Reflection and transmission of nonlinear blood waves due to arterial branching

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An approach is proposed to understand the effect of arterial branching in large arteries. Reflected and transmitted nonlinear waves at the arterial branching are constructed from incident waves analytically. Fission and reflection of a soliton due to this discontinuity are explicitly shown in the lowest order. The conclusion can be drawn that the reflection due to arterial branching can be determined by the parameter values at the arterial branching, but two solitons will be transmitted in each branch artery from one incident soliton whose wave form and velocity remain approximately unchanged. [S1063-651X(97)11101-1]

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INTRODUCTION

The most striking feature of arterial blood flow is its pulsatile character. The intermittent ejection of blood into the aorta from the left ventricle of the heart produces flow and pressure pulses in the arterial tree. Experimental studies of these pulses have revealed that they propagate with a characteristic pattern. They undergo well-defined changes in their wave form as they propagate away from the heart [1,2]. The transmission of the pressure pulse is accompanied by an increase in amplitude and a decrease in pulse width, which have been noted as ''peaking'' and ''steepening,'' respectively. The increase in amplitude is combined with the development of a dicrotic wave.

Many works on blood motion deal with linearized models [1,2]. These theories have been constructed with certain assumptions. First, the nonlinear convective terms in the equation of motion of the fluid are totally neglected. One reason for expecting these to be small is that the mean flow velocity is usually less than 10% of the wave velocity. However, the condition that justifies neglecting the nonlinear terms cannot be assumed satisfactorily in the case of arterial pulse waves with large amplitude. From the measurements of flow pulses in the artery of a dog, it is known that the pulse wave velocity is about the order of 5×10^2 cm/sec, the maximum velocity or the amplitude of flow pulse is about 10^2 cm/sec, and the time τ which is required for the flow velocity to increase from zero to the maximum is about 5×10^{-2} sec. Then the ratio of the maximum contribution of the nonlinear term to that of the linear term is estimated to be about 0.2. This suggests that it may be a crucial fault in describing the pulsatile motion of the arterial blood to neglect the nonlinear convective terms. Second, it is assumed in linearized theories that the distortion of the vessel wall is a small percentage of the radius of the vessel. In systemic arteries, the change in radius during any one cardiac cycle is usually less than 4%, but the accompanying change in cross-sectional area may not be negligible. Third, linear elastic properties of vessel walls are usually assumed in these theories. But the modulus of elasticity of arterial vessels varies quite strongly with pressure. The omission of this effect may cause another serious problem with the linearized theories.

In 1960 McDonald measured simultaneously changes in amplitude and wave form of blood pressure at five sites from the ascending aorta to the saphenous artery in a dog (Fig. 1). Because it had been assumed that blood is an ideal fluid and an artery is an elastic tube, the dynamical equation of pressure wave for this system is the KdV equation [3–5]. As is well known, for a KdV equation, a given initial wave profile decomposes in the course of propagation into separate solitons and usually a small amount of so-called radiation (an oscillatory tail not containing any solitons) that will be attenuated during the propagation. Although Fig. 1 can be well interpreted by linear theory [1,2], it suggests a possible interpretation in terms of solitons, so we assume that each blood pulse will evolve into one larger soliton and another smaller one after the blood pulse propagates into the abdominal aorta. The soliton amplitude will change slightly due to the inhomogeneity of the blood vessel wall (variation of radius and Young's modulus of the artery with the propagation distance), which has been studied previously [6,7]. KdV-type solitons in an inhomogeneous medium have been studied extensively [8,9], for example, nonlinear waves in nonlinear lattices whose masses of particle are not uniform [10], shal-

