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Pressure and wall shear stress in blood hammer – Analytical theory

Q1 Chiang C. Mei[a,*], Haixiao Jing[b]

Q2 [a] Department of Civil & Environmental Engineering, Massachusetts Institute of Technology, USA
[b] Institute of Water Resources and Hydro-electric Engineering, Xi'an University of Technology, China

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ABSTRACT

We describe an analytical theory of blood hammer in a long and stiffened artery due to sudden blockage. Based on the model of a viscous fluid in laminar flow, we derive explicit expressions of oscillatory pressure and wall shear stress. To examine the effects on local plaque formation we also allow the blood vessel radius to be slightly nonuniform. Without resorting to discrete computation, the asymptotic method of multiple scales is utilized to deal with the sharp contrast of time scales. The effects of plaque and blocking time on blood pressure and wall shear stress are studied. The theory is validated by comparison with existing water hammer experiments.

1. Introduction

Blood hammer is a phenomenon known to occur in cerebral arteries, where sudden blockage by blood clots can cause acute rise of blood pressure and lead to intracerebral hemorrhage [2,6]. In a recent article Tasraei et al. (2015) have reported a numerical model of blood hammer in a short posterior cerebral artery (length $L = 6.75$ mm, diameter $2R = 1.77$ mm). Blood hammer can also occur in the longer mid cerebral arteries of length $20 < L < 80$ mm [2] and causes rupture of the vascular wall.

As pointed out first by Damsa et al. [6], the fluid mechanics of blood hammer is similar to the classic case of water hammer in pipe flow when a valve is suddenly closed. This transient phenomenon has been well treated by linear acoustics with the inclusion of an empirical law of wall friction. To treat the nonlinear friction term many schemes of numerical computations have been developed for practical predictions of water hammer, as reviewed by Larock et. al. [18] Jovic [15] and Ghidaoui et al. [12]. Flow in small blood vessels is mostly laminar. Experiments for laminar water hammer in metal pipes have been performed by Homboe and Rouleau [13] who used a highly viscous fluid. Relevant theory accounting for laminar friction was started by Zielke [33], who solved formally the linearized Navier–Stokes equations throughout the pipe and evaluated the inverse Laplace transform by numerical approximation. Most existing simulations of water hammer have revealed that the typical time scale of wave attenuation is much longer than the typical wave period associated with multiple reflections. This sharp contrast of scales suggests the use of asymptotic methods to obtain approximate solutions which bypass discrete computations and facilitate physical understanding.

An important objective of blood flow analysis is the prediction of wall shear stress which is known to be the cause of certain diseases [25,27,28] such as the formation of atherosclerotic plaques in carotid arteries [16,17]. Crucial in heart-valve tissue engineering [9], the magnitude and directionality of oscillatory shear stress affect the interaction between blood flow and the vascular tissue [7]. Excessively high wall shear stress can cause arteriosclerosis lesion and rupture of aneurysm [22], and is the major cause of heart attack, stroke, and peripheral arterial disease [8].

In their numerical simulation of blood hammer in short posterior cerebral arteries, Taeraeia et al. [26] compared Newtonian and NonNewtonian models of blood. Based on the Carreau model with four parameters they found that the pressure is somewhat higher, and the wall shear stress somewhat lower, in the non-Newtonian model. The results by both models are qualitatively similar, however. Quantitative differences can also arise by using different models and parameter values. In this article we shall examine the pressure and wall shear stress by assuming laminar flows of a Newtonian blood with constant viscosity. As will be shown the frequency of blood hammer in a long artery can be high enough so that viscous shear is important only in a thin oscillatory boundary layer, even in small cerebral arteries. Correspondingly, the relatively slow rate of wave attenuation permits one to employ the technique of multiple-scale asymptotics [4,21]. To

* Corresponding author.
E-mail address: ccmei@mit.edu (C.C. Mei).

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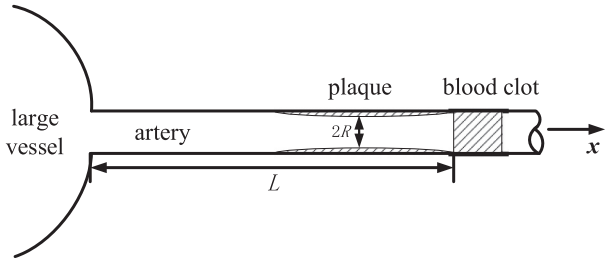

Fig. 2.1. Definition sketch.

consider the possible presence of local plaque formation we further allow a small non-uniformity of the vessel radius, and examine the dynamics associated with wave scattering by the protrusion. In hydraulics this non-uniformity may be the consequence of pipe corrosion.

2. Formulation

Referring to Fig. 2.1, we consider the classic problem of a straight and elastic tube of inner radius $R(x, t)$ in line with the x axis, with flowing blood fed by a large vessel or reservoir at $x = 0$. At $t = 0$ a valve at $x = L$ is closed abruptly. The entire tube is horizontal so that gravity has no effect.

