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Effects of thin plaque on blood hammer—An asymptotic theory

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ABSTRACT

We describe a theory of blood hammer in a long and stiffened artery affected by the presence of plaque. Based on the model of a viscous fluid in laminar flow, we derive explicit expressions of oscillatory pressure and wall shear stress when part of the blood vessel is lined with thin plaque. The asymptotic method of multiple-scale expansions is utilized to obtain information on wave reflection and frictional damping analytically. The effects of plaque thickness and length on blood pressure and wall shear stress are studied.

1. Introduction

Blood hammer is a phenomenon known to occur in cerebral arteries, where sudden blockage by blood clots can stop circulation in capillaries, cause acute rise of blood pressure and lead to intracerebral hemorrhage [1,2].

It was first pointed out by Damsa et al. [1] that the fluid mechanics of blood hammer is similar to the classic problem of water hammer in a metal pipe when a valve is suddenly closed. Water hammer is a wave phenomenon arising from the compressibility of water and affected by turbulent wall friction [3–6]. Flow in small blood vessels is mostly laminar. Laboratory experiments for laminar water hammer have been performed by [7] who used a highly viscous fluid in a metal pipe. A theory accounting for laminar friction was given by [8], who solved formally the linearized Navier–Stokes equations by Laplace transform but used a numerical approximation to evaluate the inverse transform. Waves in blood hammer are due to the elasticity of the artery wall. A mechanistic theory has been recently given by [9] who described a computational model of blood hammer in a posterior cerebral artery of 8 mm length. It is known that blood hammer can also occur in the longer mid cerebral arteries of length 20 mm $< L <$ 80 mm [2]. Since the dynamic blood pressure leads to hoop tension stress σ according to $\sigma = pR_0/h$ where h is the wall thickness and R_0 is the mean vessel radius before distention, excessive hoop tension is a threat to hemorrhage and rupture of the vascular wall.

Besides the pressure, shear stress on the wall of blood vessels is known to be relevant to certain diseases [10–15] such as the formation of atherosclerotic plaques in carotid arteries [16,17]. Excessively high wall shear stress can cause arteriosclerosis lesion and rupture of aneurysm [18], and is the major cause of heart attack, stroke, and peripheral arterial disease [19]. Hence the prediction of shear stress arising from blood hammer is also of interest in hemodynamics.

Reflection of impulsive waves by extended blockage in pipes is of importance to water supply systems, as transient signals can be useful for the detection of constrictions [20,21]. In human physiology, significant changes of cross-sectional area due to the presence of plaque causes additional reflections in the middle. Local stiffening generated by a repaired aortic coarctation can cause wave reflections near the heart and is of relevance to arterial and cardiovascular health and diagnosis of cardiovascular risk [11]. On blood hammer, the present authors have recently considered the possible presence of local plaque formation [22]. The assumptions of laminar flow and linearity allow the use of the technique of multiple-scale asymptotics [23–26] to achieve analytical solution. As little is known about the rheology of the plaque, they only considered the effect of area reduction by a thin plaque and ignored its elasticity altogether. In this paper we wish to explore another idealized model where the plaque has the same elasticity as the original vessel wall, hence an increase of plaque thickness can alter the wave speed significantly and causes strong reflection. The multiple-scales method is modified to examine the dynamics of wave scattering and attenuation analytically. Numerical results are discussed to examine the influence of plaque thickness and length on both the blood pressure and wall shear stress caused by sudden blockage. In principle the predictions here based on a simple model can be checked by laboratory experiments using

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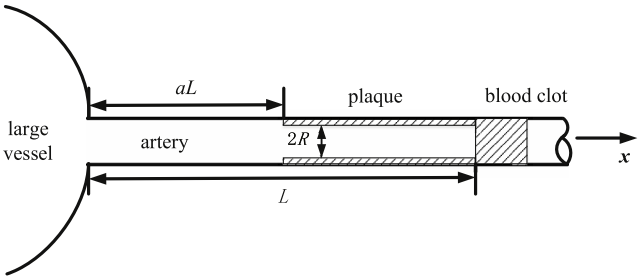

Fig. 1. Definition sketch.

artificial materials. Nevertheless further advance must wait for reliable knowledge of the wall rheology and the application of computational techniques to more realistic vascular systems. We offer this study as a preliminary step towards deeper theoretical understanding of blood hammer.

2. Equations governing the averaged flow

For convenience we summarize the linearized governing equations of fluid mass and momentum, under the assumption of small elastic distention of the wall, and negligible convective inertia of the fluid. Details of derivation can be found in [22]. Referring to Fig. 1, we consider a straight and elastic tube of inner radius $R(x, t)$ aligned with the x axis. Blood enters at $x = 0$ from a large vessel. At $t = 0$ the flow is blocked at $x = L$ abruptly. The entire artery is horizontal so that gravity has no effect.