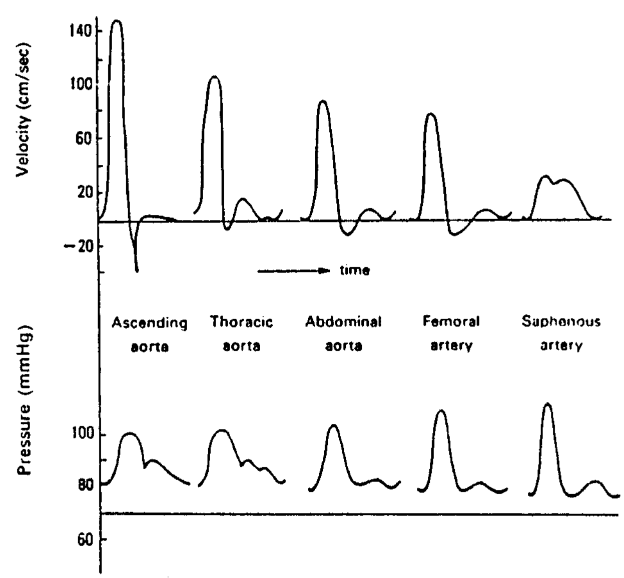

FIG. 1. A diagrammatic comparison of the behavior of the flow velocity and pressure pulse.

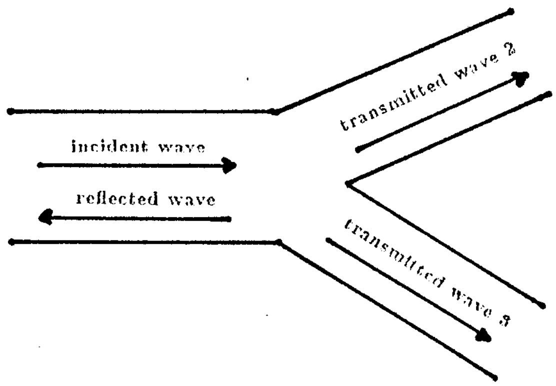

FIG. 2. Reflection and transmission of the nonlinear blood wave at the arterial branching.

low water waves with uneven bottom [14], etc. In general, we only consider the transmission and the reflection of the larger soliton, because the smaller one can be studied in much the same way.

Recently, Yomosa studied the motion of weakly nonlinear pressure waves in a thin nonlinear elastic tube filled with an incompressible fluid. He showed that the dynamics can be governed by the KdV equation [5]. On the other hand, in order to understand the principal feature of blood flow and the role of the different types of structures that influence blood motion, Paquerot and Remoissent studied the propagation of pressure and wall displacement pulses in a large elastic artery with the presence of both dispersion and variation of the radius and Young's modulus of the artery [6]. However, without considering the arterial branching effect, they did not know how the arterial branching affects the propagation of nonlinear blood waves. In order to elucidate the transmission and the reflection of blood flow at the arterial branching, a general method is proposed to understand nonlinear wave reflection and transmission from incident waves in a one-dimensional fluid-filled elastic tube at the branching (see Fig. 2). The wave equation of reflection and transmission are given analytically. The contribution of both blood viscosity and nonlinearity has been well studied [1], but we neglected blood viscosity in this paper because the viscous effects are mainly confined to the boundary layers, whose thickness is much less than the vessel radius. It can be estimated that the thickness of the boundary layers is about 10^{-2} cm by the equation of $\sqrt{\eta/\rho_0\omega}$ if we take the parameter values as η=0.012P, ρ_0=1.05 g/cm^3 (see Ref. [11]), a=0.5 cm (large artery), and $\omega\sim$10/s (there are only two pulses per second). Compared with vessel radius, the boundary layer is much less. We will not consider the boundary layer since it is so small, but we will consider the blood flow at the center of the artery, which includes almost the total cross section of the artery and of course nearly almost all of the blood flow for a large artery. The maximum and minimum values of blood velocity are approximately 100 cm/sec and 0, respectively (see Fig. 1), and there are only about two pulses per second, therefore the contribution of viscosity is estimated by $\eta(1/r)(\partial u/\partial r)\sim10^{-2}\times1/0.5\times100/0.5\sim4$ (neglecting the boundary layer contribution), and the contribution of nonlinearity is estimated by $\rho_0 u(\partial u/\partial x)\sim1.05\times100\times100/50\sim200$. The nonlinear effect is more important than the viscous effect in a large artery ($a\sim$0.5 cm) if we only consider the majority of blood flow at the center of the artery. The interesting results are that the incident wave can be transmitted into each bifurcation without changing, or nearly without changing, the amplitude, however sometimes there is substantial reflection, but often it is so small that it can be neglected. The parameter values at the arterial bifurcations determine whether there is substantial reflection or not.

