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Mathematical model of arterial stenosis

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Abstract—*A mathematical model for pulsatile flow in a partially occluded tube is presented. The problem has applications in studying the effects of blood flow characteristics on atherosclerotic development. The model brings out the importance of the pulsatility of blood flow on separation and the stress distribution. The results obtained show fairly good agreement with the available experimental results.*

Keywords—*Atherosclerosis, Biomechanics, Blood flow, Modelling, Stenosis*

1 Introduction

ONE of the present major health hazards is atherosclerosis, which refers to the occlusion of the arterial lumen, partly or fully, due to the deposition of fatty substances. Until recently, age, sex, diet, lipid metabolism, hormones, hypertension and associated diseases were considered to be the factors chiefly responsible for the development of atherosclerosis. However, the researches of TEXON (1963) have proved conclusively that none of these factors is always present, nor is any particular combination present as a primary factor responsible in a causative sense for atherosclerosis. In spite of the available statistical data correlating atherosclerosis with those factors, none of them has been established as the main cause. On the other hand, studies on human autopsy specimens and the experimental production of atherosclerosis in dogs (CLARKSON, 1968) by altering vascular configurations lead us to believe that atherosclerosis is a sequel primarily of the flow mechanism of the circulatory system. The results of TEXON (1963), MUSTARD *et al.* (1963), MITCHELL and SCHWARTZ (1965), FRY (1968, 1969) further corroborate the haemodynamic concept of atherosclerosis, which considers hydrodynamic factors to play a major role in the etiology and pathogenesis of atherosclerosis.

The recent investigations of CARO *et al.* (1969, 1971) show that the flow mechanism has a controlling and inhibitory effect, rather than a causative role in atherosclerotic development. It is further claimed that atherosclerosis is associated with the shear-dependent mass-transport phenomenon. YOUNG (1968) observed that once a vascular lesion has developed, there may be a coupling effect between its further development and the subsequent flow characteristics. Thus, though the exact role, causative or preventive, of the flow of blood in the vascular system has not yet been understood clearly, the analysis of flow in a stenosed artery based on mathematical models has assumed great importance in the study of atherosclerotic plaques in the arterial system.

Notwithstanding the importance of hydrodynamical aspects in atherosclerotic development, not much headway has been made in this direction. Interest in research along this line was triggered off by an excellent paper by YOUNG (1968), dealing with the steady flow of a fluid through a partially occluded tube. Numerous other investigators such as FORRESTER and YOUNG (1970), LEE and FUNG (1970), YOUNG and TSAI (1973), BACK *et al.* (1979) have studied various aspects of the problem, mainly presenting the experimental results. With the literature on experimental aspects of atherosclerosis increasing rapidly, it is felt that a suitable mathematical model to describe the phenomenon and to study the problem analytically is more appropriate. Thus, the purpose of the investigation reported in this paper is to examine a simple mathematical model describing the arterial stenosis. Closed form solutions are obtained for the pulsatile flow in a partially occluded tube.

2 Formulation of the problem

Young in his trend-setting work has obtained the solutions for the steady flow of blood in a stenosed vessel, considering blood to be a homogeneous viscous fluid. He has also discussed the fluid mechanics aspects of separation of flow. The applicability of steady-flow analysis to arterial blood flow is questionable, since blood flow is distinctly pulsatile. This has been appreciated in the said paper and an offshoot of this is an experimental work by YOUNG and TSAI (1973). Thus, we shall now re-examine the analysis of YOUNG (1968) of flow through an occluded tube under a pulsatile pressure gradient.

