Monovariable representation of blood flow in a large elastic artery

J. F. Paquerot and S. G. Lambrakos
Physique Non Linéaire, Laboratoire Ondes et Structures Cohérentes, Faculté des Sciences, 6 Boulevard Gabriel, 21000 Dijon, France
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We introduce a general approach for modeling blood flow and its associated structures in an arterial system of variable cross section and Young's modulus. The general structure and approximations underlying our approach are based on the natural symmetry and characteristic configuration of structures that occur in an arterial system. Our approach, which is a representation of axially symmetric flow, uses a monovariable representation for both the flow field and mechanical properties. In addition, our formalism incorporates a deformation function whose form may in principle be deduced from experimental correlations between blood pressure and blood flow. The use of a deformation function provides a means for implicitly incorporating into the model system information about Young's modulus associated with the response of the artery walls.

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I. INTRODUCTION

The blood pressure and flow pulses are generated by the intermittent ejection of blood from the left ventricle of the heart. Experimental *in vivo* studies [1,2] show that these pulses propagate along the different branches of the mammalian arterial tree with characteristic nonlinear shape changes [1]. We know that the propagation of the blood is accompanied by an increase in amplitude with the creation of a steep front for the blood pressure pulse (known as "peaking" and "steepening"). These phenomena are combined with an increase of the pulse-wave celerity c [see Eqs. (15) and (16) below], which is about 3.5 m/s at the root of the aorta and 10 m/s in the femoral artery [3]. These changes are due to the mechanical properties of the cardiovascular system, which may be characterized by three general aspects. First, the walls of the arteries are elastic, which implies that there is in general a nonlinear stress-strain relationship between the artery walls and the component of the momentum transfer perpendicular to them [4-6]. The stress-strain relation is also a function of the position along the direction of the blood flow, caused by the evolution of the Young's modulus which increases with the distance from the heart and makes the artery wall become less and less elastic [3,7,8]. Second, the radius of the artery is not uniform on average and changes drastically along the extension of the arterial system. To represent analytically this taper effect when the wall is static and the artery is at rest, i.e., when the blood pressure is equal to the diastolic pressure, we adopt an equation of the form [9]

$$S_0(z)=s_0e^{-mz}, \tag{1}$$

where z is the distance from the heart, S_0 is the cross-sectional area at the root of the aorta, and m is a parameter lying between 2 and 3 m^{-1} (values given by Patel *et al.* [10]). Finally, throughout the cardiovascular system there are bifurcations for the purpose of effecting the multiple channeling of flow to different parts of the body. In accordance with these mechanical aspects of the arterial wall, we must include the principal characteristics of the blood. First, the blood can be regarded as an incompressible fluid, which is a justifiable assumption since compressibility of blood is so small in comparison with the distensibility of vessel walls. Second, although the blood is a diphasic fluid [11,12] consisting of different molecules, e.g., red cells and proteins, it appears that the assumption of a small viscosity in major vessels is a good approximation.

The majority of the literature of wave propagation in blood flow deals with linearized models [1,3,13,14] even though there has been increasing evidence of the presence of strong nonlinear phenomena [15]. Some of the first serious attempts at including nonlinear effects were made by Rudinger [16] and Anliker *et al.* [9,15,17] who considered the flow to be one dimensional. In their models, however, they impose a relation between pressure and cross-sectional area which controls the wave celerity. Recently, Hashizume [18,19] and Yomosa [6] proposed solitary wave models of pulse waves which were based on the nonlinear elasticity of the vessel wall of the artery. However, in their theories, the evolution of the elastic properties of the artery walls is neglected.

To elucidate the principal features of blood flow in large vessels and to understand the role played by the different types of structures that influence blood propagation, we introduce a monovariable formalism where the blood is treated as an incompressible nonviscous fluid and the flow has one-dimensional space properties. Furthermore, the branching effects are ignored and artery walls are assumed to be elastic and the cross section circular. Section II presents and discusses the model that we have developed for this investigation. As part of the development of our formulation, we describe in Sec. III the construction of a deformation function deduced from experimental measurements. The use of such a function provides a means of representing information about the Young's modulus as a function of the position along the direction of blood flow. In Sec. IV the results of our calculations are presented for arteries with different properties.