EQUATION OF MOTION

By virtue of the anatomical geometry, the pulse can be represented by a one-dimensional wave. Following previous authors [5,6,12], we assume that blood can be regarded as an incompressible and inviscous fluid. Further, our model assumes that arteries are uniform inhomogenous cylindrical tubes having nonlinear elasticity. The laws of hydrodynamics governing the transport of an inviscous and incompressible fluid are the conservation of mass and the momentum equation, given, respectively, by

$$\frac{\partial A}{\partial t}+\frac{\partial(Av)}{\partial x}=0, \tag{1}$$

$$\frac{\partial v}{\partial t}+v\,\frac{\partial v}{\partial x}=-\frac{1}{\rho_0}\,\frac{\partial P}{\partial x}. \tag{2}$$

A third equation describing the radial motion of the wall under the forces exerted by the fluid is necessary in order to have a complete specification of the system [5],

$$\rho_w h\,\frac{\partial^2 R}{\partial t^2}=P-P_e-\frac{h}{R}\,\sigma, \tag{3}$$

where ρ_w is the density of the blood artery (ρ_w=1.05 g/cm^3). P_e the external pressure, σ the extending stress in the tangential direction, and h the thickness of the tube of radius $R(x,t)$. We can suppress the unknown pressure P_e if we consider an artery already inflated at the diastolic pressure P_0 with radius a, thickness h_0, and with the equilibrium relation $P_e=P_0-(h_0/a)\sigma^0$. Only considering weak waves, i.e., $A-A_0=\pi R^2-\pi a^2\approx2\pi a(R-a)$ and defining the small radial elongation of the arterial wall $\gamma=(R-a)/a$ and the differential pressure $p=P-P_0$, it follows from Eq. (3) that

$$\frac{\rho_w h}{2\pi R_0}\,\frac{\partial^2(A-A_0)}{\partial t^2}=p-\frac{h_0 a}{R^2}\,\sigma', \tag{4}$$

where $\sigma'=\gamma E(1+\alpha\gamma)$, E is Young's modulus, and α is the coefficient of nonlinear elasticity. It is assumed that the wall is incompressible, i.e., $Rh=R_0h_0$.

Introducing the dimensionless quantities through the definitions $A=\pi a^2A'$, $p=p_0p'$, $t=Tt'$, $x=Lx'$, $v=(L/T)v'$, $A_0=\pi a^2$, $L^2=\rho_w ah/2\rho_0$, $p_0=Eh/2a$, $T^2=\rho_w a^2/E$, we then have the dimensionless equations from Eqs. (1), (2), and (4),

$$\frac{\partial A'}{\partial t'}+\frac{\partial(A'v')}{\partial x'}=0, \tag{5}$$

$$\frac{\partial v'}{\partial t'}+v'\frac{\partial v'}{\partial x'}=-\frac{\partial p'}{\partial x'}, \tag{6}$$

$$p'=\frac{2}{1+A'}\frac{\partial^2 A'}{\partial t'^2}+(A'-1)\frac{2(2-\alpha+\alpha A')}{(1+A')^2}. \tag{7}$$

Now we consider the model in which the cylinder tube has one branch at the region $x<0$ and two branches at the region $x>0$. The wall of the cylinder tube has a jump of a, ρ_w, h_0, and E at $x=0$ (Fig. 2). The variables of one branch at $x>0$ are represented by p_2, v_2, A_2, etc., and the others at $x>0$ are represented by p_3, v_3, A_3, etc. Those at $x<0$ are represented by p, v, A, etc.