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Blood is taken to be a homogeneous, incompressible, Newtonian fluid. This assumption is well justified in the larger arteries, which are more prone to atherosclerotic development. The model for the stenosed artery is taken to be a rigid tube with a constriction. Various models have been proposed to define the geometry of the constriction MANTON (1971) proposed a tapered tube model, while LEE and FUNG (1970) assumed the constriction to be described by a Gaussian curve. Recently, SCHNECK and OSTRACH (1975) have modelled the stenosed artery by a channel having a small exponential divergence. However, following Young's approach, we shall take the shape of the axisymmetric constriction to be represented by a cosine curve of the form

$$R_1 = a - \delta(T)\left(1+\cos\frac{\pi X}{2Z_0}\right)$$
$$\text{for } -2Z_0 \leqslant X \leqslant 2Z_0 \qquad (1)$$
$$= a \text{ otherwise,}$$

where a is the radius of the artery outside the stenotic region and $4Z_0$ is the total length over which the stenosis extends. $2\delta(T)$ represents the maximum protuberance of the stenotic growth into the lumen of the artery, at time T. However, since the rate of growth of the stenosis is very small compared with the magnitude of the temporal fluctuations of the velocity field, δ in eqn. 1 can be treated as a constant in the present analysis.

Considering pulsatile flow of circular frequency Ω in an axisymmetric tube, the basic equations of motion are

$$\frac{1}{R}\frac{\partial}{\partial R}(RU)+\frac{\partial W}{\partial X}=0 \qquad (2)$$

$$\frac{\partial U}{\partial T}+U\frac{\partial U}{\partial R}+W\frac{\partial U}{\partial X}=-\frac{1}{P}\frac{\partial P}{\partial R}+\nu\left[\frac{\partial^2 U}{\partial R^2}+\frac{1}{R}\frac{\partial U}{\partial R}+\frac{\partial^2 U}{\partial X^2}-\frac{U}{R^2}\right] \qquad (3)$$

$$\frac{\partial W}{\partial T}+U\frac{\partial W}{\partial R}+W\frac{\partial W}{\partial X}=\nu\left[\frac{\partial^2 W}{\partial R^2}+\frac{1}{R}\frac{\partial W}{\partial R}+\frac{\partial^2 W}{\partial X^2}\right]-\frac{1}{P}\frac{\partial P}{\partial X} \qquad (4)$$

where U, W are the velocity components in the R, X directions, respectively, taking the cylindrical polar co-ordinate system (R, θ, X) with the X-axis coinciding with the axis of symmetry of the artery, ρ the density of the fluid and ν the coefficient of kinematic viscosity.

The corresponding boundary condition is the no slip condition, which is given by

$$U = W = 0 \quad \text{on} \quad R = R_1 \qquad (5)$$

Introducing the following nondimensional variables,

$$r = R/a, \quad x = X/a, \quad p = P/\rho U_0^2,$$
$$u = \frac{UZ_0}{U_0\delta}, \quad w = \frac{W}{U_0}, \quad t = T\Omega Z_0/a \qquad (6)$$

where $U_0 e^{i\Omega t}$ is the upstream velocity with which the flow is entering the tube, the basic equations are transformed into

$$\frac{\delta}{a}\frac{\partial}{\partial r}(ru)+\frac{\partial w}{\partial x}=0 \qquad (7)$$

$$\frac{\alpha^2}{\mathrm{Re}}\frac{\partial u}{\partial t}+\frac{\delta}{Z_0}u\frac{\partial u}{\partial r}+\frac{a}{Z_0}w\frac{\partial u}{\partial x}=-\left(\frac{Z_0}{\delta}\right)\frac{\partial p}{\partial r}+\frac{1}{\mathrm{Re}}\left[\frac{\partial^2 u}{\partial r^2}+\frac{1}{r}\frac{\partial u}{\partial r}+\left(\frac{a}{Z_0}\right)^2\frac{\partial^2 u}{\partial x^2}-\frac{u}{r^2}\right] \qquad (8)$$

$$\frac{\alpha^2}{\mathrm{Re}}\left(\frac{Z_0}{a}\right)^2\frac{\partial w}{\partial t}+\frac{\delta}{a}u\frac{\partial w}{\partial r}+w\frac{\partial w}{\partial x}=-\frac{\partial p}{\partial x}+\left(\frac{Z_0}{a}\right)\frac{1}{\mathrm{Re}}\left[\frac{\partial^2 w}{\partial r^2}+\frac{1}{r}\frac{\partial w}{\partial x}+\left(\frac{a}{Z_0}\right)^2\frac{\partial^2 w}{\partial x^2}\right] \qquad (9)$$

where $\alpha = a\sqrt{\Omega/\nu}$, Womersley's parameter, and $\mathrm{Re} = aU_0/\nu$ is the Reynold's number.