Let $A(x, t)$ be the interior cross-sectional area of the tube and $U(x, t)$ the flow velocity averaged over the cross section, and $p(x, t)$ the dynamic pressure in the blood. Mass conservation requires

$$\frac{\partial \rho A}{\partial t} = -\frac{\partial (\rho A U)}{\partial x}, \quad A = \pi R^2, \tag{2.1}$$

where $R(x, t)$ is the interior radius of the artery. In an artery, the blood density ρ is assumed to be constant. We shall model the inward growth of plaque is by the reduction of artery area. Furthermore, we assume the plaque to be much thinner than R and does not contribute to the elastic resistance which is borne solely by the artery wall of thickness h. Let $\bar{R}(x)$ denotes the radius in the absence of distention and dR the incremental distention, i.e.,

$$R(x, t) = \bar{R}(x) + dR, \quad dR \ll \bar{R}. \tag{2.2}$$

We shall also consider small elastic deformation of the vessel wall so that the stress–strain is linear. Let dR be the elastic deformation which is related to the blood pressure increment by

$$\bar{R} dp = \frac{Eh}{\bar{R}} dR \tag{2.3}$$

where E is the Young's modulus and h the thickness of the wall [11]. In the hydraulic system the pipe is rigid but water is compressible,

$$\rho = \rho_0 + d\rho, \quad \text{with} \quad d\rho \ll \rho_0, \quad \text{and} \quad dp = C_s^2 d\rho \tag{2.4}$$

where C_s is the velocity of sound in water. In either case for small amplitude waves the fluid mass conservation is governed by

$$\frac{1}{\rho \bar{C}^2} \frac{\partial p}{\partial t} + \frac{1}{\bar{A}} \frac{\partial (\bar{A} U)}{\partial x} = 0. \quad \bar{A} = \pi \bar{R}^2(x), \quad 0 < x < L \tag{2.5}$$

where $\bar{C}$ is the Moen–Korteweg wave speed in an artery

$$\bar{C} = \sqrt{\frac{Eh}{2\rho \bar{R}}} \tag{2.6}$$

where $\bar{C}(x)$ can vary in x via $\bar{R}(x)$. Unlike the sound speed in water hammer which is a constant ($C_s = 1,500$ m/s), $\bar{C}$ is larger for larger E (stiffer wall), or larger h (thicker wall) or smaller $\bar{R}$ (smaller cross section). In human arteries of uniform radius R, $\bar{C}$ is normally less than 10 m/s [3]. However, it is well known that $\bar{C}$ can be much higher owing to the stiffening of the vascular walls [1]. A 10–30 fold increase can be produced by sclerosis of the arteries, arterioles and capillaries, and by a powerful initial distension of the vascular wall, i.e., $\bar{C} = 100$–300 m/s [6].

To account for wall friction, we consider the linearized equation of local fluid momentum

$$\frac{\partial u}{\partial t} = -\frac{1}{\rho_0} \frac{\partial p}{\partial x} + \frac{\nu}{r} \frac{\partial}{\partial r} \left(r \frac{\partial u}{\partial r} \right), \quad 0 < x < L, \quad 0 < r < \bar{R}. \tag{2.7}$$

where $u(r, t)$ is the local velocity which is related to the area average by

$$U(t) = \frac{2\pi}{\pi \bar{R}^2} \int_0^{\bar{R}} u(r, t)\, r dr \tag{2.8}$$

Convective inertia is negligible since $O(u, U)$ is typically just a few centimeters per second and the time and length scales are $t = O(L/\bar{C})$ and $x = O(L)$ respectively, so that,

$$u \frac{\partial u}{\partial x} \Big/ \frac{\partial u}{\partial t} = O\left(\frac{u}{\bar{C}} \right) \ll 1 \tag{2.9}$$

By taking the area average, the conservation law of mean momentum reads,

$$\rho_0 \frac{\partial U}{\partial t} = -\frac{\partial p}{\partial x} + \frac{2\tau_w}{\bar{R}}, \tag{2.10}$$

where τ_w is the wall stress:

$$\tau_w = \rho \nu \frac{\partial u}{\partial r}\bigg|_{r=\bar{R}}, \tag{2.11}$$

which is yet unknown. By cross differentiation of (2.5) and (2.10), we obtain formally the equation governing the dynamic fluid pressure,

$$\frac{1}{\bar{A}} \frac{\partial}{\partial x} \left(\bar{A} \frac{\partial p}{\partial x} \right) - \frac{1}{\bar{C}^2} \frac{\partial^2 p}{\partial t^2} = \frac{2}{\bar{R}} \frac{\partial \tau_w}{\partial x}, \quad 0 < x < L, \ t > 0. \tag{2.12}$$

It can be shown that wave radiation into the reservoir due to the piston motion at the inlet is negligible if $\bar{R}/L \ll 1$ [29], hence the dynamic pressure vanishes

$$p = 0, \quad x = 0. \tag{2.13}$$

Let the initial averaged flow velocity at the station of sudden blockage $x = L$ be U_0. We impose the following boundary condition

$$\frac{\partial p}{\partial x} = -\rho_0 \frac{\partial U}{\partial t} = \rho_0 U_0 D(t), \quad x = L, \tag{2.14}$$

The simplest mathematical model for $D(t)$ is $\delta(t)$. For a more realistic model we adopt the following function which has sharp but finite peak over a very short duration $t_0 \ll L/C$,

$$D(t) = \frac{1}{t_0} \begin{cases} \dfrac{t}{t_0}, & 0 < t < t_0 \\ 2 - \dfrac{t}{t_0}, & t_0 < t < 2t_0 \\ 0, & t > 2t_0. \end{cases} \tag{2.15}$$

which is a less singular version of the delta function and has the property that

$$\int_0^{2t_0} D(t)\, dt = 1 \tag{2.16}$$

This simple choice is less singular and improves the numerical convergence of the series solution to be developed later.

In addition we impose the initial conditions

$$p = \frac{\partial p}{\partial t} = 0, \quad t = 0, \quad 0 < x < L \tag{2.17}$$

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