For $x>0$ we only consider the transmitted waves and introduce new independent variables

$$\xi=\epsilon(x'-t'), \tag{8}$$

$$\eta=\frac{\epsilon^3}{2}x'. \tag{9}$$

Introducing the perturbation expansions

$$p_2'=\epsilon^2 p_{21}^t+\epsilon^4 p_{22}^t+\cdots, \tag{10}$$

$$A_2'=1+\epsilon^2 A_{21}^t+\epsilon^4 A_{22}^t+\cdots, \tag{11}$$

$$v_2'=\epsilon^2 v_{21}^t+\epsilon^4 v_{22}^t+\cdots, \tag{12}$$

$$p_3'=\epsilon^2 p_{31}^t+\epsilon^4 p_{32}^t+\cdots, \tag{13}$$

$$A_3'=1+\epsilon^2 A_{31}^t+\epsilon^4 A_{32}^t+\cdots, \tag{14}$$

$$v_3'=\epsilon^2 v_{31}^t+\epsilon^4 v_{32}^t+\cdots, \tag{15}$$

substitution of Eqs. (8)–(12) into Eqs. (5)–(7) yields at $O(\epsilon^2)$

$$p_{21}^t=A_{21}^t=v_{21}^t. \tag{16}$$

At $O(\epsilon^4)$ we can get

$$\frac{\partial v_{21}^t}{\partial\eta}+(1+\alpha)v_{21}^t\frac{\partial v_{21}^t}{\partial\xi}+\frac{\partial^3 v_{21}^t}{\partial\xi^3}=0. \tag{17}$$

Similarly it can be obtained

$$\frac{\partial v_{31}^t}{\partial\eta}+(1+\alpha)v_{31}^t\frac{\partial v_{31}^t}{\partial\xi}+\frac{\partial^3 v_{31}^t}{\partial\xi^3}=0, \tag{18}$$

$$p_{31}^t=A_{31}^t=v_{31}^t. \tag{19}$$

For $x<0$ we need to consider both incident and the reflected waves. For this purpose we introduce the following transformations of independent and dependent variables:

$$\xi=\epsilon(x'-t'), \tag{20}$$

$$\tau=\epsilon(x'+t'), \tag{21}$$

$$\eta=\frac{\epsilon^3}{2}x', \tag{22}$$

and the perturbation equations

$$p'=p'(\xi,\tau,\eta)=\epsilon^2 p_1+\epsilon^4 p_2+\cdots, \tag{23}$$

$$v'=v'(\xi,\tau,\eta)=\epsilon^2 v_1+\epsilon^4 v_2+\cdots, \tag{24}$$

$$A'=A'(\xi,\tau,\eta)=1+\epsilon^2 A_1+\epsilon^4 A_2+\cdots. \tag{25}$$

Substitution of Eqs. (20)–(25) into Eqs. (5)–(7) yields at $O(\epsilon^2)$

$$-\frac{\partial A_1}{\partial\xi}+\frac{\partial A_1}{\partial\tau}+\frac{\partial v_1}{\partial\xi}+\frac{\partial v_1}{\partial\tau}=0, \tag{26}$$

$$-\frac{\partial v_1}{\partial\xi}+\frac{\partial v_1}{\partial\tau}+\frac{\partial p_1}{\partial\xi}+\frac{\partial p_1}{\partial\tau}=0, \tag{27}$$

$$p_1=A_1. \tag{28}$$

Considering the equilibrium condition of $p=0$ when $v=0$, we can obtain the following equations:

$$A_1=A_1^I(\xi,\eta)+A_1^R(\tau,\eta), \tag{29}$$

$$p_1=p_1^I(\xi,\eta)+p_1^R(\tau,\eta), \tag{30}$$

$$v_1=v_1^I(\xi,\eta)+v_1^R(\tau,\eta), \tag{31}$$

and

$$A_1^I=p_1^I=v_1^I, \tag{32}$$

$$A_1^R=p_1^R=-v_1^R. \tag{33}$$

Similarly at $O(\epsilon^4)$ we can get

$$-\frac{\partial A_2}{\partial\xi}+\frac{\partial A_2}{\partial\tau}+\frac{\partial(A_1v_1)}{\partial\xi}+\frac{\partial v_2}{\partial\xi}+\frac{\partial v_2}{\partial\tau}+\frac{\partial(A_1v_1)}{\partial\tau}+\frac{1}{2}\frac{\partial v_1}{\partial\eta}=0, \tag{34}$$

$$-\frac{\partial v_2}{\partial\xi}+\frac{\partial v_2}{\partial\tau}+v_1\frac{\partial v_1}{\partial\xi}+v_1\frac{\partial v_1}{\partial\tau}+\frac{\partial p_2}{\partial\xi}+\frac{\partial p_2}{\partial\tau}+\frac{1}{2}\frac{\partial p_1}{\partial\eta}=0, \tag{35}$$

$$p_2=A_2+\frac{\alpha-2}{2}+\frac{\partial^2 A_1}{\partial\xi^2}+\frac{\partial^2 A_1}{\partial\tau^2}-2\frac{\partial^2 A_1}{\partial\xi\partial\tau}, \tag{36}$$

considering that $A_1v_1=A_1^Iv_1^I+A_1^Rv_1^R$, which can be given from Eqs. (29)–(33) and rewriting Eqs. (34)–(36),

$$2\frac{\partial A_2}{\partial\tau}+2\frac{\partial v_2}{\partial\tau}+\frac{\partial(A_1^Iv_1^I)}{\partial\xi}+\frac{\partial(A_1^Rv_1^R)}{\partial\tau}+\frac{1}{2}\frac{\partial v_1^I}{\partial\eta}+\frac{1}{2}\frac{\partial v_1^R}{\partial\eta}+\frac{\partial^3 A_1^I}{\partial\xi^3}+\frac{\partial^3 A_1^R}{\partial\tau^3}+v_1\frac{\partial v_1^I}{\partial\xi}+v_1\frac{\partial v_1^I}{\partial\tau}+(\alpha-2)A_1\frac{\partial A_1^I}{\partial\xi}+(\alpha-2)A_1\frac{\partial A_1^R}{\partial\tau}+\frac{1}{2}\frac{\partial p_1^I}{\partial\eta}+\frac{1}{2}\frac{\partial p_1^R}{\partial\eta}=0, \tag{37}$$

$$-2\frac{\partial A_2}{\partial\xi}+2\frac{\partial v_2}{\partial\xi}+\frac{\partial(A_1^Iv_1^I)}{\partial\xi}+\frac{\partial(A_1^Rv_1^R)}{\partial\tau}+\frac{1}{2}\frac{\partial v_1^I}{\partial\eta}+\frac{1}{2}\frac{\partial v_1^R}{\partial\eta}-\frac{\partial^3A_1^I}{\partial\xi^3}-\frac{\partial^3A_1^R}{\partial\tau^3}-v_1\frac{\partial v_1^I}{\partial\xi}-v_1\frac{\partial v_1^R}{\partial\tau}-(\alpha-2)A_1\frac{\partial A_1^I}{\partial\xi}-(\alpha-2)A_1\frac{\partial A_1^R}{\partial\tau}-\frac{1}{2}\frac{\partial p_1^I}{\partial\eta}-\frac{1}{2}\frac{\partial p_1^R}{\partial\eta}=0. \tag{38}$$