It is evident that, eqns. 8 and 9 being highly nonlinear, it is not possible to solve this system of eqns. 7 to 9 analytically. Nevertheless, using the available experimental data and certain order analysis the above equations are simplified as follows.

In the human arterial system, the parameter α takes numerical values between 1 and 4 (FUNG *et al.*, 1972) and hence we can take $\alpha \sim 0(1)$. Further, during the initial stages of the formation of the stenosis, δ/a can be taken as $\sim 0{\cdot}1$ (FRY, 1968). Thus, taking

$$\frac{a}{Z_0} \sim 0(1), \quad \frac{\delta}{a} \sim 0{\cdot}1, \quad \mathrm{Re}\frac{\delta}{Z_0} \ll 1 \qquad (10)$$

corresponding with a mild stenosis, we find that $\partial p/\partial r \ll \partial p/\partial x$. Further, from the equation of continuity (eqn. 7) $\partial w/\partial x \sim 0(\delta/a)$. Using this fact, we find that the connective terms in eqn. 9 are very small compared with the corresponding viscous terms. Moreover, among these viscous terms, the term $(\delta/a)^2\,\partial^2 w/\partial x^2$ is negligible compared with the other

two terms. Thus, the equations of motion can be simplified to a great extent to yield

$$\frac{\partial p}{\partial r} = 0, \quad . \quad . \quad . \quad . \quad . \quad . \quad . \quad . \quad . \quad . \quad (11)$$

$$\frac{\alpha^2 \beta^2}{\mathrm{Re}} \frac{\partial w}{\partial t} = -\frac{\partial p}{\partial x} + \frac{\beta}{\mathrm{Re}} \left[\frac{\partial^2 w}{\partial r^2} + \frac{1}{r} \frac{\partial w}{\partial r} \right] \quad . \quad . \quad . \quad . \quad (12)$$

where $\beta = Z_0/a$.

Since the flow is pulsatile, and the eqns. 11 and 12 are linear, we seek solutions of the form

$$Y = Y^* e^{it/\beta} \quad . \quad . \quad . \quad . \quad . \quad . \quad . \quad . \quad . \quad (13)$$

where Y stands for w or p. Substituting eqn. 3 in eqn. 12, the equation for w^* is

$$\frac{\partial^2 w^*}{\partial r^2} + \frac{1}{r} \frac{\partial w^*}{\partial r} - i\alpha^2 w^* = \frac{\mathrm{Re}}{\beta} \frac{\partial p}{\partial x} \quad . \quad . \quad (14)$$

The corresponding boundary conditions are

$$w^* = 0 \quad \text{on} \quad r = \frac{R_1(x)}{a} \quad . \quad . \quad . \quad . \quad . \quad . \quad (15)$$

and the velocity is finite on $r = 0$. The complete solution for the axial velocity is then given by

$$w(r, x, t) = R.P. \left\{ \left[\frac{I_0(i^{\frac{3}{2}} \alpha r)}{I_0(i^{\frac{3}{2}} \alpha R(x))} - 1 \right] \times \frac{\partial p}{\partial x} \frac{\mathrm{Re}}{1\alpha^2 \beta} e^{it/\beta} \right\} \quad . \quad . \quad . \quad . \quad . \quad . \quad (16)$$

where I_0 is the modified Bessel function.

The steady-state solution is obtained from eqn. 16 by taking $\alpha \to 0$

$$w = \frac{\partial p}{\partial x} \left(\frac{r^2 - R^2}{4\mu} \right)$$

which coincides with the solution of YOUNG (1968).