II. MONOVARIABLE REPRESENTATION OF QUASI-ONE-DIMENSIONAL FLOW

Given the conditions on our system, we are able to represent the system via a *monovariable* formalism in which the different parameters characterizing the state of the system, i.e., fluid and artery, at a given instant depend only on time t and the space variable z. The laws of hydrodynamics governing the transport within a nonviscous Newtonian fluid are given by the equation of continuity and Euler's equation. The equations underlying our monovariable approach are Eqs. (2) and (3). Equation (2) is the equation of continuity in which we have integrated directly the geometric conditions at the artery wall—that is, changes in the cross section of the artery with respect to time and space. It is important to note that this equation is valid only within the approximation of a monovariable spatial dependence of the different parameters. Equation (3) is the classical equation of Euler where ρ is the density of the blood ($\rho=1.06$ g/cm^3), u the velocity along z, and P the dynamic pressure of the fluid.

We have adopted the terminology *monovariable formalism* in contrast to *one-dimensional representation* to emphasize the quasi-one-dimensional character of the flow, i.e., the expansion of the artery due to pressure gradients that are perpendicular to the axis of symmetry. The values of u and P obtained with our formalism correspond in reality to the values along the axis of the artery that one would obtain if one considered explicitly the expansion of the artery. A third relation is necessary in order to have a complete specification of the system, i.e., the same number of equations as system variables. This equation describes the deformation of the artery wall under the forces exerted by the fluid and is considered as the equation of state of the system. Therefore, the system to be solved is specified by Eqs. (2) and (3), and an equation of state.

A complete specification of the state of the system at a given instant of time can be effected by means of three state variables. These three state variables are the speed of the blood flow along the artery $u(z,t)$, the pressure $P(z,t)$, and the cross-sectional area $S(z,t)$. Among the three state variables, two are independent and linked via a relation which is analogous to that of an equation of state. There exist two possibilities for a choice of this relation. We are able to specify a deformation function of the artery whose value at a given position z along the artery is either a function of pressure or a function of blood velocity—that is to say, $S=S(P,z)$ or $S=S(u,z)$. A relation of the form $S=S(P,z)$ is used in many theories where the cross-sectional area is represented as a function of location and medical pressure. Anliker *et al.* [15,17] have used experimental results to formulate this type of relation and Yomosa [6] used an equation of motion for the vessel wall. There are major assumptions in these models, however, because the relationship obtained, experimentally or analytically, is not between the dynamic pressure P along the axis of the artery and the cross-sectional area. Our formulation is based on a deformation function of the form $S=S(u,z)$. This choice is based on the fact that it is possible to deduce the characteristics of the system by the measurement of quantities related to the dynamic response of the system with respect to the deformation of the artery walls. It follows that the model system is given by the system of equations

$$\frac{\partial S}{\partial t}+\frac{\partial}{\partial z}(Su)=0 , \tag{2}$$

$$\frac{\partial u}{\partial t}+u\frac{\partial u}{\partial z}=-\frac{1}{\rho}\frac{\partial P}{\partial z} , \tag{3}$$

$$S=S(u,z) , \tag{4}$$

where the state variables of the system are (u,S,P).

An expression for the velocity field follows by combining Eqs. (2) and (4). We note first that

$$\begin{aligned}\frac{\partial S(z,t)}{\partial t}&\equiv\left[\frac{\partial S(u,z)}{\partial u}\right]_z\frac{\partial u}{\partial t} ,\\ \frac{\partial S(z,t)}{\partial z}&\equiv\left[\frac{\partial S(u,z)}{\partial u}\right]_z\frac{\partial u}{\partial z}+\left[\frac{\partial S(u,z)}{\partial z}\right]_u .\end{aligned} \tag{5}$$

Substituting these expressions into Eq. (2) and rearranging, we obtain the equation governing the velocity-field evolution related to an arbitrary deformation function $S(u,z)$, i.e.,

$$\frac{\partial u}{\partial t}+u\frac{\partial u}{\partial z}=\Phi(u,z) , \tag{6}$$

where

$$\Phi(u,z)=-\frac{1}{\left[\frac{\partial S}{\partial u}\right]_z}\left[u\left[\frac{\partial S}{\partial z}\right]_u+S\frac{\partial u}{\partial z}\right] .$$

III. CONSTRUCTION OF DEFORMATION FUNCTION $S(u,z)$

In order to solve Eq. (6), we must know the form of the function $S(u,z)$ which relates the speed of the fluid at the point z to the radial deformation of the artery. Equation (6) can be compared to the Euler equation written in the form of Eq. (3), which shows that the function $\Phi(u,z)$, and therefore $S(u,z)$, possess all the information and properties relating the system that we are studying (conicity, Young's modulus, blood density, etc.). We are therefore able to deduce the conditions necessary for the existence of the function $\Phi(u,z)$ and the equation of state $S(u,z)$ by comparison with the Euler equation.

