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DEFORMATION OF THE WALL OF THE ARTERY WITH TIME-DEPENDENT STENOSIS DUE TO PULSATILE FLOW OF BLOOD

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ABSTRACT

Investigation has been carried out to see how the presence of a growing stenosis affects the deformation of the wall of an artery in the pulsatile flow of blood. The expressions for the resulting deformations in the radial and axial directions were obtained. From analysis of these results, we noted that at the beginning of the growth of the stenosis, its effect on the deformation of the wall was minimal but that it increases subsequently until it assumes the same alarming destructive level that had earlier been reported (Katiyar and Mbah 1996). However, the gradual growth of the stenosis distorts the flow characteristics more as compared to the situation where the maximum height of the stenosis had been attained. Detailed analysis of the results obtained is as contained in the body of this work.

KEY WORDS: Deformation, Artery, Stenosis, Pulsatile, Flow

INTRODUCTION

The effect of time-dependent stenosis on the pulsatile flow of blood through an elastic tube has been considered. It was reported that the time-dependent stenosis on the artery under pulsatile flow of blood through this artery exposes the cell walls of the artery at the stenotic regions to variation in velocites (radial and axial), Pressures, Volumetric flows as well as stress. It was concluded that it might affect those cells at the stenotic regions to the extent that it may cause hardening of the artery. With this fact, we decided to investigate the effect of the stenosis on the deformation of the wall of the artery due to pulsatile flow of blood. We decided to do this because the arteries are elastic and that under pulsatile flow of the blood, it can dilate or contract (Kapur, 1980) . Young (1968) worked similarly on the effect of the time-dependent stenosis on flow through a tube. However, he did not consider the artery as being elastic. The concept of elasticity of the walls of the artery introduces radial flow in addition to the axial flow. In the same way, the introduction of elasticity of the wall introduces radial deformation in addition to axial deformation of the wall, due to pulsatile flow of blood through it.

The case of deformation of the arterial wall with full-grown stenosis due to pulsatile flow of blood, has been considered. It was found that the reported cases of possible back flow before the stenotic point and the separated/turbulent flows beyond the stenotic region, Chaturani, (1984), might have been caused by the contraction rather than dilation of the stenotic regions on the start of the pulsatile motion. We therefore, in this work tried to verify if similar trend exists right from the start of the growth of the stenosis. Similarly, we took a look at the stress distribution to see if this is more in the stenotic regions than in any other part of the artery. This present work will give us a more general over-view of the effect of stenosis, whether fully-grown or still growing, in our blood circulatory system. It equally tried to give the physiological implications of the presence of stenosis in our artery under pulsatile flow of blood at all level of its developments.

In the formulation of the problem here, we had assumed that the rate of growth of the stenosis is constant. This is a deviation from reality and would therefore imply that changes in the radial and axial deformations of the wall with time, is also constant. The solutions to the resulting differential

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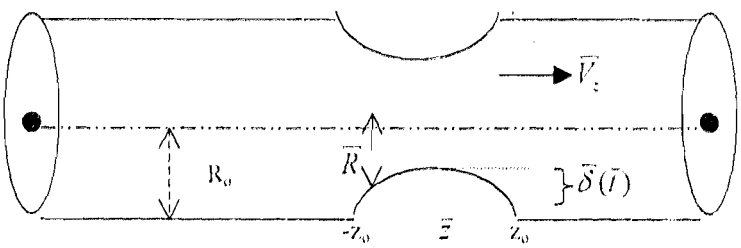
equations describing the deformation of the walls either radially or axially are of the form of Bessel's differential equations. Discussions of the results obtained are made with the possible biological and physiological implications stated.

Formulation of the Problem

Let us show the artery with the time-dependent stenosis as:



We shall denote the material of the wall of the artery by **U** so that U_r and U_z shall denote the deformation in the radial and axial directions **respectively.** Since we are talking of arteries (with exception of the Carotid artery) which can dilate or contract, we assumed that the material of the wall is elastic so that rightly speaking, we have a flow through an elastic cylindrical tube which can be described by the Navier-Stokes equation of motion of fluid flow. Because of the elasticity of the wall, we have radial and axial deformation of the wall. Hence,

$U_r \neq 0$, $U_z \neq 0$ and $U_0 = 0$. The deformation of the material of the wall is due to the pulsatile motion of the blood through the artery. By deformation here, we mean the movement of the wall in the form of dilation or contraction, which enhances the free flow of the blood being, pumped by the heart whose motion we termed pulsatile. Incorporating the stenosis in the artery, we discover that the radius of the lumen of the artery is no longer the same. Hence, we need an expression for the radius, which will represent the actual radius at any point of the artery. We shall recall that the height of the stenosis is time-dependent meaning that the stenosis is still growing which shall also mean that the radius is changing with time. If