The resistive impedance, which is a measure of resistance to flow, is defined as (MCDONALD, 1974)

$$\Lambda = \frac{\Delta p}{Q}, \quad . \quad . \quad . \quad . \quad . \quad . \quad . \quad . \quad . \quad . \quad (17)$$

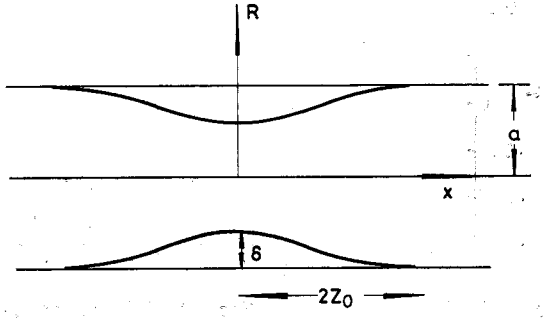

Fig. 1 Geometry of the stenosed artery

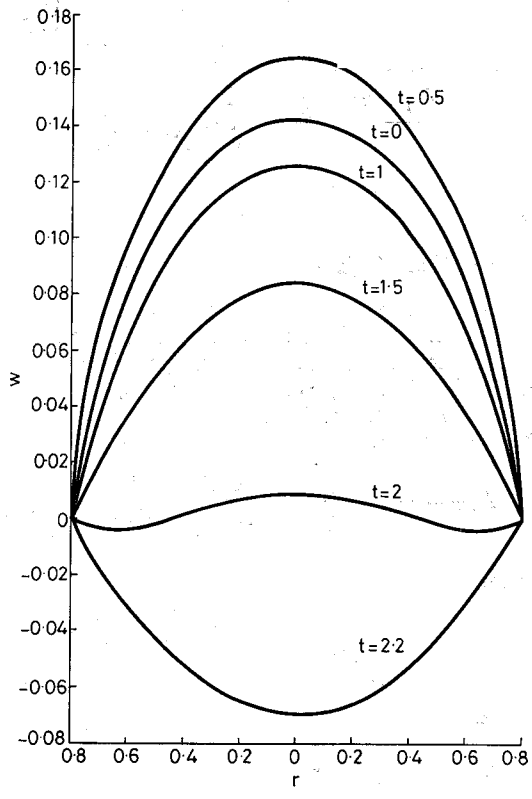

Fig. 2 Axial velocity distribution at x = 0 cross-section when α = 1, δ = 0·2 and β = 1

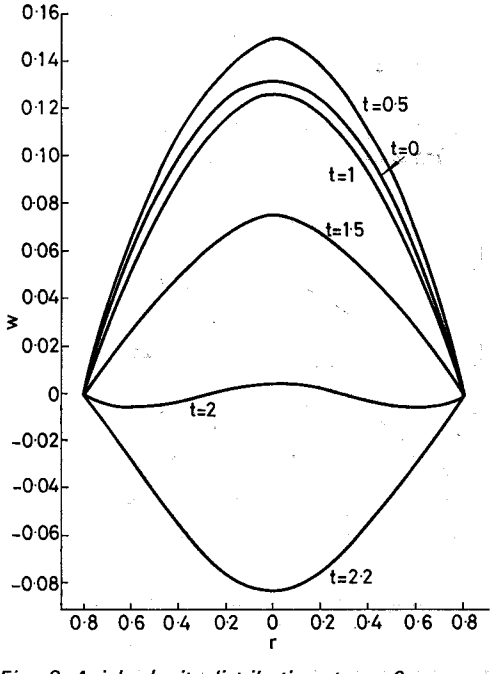

Fig. 3 Axial velocity distribution at x = 0 cross-section when α = 2, δ = 0·2 and β = 1

where Δp is the pressure drop across the stenosis, and Q is the discharge through the tube. In this particular context, therefore

$$\Lambda = R.P.\left[\frac{i e^{-it/\beta}\alpha^2\,\beta a^2}{\pi \mathrm{Re}} \int_{-2}^{2} \frac{I_0(z)}{R_1{}^2(x) I_1(z)}\,dx\right] \quad . \quad . \quad . \quad . \quad (18)$$

where

$$z = i^{\frac{3}{2}}\alpha R_1(x)/a$$

The integrand in eqn. 18 can be put in a neat form, in terms of Kelvin functions. However, the integration is performed numerically, for closed form integration is not possible. To highlight the role played by a stenosis, the impedance is calculated in the absence of a stenosis, and is given by

$$\Lambda_{WS} = -R.P.\left\{\frac{i e^{-it/\beta}\alpha^2\,\beta}{\pi \mathrm{Re}} \frac{2I_0(\alpha i^{\frac{3}{2}})}{I_2(\alpha i^{\frac{3}{2}})}\right\} \quad (19)$$