A. Mathematical conditions

Several mathematical conditions follow by comparing Eq. (6) to Eq. (3). First, we must have $\Phi(u=0,z)=0$ because if the fluid velocity is zero at a given point z, it must be that the gradient of the pressure at that point is zero. That is,

$$\frac{1}{\rho}\frac{\partial P}{\partial z}=0\Longleftrightarrow\Phi(u=0,z)=0 . \tag{7}$$

Second, the denominator of $\Phi(u=0,z)$ must never be zero, which requires that the function $S(u,z)$ be such

that its derivative with respect to velocity is never zero for all u and z. We require equally that $S(u,z)$ by an even function of u because the response of the artery must be independent of the direction of propagation of the fluid.

In summary, one must find an equation of state which satisfies

$$\Phi(u=0,z)=0\ ,$$
$$\left[\frac{\partial S}{\partial u}\right]_z \neq 0\ , \tag{8}$$
$$S(-u,z)=S(u,z)\ ,$$

where the second two conditions are for all u and z. The first two conditions are satisfied if we require that $S(u,z)$ be such that

$$\lim_{u\to 0}\left[\frac{\partial S}{\partial u}\right]=\infty \tag{9}$$

and never have a zero slope.

B. Experimentally measured response

We describe graphically the different observations *in vivo* that are feasible with respect to time during the course of the deformation of the artery wall and of the evolution of the blood speed at the same point of the cardiovascular system. These curves have been deduced from measurements taken in the ascending aorta at the point of the heart (from Fry, Mallos, and Caspert [20]). The curve $S=S_0(t)$ [Fig. 1(a)] combined with the curve $u=u_0(t)$ [Fig. 1(b)] provides a means of obtaining the rate of deformation of the artery wall $\Delta S/S$ as a function of the value of the velocity of the blood at the point of the heart (see Fig. 2). The first part of this curve, extending from point A to point B, corresponds to the combined response of the system consisting of the heart and artery, whereas the second part, extending from point B to point A through point C, corresponds to the natural response of the wall when the heart no longer exerts any pressure. It is this second part of the curve that we use in our present development. It is important to note that we have neglected any contribution associated with relaxation structure occurring in the artery after completion of the cardiac cycle. This corresponds to the section of the curve shown in Fig. 2 that is contained within the dashed circle.

A simple function giving the fraction of deformation which satisfies the three mathematical requirements cited above and which provides a correct interpolation, in the first approximation, of the experimental curve extending from point B to C and then to A is

$$\frac{\Delta S}{S}(u,z)=\frac{S-S_0}{S_0}=D(z)\left|\frac{u}{u_0}\right|^{\beta}\ , \tag{10}$$

where $0<\beta<1$ and β is a dimensionless number representing the curvature of the experimental curve. The quantities $D(z)$ and u_0 are adjustable parameters representing changes in the physical properties of each portion of the artery, e.g., evolution of Young's modulus.

The evolution of Young's modulus of the artery wall with distance is implicitly connected to the function $D(z)$ because it corresponds to the maximum deformation at a given position when the blood velocity has the maximum value u_0. We know from measurements that the maximum change in section of the artery wall is about 20% in the ascending aorta and 10% in the abdominal aorta. Therefore, in order to take into account the stiffening of the artery wall, we have adapted a function of the form

$$\frac{\Delta S}{S}(u,z)=D_0(1-\lambda z)\left|\frac{u}{u_0}\right|^{\beta}\ , \tag{11}$$