Let $\bar{A}(x) = \pi\bar{R}^2$ be the interior cross-sectional area and $\bar{R}(x)$ the interior radius of the artery before distention. For small elastic deformation of the arterial wall, the conservation law of fluid mass is

$$\frac{1}{\rho\bar{C}^2}\frac{\partial p}{\partial t} + \frac{1}{\bar{A}}\frac{\partial(\bar{A}U)}{\partial x} = 0. \quad \bar{A} = \pi\bar{R}^2(x), \quad 0 < x < L \tag{2.1}$$

where p is the blood pressure, U the mean blood velocity and ρ the blood density. $\bar{C}$ is the Moens–Korteweg wave speed [27] which depends on Young's modulus E, the thickness h of the vessel wall, and the vessel radius $\bar{R}$ before distention,

$$\bar{C} = \sqrt{\frac{Eh}{2\rho\bar{R}}}. \tag{2.2}$$

Note that $\bar{C}(x)$ can vary in x through $h(x)$ and $\bar{R}(x)$. Unlike the hydraulic problem of water hammer in a metal pipe where the wave speed is the constant sound speed in water, here $\bar{C}$ depends not only on the wall elasticity E, but also the wall thickness h. In human arteries of uniform radius, $\bar{C}$ is normally less than 10 m/s [28]. However, it is known that $\bar{C}$ can be much higher owing to age-related increase of E [29]. According to Damsa et al. [1], a ten to thirty fold increase can be produced by sclerosis of the arteries, arterioles and capillaries, and by a powerful initial distention of the vascular wall, i.e., $\bar{C} = 100 \sim 300$ m/ s. Thus far no quantitative information of the rheological properties of the plaque is available. In [22], a simple assumption was adopted where the thin plaque is perfectly soft and merely reduces the vessel radius $\bar{R}$ by a small amount without affecting h. Analytical solution was found by a perturbation analysis, leading to approximate equations with constant coefficients, which were solved explicitly for the first two orders by Laplace transform. In this work we consider another limit where the plaque has the same elasticity as the artery wall. Hence the plaque not only decreases $\bar{R}$ but also increases h, leading to a significant increase of the wave speed C. Strong wave reflections can now occur if the vessel is only partially covered by plaque. A different method of analysis is employed here.

As the local blood velocity $u(x, r, t)$ is typically just a few centimeters per second, while the time and length scales are $t = O(L/\bar{C})$ and $x = O(L)$ respectively, convective inertia is quite negligible since

$$u\frac{\partial u}{\partial x}\Big/\frac{\partial u}{\partial t} = O\left(\frac{u}{\bar{C}}\right) \ll 1. \tag{2.3}$$

The area-averaged conservation law of mean fluid momentum reads

$$\rho\frac{\partial U}{\partial t} = -\frac{\partial p}{\partial x} + \frac{2\tau_w}{\bar{R}}, \tag{2.4}$$

where $U(x, t)$ is related to $u(x, r, t)$ by

$$U(x, t) = \frac{2\pi}{\pi\bar{R}^2}\int_0^{\bar{R}} u(x, r, t)\, r dr. \tag{2.5}$$

The wall stress τ_w

$$\tau_w = \rho\nu\frac{\partial u}{\partial r}\bigg|_{r=\bar{R}}, \tag{2.6}$$

will be determined later by a boundary-layer analysis. Combination of (2.1) and (2.4) yields the governing equation of the dynamic fluid pressure,

$$\frac{1}{\bar{A}}\frac{\partial}{\partial x}\left(\bar{A}\frac{\partial p}{\partial x}\right) - \frac{1}{\bar{C}^2}\frac{\partial^2 p}{\partial t^2} = \frac{2}{\bar{R}}\frac{\partial\tau_w}{\partial x}, \quad 0 < x < L, \ t > 0. \tag{2.7}$$

As is known in linear acoustics [30], wave radiation into the very large vessel due to the piston motion at $x = 0$ is negligible if $\bar{R}/L \ll 1$. Hence the dynamic pressure vanishes

$$p = 0, \qquad x = 0. \tag{2.8}$$

Let the initial flow velocity be U_0 just before the blockage, and is stopped by the blood clot at $x = L$ suddenly, then

$$\frac{\partial p}{\partial x} = -\rho_0\frac{\partial U}{\partial t} = \rho_0 U_0 D(t), \quad x = L, \ t > 0 \tag{2.9}$$

where $D(t)$ is an impulsive function. It has been reasoned in [2] that the blockage time must be less than $O(L/\bar{C})$ to cause blood hammer. We therefore choose the following simple model for $D(t)$ which has a sharp but finite peak over a very short duration $t_o \ll L/\bar{C}$,

$$D(t) = \frac{1}{t_o}\begin{cases} \dfrac{t}{t_o}, & 0 < t < t_o \\ 2 - \dfrac{t}{t_o}, & t_o < t < 2t_o \\ 0, & t > 2t_o. \end{cases} \tag{2.10}$$

Note that $D(t)$ has the property

$$\int_0^{2t_o} D(t)\, dt = 1. \tag{2.11}$$

At the edge of a plaque partially covering a vessel, we require the continuity of pressure and mass flux. In addition the following initial conditions are imposed:

$$p = \frac{\partial p}{\partial t} = 0, \quad t = 0, \ \ 0 < x < L. \tag{2.12}$$

The central task is to solve for $p(x, t)$, from which the flow velocity u, U and wall stress τ_w can be deduced.