The stress distribution on the wall, in view of the order of analysis used earlier, simplifies to

$$\tau = R.P.\frac{a^3\,y}{2\pi R^3}\frac{I_1(y)}{I_2(y)}e^{it/\beta} \quad . \quad . \quad . \quad . \quad (20)$$

where

$$y = i^{\frac{3}{2}}\alpha\frac{R(x)}{a}$$

It is interesting to note that the stress distribution on the wall tends to the steady-state value, as $\alpha \to 0$.

3 Discussion

The eqns. 11 and 12 warrant some comments. Even though they look very much similar to the linearised boundary layer equations, it must be noted that the approximation made are not of the usual boundary-layer type, but are dictated by

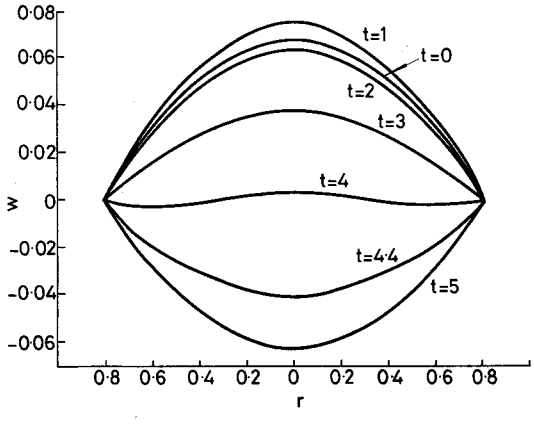

Fig. 4 Axial velocity distribution at x = 0 cross-section when $\alpha = 2$, $\delta = 0{\cdot}2$ and $\beta = 2$

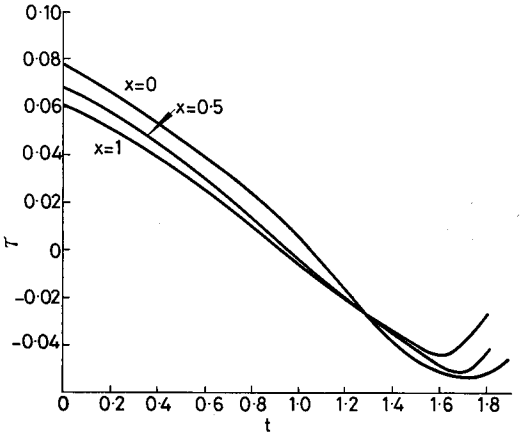

Fig. 6 Shear-stress distribution at different cross-sections when $\alpha = 1$, $\delta = 0{\cdot}1$ and $\beta = 1$

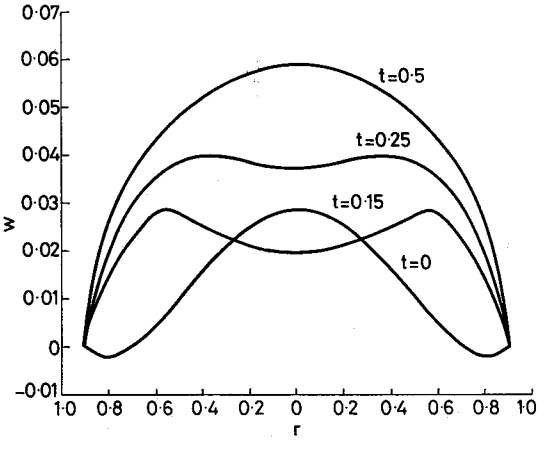

Fig. 5 Axial velocity distribution at x = 0 cross-section when $\alpha = 1$, $\delta = 0{\cdot}1$ and $\beta = 1$