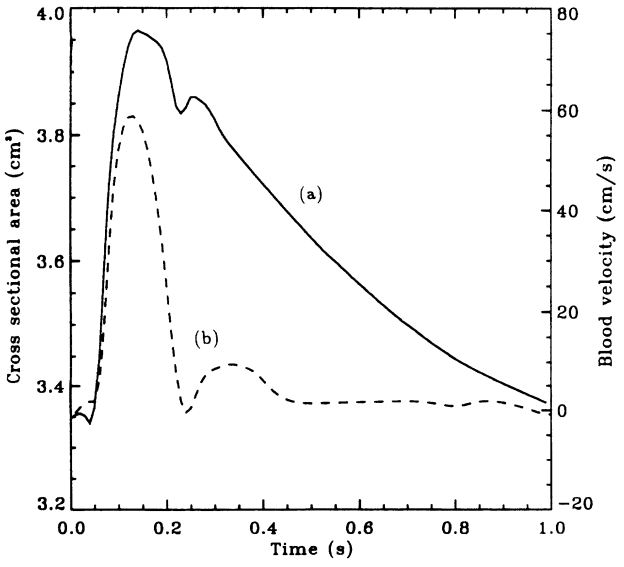

FIG. 1. (a) Cross-sectional area variations in the ascending aorta. The data were obtained from measurements by Fry, Mallos, and Casper [20]. (b) Blood velocity profile in the ascending aorta during one cardiac cycle.

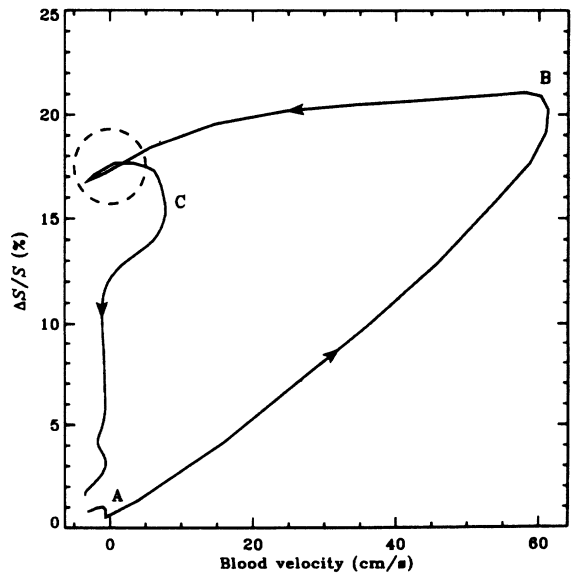

FIG. 2. Dynamic relationship between blood velocity and rate of deformation of the ascending aorta.

where $D_0=0.2$, $u_0=62$ cm/s, and $\beta=0.4$ in the ascending aorta and where λ is a parameter representing the variation of Young's modulus ($\lambda\approx 1$ m^{-1} in man). It follows that the deformation-velocity relation $S(u,z)$ is

$$S(u,z)=S_0(z)\left[1+\frac{\Delta S}{S}(u,z)\right]$$
$$=S_0 e^{-mz}\left[1+D_0(1-\lambda z)\left|\frac{u}{u_0}\right|^{\beta}\right]. \quad (12)$$

Combining Eqs. (12) and (6), we obtain

$$\frac{\partial u}{\partial t}+u\frac{\partial u}{\partial z}=-f(u,z)\left[\frac{\partial u}{\partial z}+[g(u,z)-m]u\right], \quad (13)$$

where

$$f(u,z)=\left[\frac{u_0\left|\frac{u}{u_0}\right|^{1-\beta}}{\beta D_0(1-\lambda z)}+\frac{|u|}{\beta}\right]\mathrm{sgn}(u)$$

and

$$g(u,z)=\frac{-\left|\frac{u}{u_0}\right|^{\beta}}{1+D_0(1-\lambda z)\left|\frac{u}{u_0}\right|^{\beta}}D_0\lambda .$$

Equation (13) shows that the parameter of conicity m and the parameter of change in Young's modulus λ appear in the same term, showing that these two effects play approximately the same role in the velocity evolution.

IV. RESULTS OF CALCULATIONS

Equation (13) governs the velocity pulse propagation along the artery. Its solution is effected numerically via first order finite-difference integration. At the boundary corresponding to the root of the aorta ($z=0$ in the tube), we impose the ejection pattern of the left ventricle in the form of the blood velocity $u_0(t)$, whereby the shape of the curve is assumed to be equal to the one given by Fry, Mallos, and Casper [20].

Our model was used to investigated pulse propagation in a human arterial conduit which extends from the root of the aorta to the end of the abdominal aorta, corresponding to 50 cm of propagation. In order to obtain information related to blood pressure, we used the pressure-radius relation given by Olsen and Shapiro [21]. This relation, expressed in a form that includes the nonlinear elastic properties of the wall, is

$$P=P_0+\frac{h_0}{R_0}E(z)\left[\frac{R_0}{R}-\frac{R_0^2}{R^2}\right]\left[1+a\left[\frac{R}{R_0}-1\right]\right], \quad (14)$$

where h_0 denotes the thickness of the wall when the tube radius R has the equilibrium value R_0 and the pressure is the diastolic pressure P_0. In the above relation, E and a are Young's modulus and the nonlinear coefficient of elasticity, respectively. For the ratio h_0/R_0 and for a, we adopt the mean values $h_0/R_0=0.12$ and $a=1.95$ proposed by Yomosa [6], and the evolution of Young's modulus is assumed to be $E(z)=40(1+2\lambda z)$ N/cm^2 (from Pedley [3]). Results obtained for arteries with different mechanical properties are as follows.