From Eqs. (32) and (37) we can obtain

$$\frac{\partial v_1^I}{\partial\eta}+(\alpha+1)v_1^I\frac{\partial v_1^I}{\partial\xi}+\frac{\partial^3v_1^I}{\partial\xi^3}=0 \tag{39}$$

if we set

$$2\frac{\partial A_2}{\partial\tau}+2\frac{\partial v_2}{\partial\tau}+\frac{\partial(A_1^Rv_1^R)}{\partial\tau}+\frac{\partial^3A_1^R}{\partial\tau^3}+v_1^R\frac{\partial v_1^I}{\partial\xi}+v_1^I\frac{\partial v_1^R}{\partial\tau}+v_1^R\frac{\partial v_1^R}{\partial\tau}+(\alpha-2)A_1^R\frac{\partial A_1^I}{\partial\xi}+(\alpha-2)A_1\frac{\partial A_1^R}{\partial\tau}=0. \tag{40}$$

From Eqs. (33) and (38) we can obtain

$$\frac{\partial v_1^R}{\partial\eta}-(\alpha+1)v_1^R\frac{\partial v_1^R}{\partial\tau}+\frac{\partial^3v_1^R}{\partial\tau^3}=0 \tag{41}$$

if we set

$$-2\frac{\partial A_2}{\partial\xi}+2\frac{\partial v_2}{\partial\xi}+\frac{\partial(A_1^Iv_1^I)}{\partial\xi}-\frac{\partial^3A_1^I}{\partial\xi^3}-v_1^I\frac{\partial v_1^I}{\partial\xi}-v_1^R\frac{\partial v_1^I}{\partial\xi}-v_1^I\frac{\partial v_1^R}{\partial\tau}-(\alpha-2)A_1^I\frac{\partial A_1^R}{\partial\tau}-(\alpha-2)A_1\frac{\partial A_1^I}{\partial\xi}=0. \tag{42}$$

Letting

$$v_{21}^t=\frac{-6}{1+\alpha}v_2^t, \tag{43}$$

$$v_{31}^t=\frac{-6}{1+\alpha}v_3^t, \tag{44}$$

$$v_1^I=\frac{-6}{1+\alpha}v^I, \tag{45}$$

$$v_1^R=\frac{6}{1+\alpha}v^R, \tag{46}$$

we can rewrite Eqs. (17), (18), (39), and (41) as the KdV equations,

$$\frac{\partial v_2^t}{\partial\eta}-6v_2^t\frac{\partial v_2^t}{\partial\xi}+\frac{\partial^3v_2^t}{\partial\xi^3}=0. \tag{47}$$

Similar equations can be given for v_3^t, v^I, and v^R.

CONSTRUCTION OF TRANSMITTED AND REFLECTED WAVES FROM INCIDENT WAVES

We examine the conditions for the continuity of pressure and mass at $x=0$. Neglecting $O(\epsilon^4)$ quantities we have

$$k_1[p_1^I(-\epsilon t',0)+p_1^R(\epsilon t',0)]=k_2p_{21}^t(-\epsilon t',0), \tag{48}$$

$$k_2p_{21}^t(-\epsilon t',0)=k_3p_{31}^t(-\epsilon t',0), \tag{49}$$

$$a_1^2\left(\frac{k_1}{2\rho_0}\right)^{1/2}[v_1^I(-\epsilon t',0)+v_1^R(\epsilon t',0)]=a_2^2\left(\frac{k_2}{2\rho_0}\right)^{1/2}v_{21}^t(-\epsilon t',0)+a_3^2\left(\frac{k_3}{2\rho_0}\right)^{1/2}v_{31}^t(-\epsilon t',0), \tag{50}$$

where $k_1=E_1h_{10}/a_1$, $k_2=E_2h_{20}/a_2$, and $k_3=E_3h_{30}/a_3$.