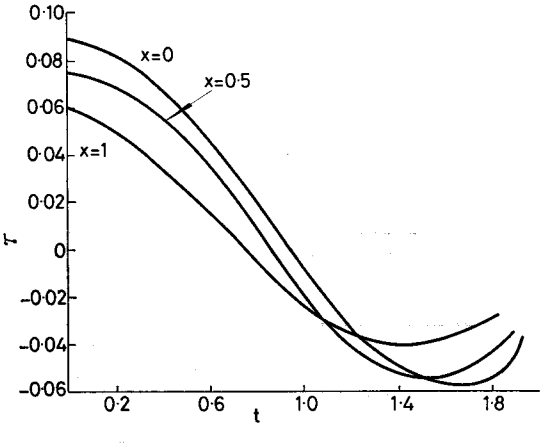

Fig. 7 Shear-stress distribution at different cross-sections when $\alpha = 2$, $\delta = 0{\cdot}1$ and $\beta = 1$

physiological considerations. This is in contrast to the work of FORRESTER and YOUNG (1970) who have assumed the flow to be only of the boundary-layer type and used the Karman–Pohlhausen method.

A mathematical model for the pulsatile flow of blood through a stenotic region has been studied. All the physiologically relevant factors have been incorporated in the analysis and the impedance and stress distribution are depicted graphically. Figs. 2 to 5 describe the axial velocity profile at the middle of the stenosis. These figures clearly show that the profiles depend very much on time and that they change markedly as t varies. For instance, in Fig. 2, when time increases from $t = 0$, the peak velocity increases until at $t = 0{\cdot}5$, a reduction in the velocity profile commences. At $t = 2$, the flow near the tube axis is in the forward direction, while near the wall, there is a slight back flow. As time increases, there is an indication of the flow reversal in the cross-section. Later, the cycle repeats. These four figures, though essentially the same, show the dependence of the velocity on the parameters α, β and δ. For example, in Fig. 5, even at $t = 0$, there is a back flow at the wall of the tube. This indicates that there is clearly a separated region, in which back flow takes place. In fact, evidence of back flow was observed in the experiments of YOUNG and TSAI, (1973). Another observation from Figs. 2 and 3 is the significant drop in the peak velocity as α increases.

Figs. 6, 7 and 8 depict the variation of stress distribution and impedance, respectively. It is noticed from the stress distribution curve that separation first occurs far downstream. From Figs. 7 and 8 it is seen that the point of separation shifts towards the centre of the stenotic region as Womersley's parameter increases. In Fig. 6, we see that the stress distribution is negative for some time, before becoming positive again. This period of negative stress corresponds to the separation of flow.

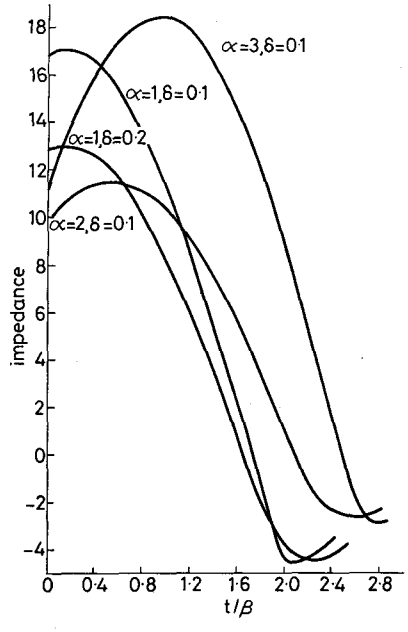

Fig. 8 Distribution of impedance (I) for different combinations of α and δ

4 Conclusion

In this paper, the importance of time dependence on the flow characteristics through a stenotic artery has been brought out. From the stress distribution curve, it is concluded that separation in the flow field is predominant for larger values of α. Hence, in view of the observations of CARO *et al.* (1971), the larger arteries, where the value of α is greater than in smaller vessels, are more prone to plaque formation. This is in general agreement with the experimental observations available.

The other physiological factor which has been studied is impedance. From the definition of impedance, it is clear that, under a given pressure gradient, a greater impedance will imply less flow of fluid. Thus the impedance gives a measure of the volume of blood received by different organs; this is an important factor which might play a crucial role in the diagnosis and treatment of some diseases. In the end, it must be mentioned that branching and elasticity play a major role in the development of atheroscolerosis (O'BRIEN *et al.*, 1976). In a subsequent paper, we intend studying their effects in stenosis.

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