A. Artery with constant Young's modulus and no conicity

For this case $m=0$ and $\lambda=0$. These parameter values correspond to an artery that has the same properties everywhere. As a result, the deformation function $S(u,z)$ does not have a dependence on the distance z. Changes in pressure and velocity corresponding to pulse propagation are shown in Fig. 3. We observe a decrease in the maximum blood velocity and a reduction of the pulse wave celerity from approximately 4 m/s at the beginning of the tube to 3 m/s at $z=50$ cm. Furthermore, these two effects are accompanied by a decrease in amplitude of the pressure pulse during its propagation.

B. Artery with increasing Young modulus and no conicity

For this case $m=0$ and $\lambda=1$ m^{-1}. Results for this case are shown in Fig. 4. We note that a significant difference between this case and the previous case is the typical "peaking" of the pressure pulse associated with the acceleration of the wave. For example, the pulse celerity is about 4 m/s at the heart and 8 m/s in the abdominal aorta, which is in perfect correlation with the experimental measurements. These two behaviors are in accordance with the rapid development of a steep front. This demonstrates the importance of Young's modulus and its variation along the arterial tree for blood-pressure–wave evolution.

C. Artery with conicity and increasing Young's modulus

For this case $m=2$ and $\lambda=1$ m^{-1}. Results for this case are shown in Fig. 5. An important observation in this case is the relatively large increase in maximum blood velocity which causes a very large increase in pressure amplitude with z. This increase in maximum velocity, however, is understandable because of the reduction of artery diameter. All characteristics occurring in the cardiovascular system are present in this case except bifurcations. The presence of bifurcations causes the flow to lose blood volume at each level of branching. This loss of volume results in a loss of the fluid kinetic energy, $e=\frac{1}{2}\rho u^2$, which in turn results in a decrease in the maximum blood velocity. This result supports the conclusion that the role played by the taper effect of the artery is to counterbalance the effect of bifurcations and thus minimize the decrease in flow energy (or fluid velocity) in order to maintain a nonzero blood velocity everywhere.

V. DISCUSSION AND CONCLUSION

In this paper we present a model of blood motion based on a monovariable representation of both the arteries and their properties. This model permits the inclusion of information about the elastic response and geometric prop-