Equations (48)–(50) can be rewritten in the following form:

$$v_2^t(-\epsilon t',0)=\frac{2}{k_2/k_1+\sqrt{k_2/k_1}(a_2/a_1)^2+k_2/\sqrt{k_3k_1}(a_3/a_1)^2}\times v^I(-\epsilon t',0), \tag{51}$$

$$v_3^t(-\epsilon t',0)=\frac{2}{k_3/k_1+\sqrt{k_3/k_1}(a_3/a_1)^2+k_3/\sqrt{k_2k_1}(a_2/a_1)^2}\times v^I(-\epsilon t',0), \tag{52}$$

$$v^R(-\epsilon t',0)=\frac{1-\sqrt{k_1/k_2}(a_2/a_1)^2-\sqrt{k_1/k_3}(a_3/a_1)^2}{1+\sqrt{k_1/k_2}(a_2/a_1)^2+\sqrt{k_1/k_3}(a_3/a_1)^2}\times v^I(-\epsilon t',0). \tag{53}$$

Let us consider one soliton solution of

$$\frac{\partial v^I}{\partial\eta}-6v^I\frac{\partial v^I}{\partial\xi}+\frac{\partial^3v^I}{\partial\xi^3}=0, \tag{54}$$

$$v^I=-2\frac{1}{d^2}\operatorname{sech}^2\left(\frac{\xi}{d}-\frac{4\eta}{d^2}\right), \tag{55}$$

where d denotes the width of the soliton.

From an initial condition

$$V(X,0)=AL^{-2}\operatorname{sech}^2\frac{X}{L} \tag{56}$$

for the KdV equation

$$V_T-6VV_X+V_{XXX}=0, \tag{57}$$

we can determine the number N of generated solitons by the inverse scattering method [13]. The number N is the maximum integer which satisfies

$$\sqrt{A+\tfrac{1}{4}}+\tfrac{1}{2}-N>0 \tag{58}$$

and the amplitude of the generated solitons is given by

$$2\left(\sqrt{A+\tfrac{1}{4}}+\tfrac{1}{2}-j\right)^2L^{-2} \tag{59}$$

$(j=1,2,3,\ldots,N)$.

Using these formulas we determine the amplitudes of reflected and transmitted solitons. From Eqs. (51), (52), (53), and (55) we can determine the initial conditions of v_2^t, v_3^t, and v^R,

$$v_2^t(-\epsilon t',0)=\frac{2}{k_2/k_1+\sqrt{k_2/k_1}(a_2/a_1)^2+k_2/\sqrt{k_3k_1}(a_3/a_1)^2}\times(-2d^{-2})\mathrm{sech}^2\left(\frac{-\epsilon t'}{d}\right), \tag{60}$$

$$v_3^t(-\epsilon t',0)=\frac{2}{k_3/k_1+\sqrt{k_3/k_1}(a_3/a_1)^2+k_3/\sqrt{k_2k_1}(a_2/a_1)^2}\times(-2d^{-2})\mathrm{sech}^2\left(\frac{-\epsilon t'}{d}\right), \tag{61}$$

$$v^R(-\epsilon t',0)=\frac{1-\sqrt{k_1/k_2}(a_2/a_1)^2-\sqrt{k_1/k_3}(a_3/a_1)^2}{1+\sqrt{k_1/k_2}(a_2/a_1)^2+\sqrt{k_1/k_3}(a_3/a_1)^2}\times(-2d^{-2})\mathrm{sech}^2\left(\frac{-\epsilon t'}{d}\right). \tag{62}$$

It can be obtained that for the reflected wave the number N is 1 or 0. For the transmitted waves one or more solitons are always generated.

