside a tube with large elastic modulus compared to transmural pressure. The area gradient force, arising from the transverse vibration of the wall, contributes greatly to drive the flow. Its effect is much greater than other nonlinear contributions calculated by starting from the Navier-Stokes equation.

As blood bursts out periodically with high velocity from the left ventricle into the arterial system, it transfers some of its axial kinetic energy to the elastic vibration energy of the arterial wall. A transverse  $PS$  wave propagating along elastic tubes suffers less attenuation compared to a longitudinal  $PQ$  wave. One can imagine this  $PS$  wave propagation as the power transportation in a long electrical transmission line, with high voltage ( $S$ ) and low current ( $Q$ ) to reduce power loss during transmission.

From the physical and the physiological point of view, we conclude that in large artery, transverse wave is a signifi-

cant wave mode and the pulse wave cannot be governed by the  $PQ$  wave equation alone. It is until the end of the arterial system that the pressure gradient drives the blood into the highly resistive arterioles. Thus, any formula derived from the hydraulic Navier-Stokes equation, with either linear or nonlinear model, cannot describe the pulse wave completely in large arteries.

In the transverse  $PS$  wave mode, the longitudinal stress force can be considered as a significant factor that influences the propagation of the arterial pulse. Thus, the whole circulatory system is oscillating in a coupled resonance condition<sup>24</sup> and with the pressure distribution governed by a transverse wave equation.<sup>14</sup>

The extra forces associated with the nonlinearity of the hoop stress<sup>8</sup> or blood motion should be taken into consideration as perturbation terms for the transverse  $PS$  wave equation.

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