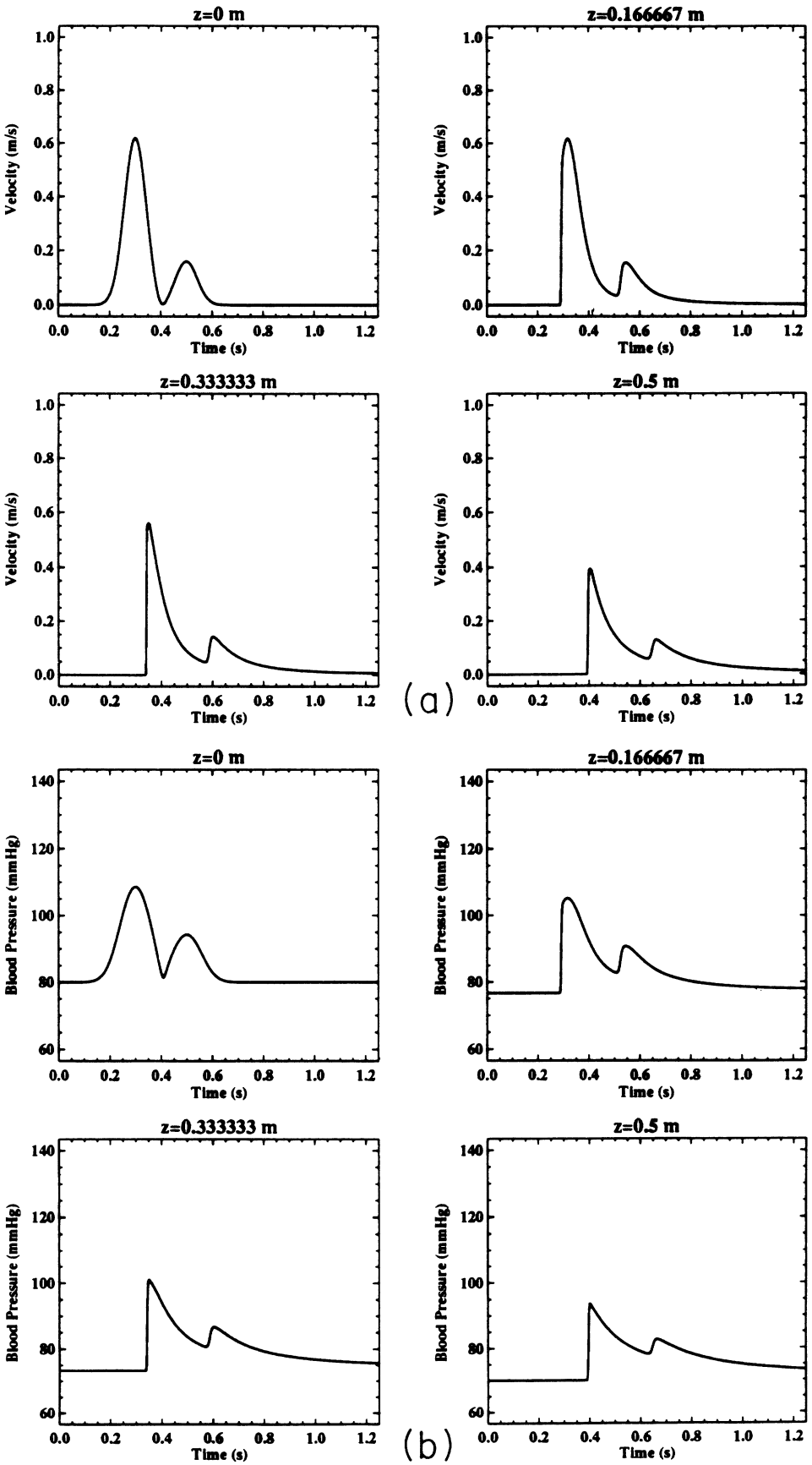

FIG. 3. Calculated (a) velocity and (b) pressure profiles at different positions along the aorta for an artery with constant radius and Young's modulus. $z=0$ is the position of the aortic valve.