DISCUSSION AND CONCLUSION

We only study transmission and reflection of blood pulse waves for a dog. The parameter values at arterial bifurcations for a dog are approximately given to estimate reflection and transmission from incident waves. The experimental results tell us that the radius of an artery changes slowly along the propagation distance. The radius is assumed to have a weak exponential evolution, that is, $a=a_0e^{-mx}$, where m and a_0 are positive constants (see Ref. [11]). According to its experimental data, the approximate parameter values at arterial bifurcation of the abdominal aorta into the femoral arteries are given, respectively, as follows: $a_1=0.7$ cm, $h_1=0.05$ cm, $E_1=10\times10^6$ dyn/cm^2 (for the abdominal aorta), and $a_2=a_3=0.6$ cm, $h_2=h_3=0.04$ cm, $E_2=E_3=10\times10^6$ dyn/cm^2 (for the femoral aorta). The initial values of reflected and transmitted waves at the bifurcation in branches 2 and 3 are given from Eqs. (51), (52), and (53), respectively, by

$$v_2^t(-\epsilon t',0)\approx0.8v^I(-\epsilon t',0), \tag{63}$$

$$v_3^t(-\epsilon t',0)\approx0.8v^I(-\epsilon t',0), \tag{64}$$

$$v^R(-\epsilon t',0)\approx-0.2v^I(-\epsilon t',0), \tag{65}$$

and then the amplitude of solitons propagating in branches 2 and 3 can be given by Eq. (59),

$$v_{2s}^t=v_{3s}^t\approx0.86v^I. \tag{66}$$

It can be concluded from Eq. (58) that there is only one transmitted soliton in each branch and the soliton amplitude is larger than that of the initial value for the transmitted waves. The reflection is substantial (20% of the incident waves). It has been well known that there are many small arterial bifurcations in the thoracic or abdominal aorta, and we now estimate the reflection and the transmission at these bifurcations. There is, for example, a renal artery in the abdominal aorta [11], for which the experimental data are given by $a_1=a_2\approx0.8$ cm, $E_1=E_2\approx10\times10^6$ dyn/cm^2 (for the abdominal aorta) and $a_3\approx0.2$ cm, $E_3\approx10\times10^6$ dyn/cm^2 (for the renal aorta). h_0/a is approximately a constant. The transmission and the reflection are given by Eqs. (51), (52), and (53) as follows:

$$v_2^t(-\epsilon t',0)\approx0.97v^I(-\epsilon t',0), \tag{67}$$

$$v_3^t(-\epsilon t',0)\approx0.97v^I(-\epsilon t',0), \tag{68}$$

$$v^R(-\epsilon t',0)\approx-0.03v^I(-\epsilon t',0), \tag{69}$$

and then the amplitude of solitons propagating in branches 2 and 3 can be given by Eq. (59),

$$v_{2s}^t=v_{3s}^t\approx0.98v^I, \tag{70}$$

but the reflection is negligibly small. We can conclude that sometimes the reflection can be neglected, but sometimes it cannot. The parameter values at the bifurcation determine whether there is substantial reflection or not.

It has been well known that the KdV equation is one of the typical nonlinear equations that have soliton solutions. Any separate pulses with constant or nearly constant wave form and velocity described by the KdV equation are solitons. We propose that transmitted waves are solitons, and this can explain why blood pulse waves can propagate into any artery, because solitons have this character. When blood pulses are transmitted at the bifurcations, their amplitudes are approximately unchanged (experimental results). This is consistent with soliton theory. These observations are also consistent with linear theory (see Ref. [1]). Actually, the amplitudes of blood pulses change slightly as they propagate away along the artery. This is due to the variation of the rest radius and Young's modulus of an artery or blood viscosity in a very small artery. These solitons are sometimes called quasisolitons. There have been many studies on so-called quasisolitons in one-dimensional inhomogeneous systems such as nonlinear lattices whose masses are not uniform [10], shallow water waves with an uneven bottom [14], etc. Sometimes no solitons can be measured when the arterial parameter values are not suitable. In this case, the artery is abnormal, and probably some information on disease can be obtained from the blood pulse waveform. Soliton theory can tell us whether the artery is in a normal condition by measuring the blood pulse wave form. If some vascular disease exists, the soliton amplitude and velocity will change, because the radius, thickness, and Young's modulus of an artery are changed by vascular disease.

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