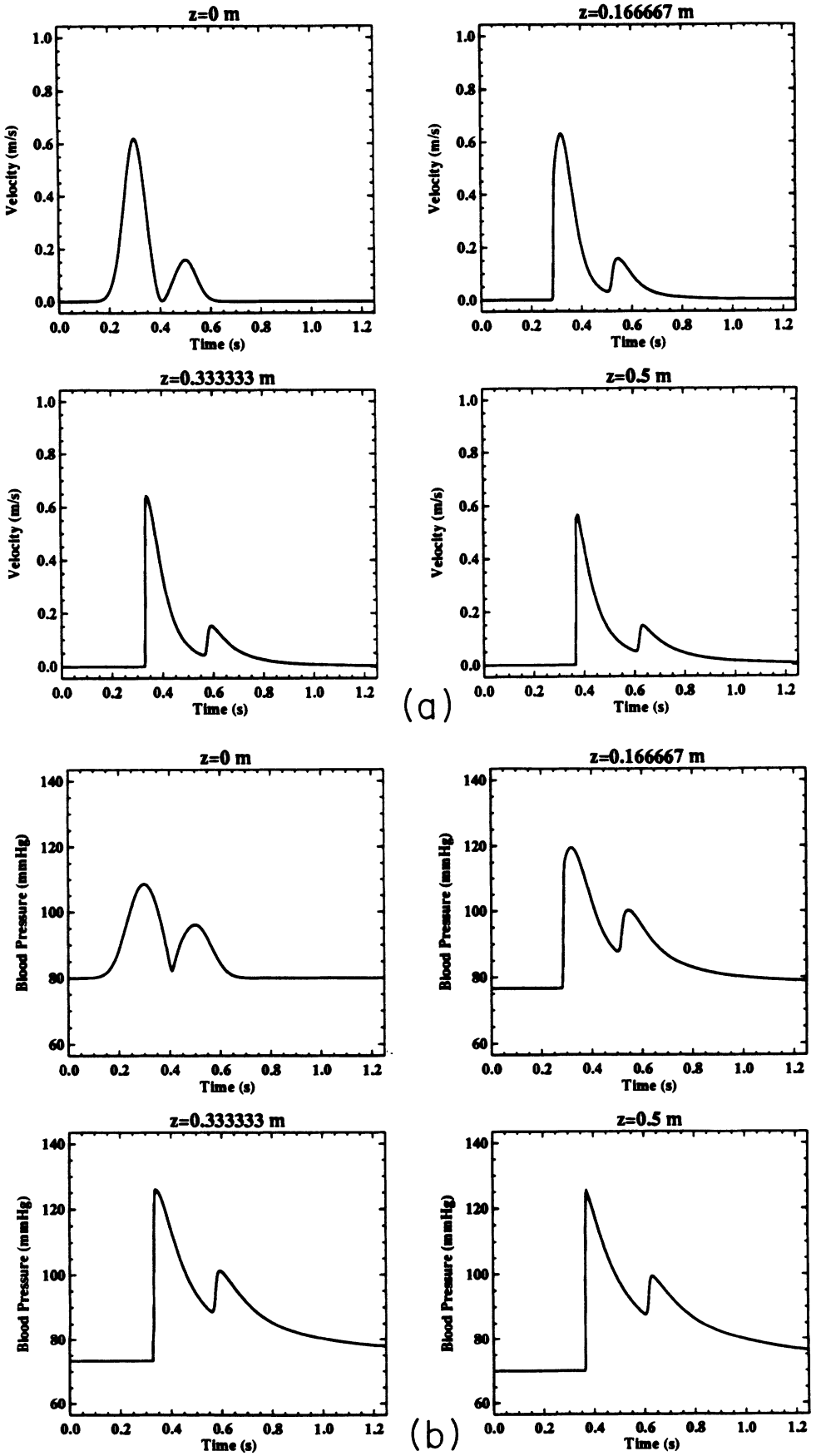

FIG. 4. (a) Velocity and (b) pressure calculated for an artery with constant radius and with an increasing Young's modulus. Note the steepening of the wave front and peaking of the pulses during propagation.

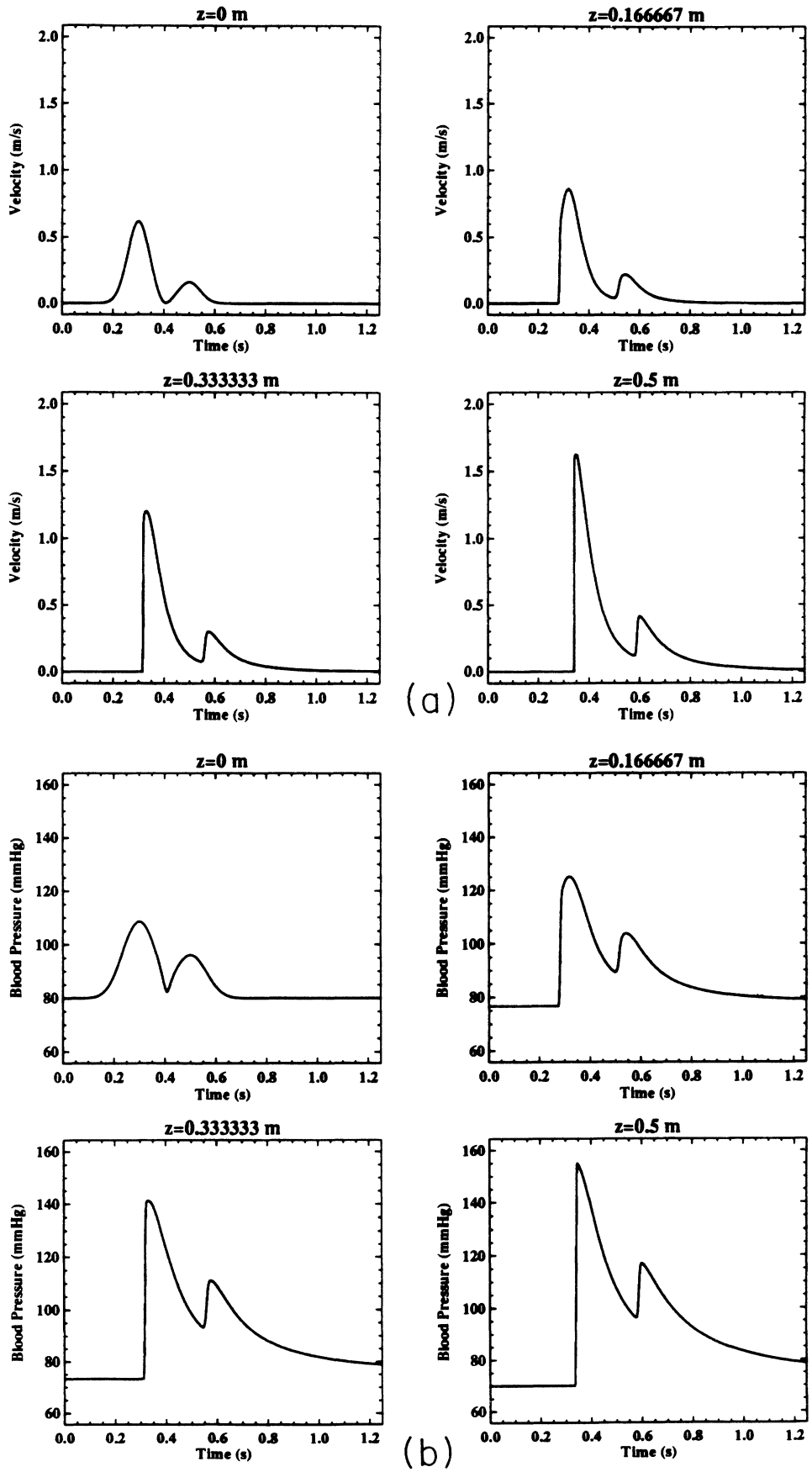

FIG. 5. (a) Velocity and (b) pressure calculated for a cone-shaped artery with increasing Young's modulus.

erties of the arteries via a deformation function $S(u,z)$. Using our model, we have simulated several effects that are present *in vivo* such as the increase in amplitude of the pressure pulse and the acceleration of the wave. In addition, we observe in our simulations the creation of a steep wave front. This result is consistent with the results of other experimental and numerical studies [1,3,9,16,22,23] where steep wave fronts have been observed. These studies, however, have not given any explanation of the underlying physical mechanism for generation of this effect. Our simulations show that this steepening of the front occurs even in a tube with a constant diameter and Young's modulus. Moreover, the degree of this effect increases with the wave celerity and the stiffening of the vessel walls (see Figs. 4 and 5). This result implies that steepening of the wave front in the artery is due to the nonlinear elastic properties of the artery walls. Furthermore, it appears clearly that the pressure and blood velocity pulses do not have the solitonlike behavior proposed by Hashizume [18,19] and Yomosa [6]. This follows because of their continuous change in shape and amplitude as they travel from the heart.

Our results demonstrate that the shapes of these pulses are determined by the geometry and elastic properties of the arteries. In particular, we note the following.

It is the elasticity of the artery wall which causes the wave. If we assume a rigid tube where there is no time dependence of the cross section, i.e., $S(z,t)=S_0(z)$, it follows from the continuity equation that

$$u(z,t)=u_0(t)/S_0(z)\neq u(z-ct)\ . \tag{15}$$

The existence of a solution of the form $u(z-ct)$ is biologically necessary because the heart is a pulsatory pump and not a continuous one.

There is a strong dependence of the value of the blood pressure and wave celerity on the value of Young's modulus and its variation with distance. This follows since, according to the classical Moens-Korteweg relation for a circular cylindrical tube [1,3], the wave celerity c is given by

$$c=\left[\frac{Eh}{2\rho_0 r}\right]^{1/2}, \tag{16}$$

where E is Young's modulus, r the internal radius, ρ_0 the density of the wall, and h the wall thickness.

There is an equivalence between both the taper effect and the increase in Young's modulus with respect to their influence on the velocity field. The influence of the taper effect is large relative to the other influences. This implies that the purpose of this effect is to counterbalance the effect of the loss of blood volume due to branching. A more detailed analysis of blood motion must therefore consider the construction of a deformation function which takes the effect of branching into account.

The fact that we have neglected to include the influence of relaxation phenomena in the construction of the deformation-velocity relation seems to have no effect on the velocity-field evolution. Because of this approximation, however, the shape of the pressure pulse shows a sharp-spike structure in contrast to the local minimum which occurs in biological measurements.

Finally, it is important to stress that a major limitation for our model is the availability of detailed experimental measurements providing information for the construction of a quantitative deformation function. With respect to this point, it is necessary to investigate the possibility of making clinical measurements that are more directly linked to the dynamic properties of the cardiovascular system rather than indirect measurements such as blood pressure.

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