RADIAL DEFORMATION

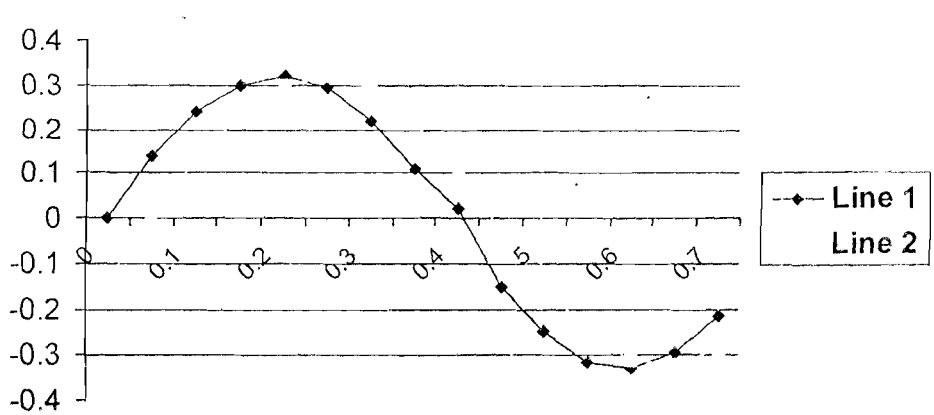
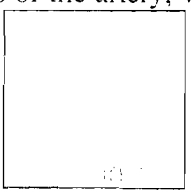
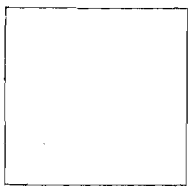
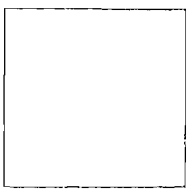
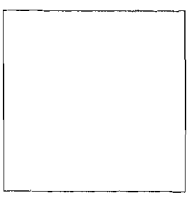


FIG. 1. TIME (t)

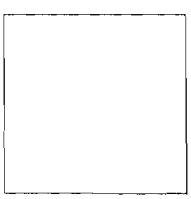
Line 1 = Without Stenosis **Line 2** = With Stenosis

is the required expression for the radius at all points of the artery, we shall write this in terms of the normal radius of the artery R_0 and the height of the stenosis  as thus:

 =  (1)

where  $= \delta_m(1 - e^{-t/\tau})$, δ_m = maximum height of the stenosis attainable, τ = Rate of increase in the height of the stenosis.

Since the deformation is induced by the flow of blood, it means that the deformation of the material of the wall proceeds along with the flow of blood. Hence the Navier-Stokes equation describing the flow can rightly describe the deformation. Thus, the equations describing the deformation of the material of the wall both radially and axially with the accompanying continuity equation are given respectively as:

 (2)

and (3)

$$\frac{\partial \bar{U}_z}{\partial \bar{z}} + \frac{\partial \bar{U}_r}{\partial \bar{r}} + \frac{\bar{U}_r}{\bar{r}} = 0 \qquad (4)$$

where ρ_w is the density of the material of the wall, **G** is the shear modulus and Ω is the negative of the mean normal stress.

To avoid dimensional problem that might arise in subsequent calculation, we introduce the following non-dimensional variables:

$$U_r = \frac{\bar{U}_r}{V_0}, \quad U_z = \frac{\bar{U}_z}{V_0}, \Omega = \frac{\bar{\Omega}}{\rho_\omega V_0}, t = \frac{[\bar{t}V_0]^2}{R_0}, r = \frac{\bar{r}}{R_0}, z = \frac{\bar{z}}{z_0}, \delta = \frac{\bar{\delta}(\bar{t})}{R_0}$$

With these variables, the deformation equations given above become:

$$\frac{\partial^2 U_r}{\partial t^2} = -\frac{\partial \Omega}{\partial r} + \frac{G}{\rho_\omega V_0 R_0}\left[\frac{\partial^2 U_r}{\partial r^2} + \frac{1}{r}\frac{\partial U_r}{\partial r} - \frac{U_r}{r} + \frac{\partial U_r}{\partial z^2}\right] \qquad (5)$$

$$\frac{\partial^2 U_z}{\partial t^2} = -\frac{\partial \Omega}{\partial z} + \frac{G}{\rho_\omega V_0 R_0}\left[\frac{\partial^2 U_z}{\partial r^2} + \frac{1}{r}\frac{\partial U_z}{\partial r} + \frac{\partial U_z}{\partial z^2}\right] \qquad (6)$$

Hence we have assumed that $R_o \approx Z_o$ and V_o is a characteristic velocity of the blood flowing through the artery. Let us put $(\rho_w V_o R_o)/G = R_e^*$ for further simplification.

METHOD OF SOLUTION.

As the pulsatile motion of the blood causes the deformation of the material of the wall of the artery, it means that the deformation both in the radial and axial directions is time-dependent. Depending on time, the deformation can be that of dilation or contraction. As the wall is assumed to be elastic, it also means that the deformation occurs both radially and axially so that together with mean normal stress we have them given as:

$$U_r = U_1(r,z,t) = U_1(r)\, e^{inwt - iy_n z}$$

$$U_z = U_z(r,z,t) = U_z(r)\, e^{inwt - iy_n z} \qquad (7)$$

$$\Omega = \Omega(r,z,t) = \Omega(r)\, e^{inwt - iy_n z}$$

where $y_n = \frac{2\pi}{\lambda_n} - i\delta(t)$

Substituting equation (7) into equation (5) and (6) accordingly, we have:

$$\frac{d^2U_1}{dr^2} + \frac{1}{r}\frac{dU_1}{dr} - \left[y_n^2 + (inw - b)^2 R_e^* + \frac{1}{r^2} \right] U_1 = R_e^* \left[2\beta (inw - b) + \frac{d\Omega(r)}{dr} \right] \qquad (8)$$

$$\frac{d^2U_2}{dr^2} + \frac{1}{r}\frac{dU_2}{dr} - \left[y_n^2 + (inw - b)^2 R_e^* \right] U_2 = - iy_n R_e^* \Omega(r) \qquad (9)$$

where $b = \frac{\delta_m z}{\tau}$

Let us for simplicity define $k_n^2 = y_n^2 + (inw - b)^2 R_e^*$. Then we have:

$$\frac{d^2U_1}{dr^2} + \frac{1}{r}\frac{dU_1}{dr} - \left[k_n^2 + \frac{1}{r^2} \right] U_1 = R_e^* \left[2\beta (inw - b) + \frac{d\Omega(r)}{dr} \right] \qquad (10)$$

$$\frac{d^2U_2}{dr^2} + \frac{1}{r}\frac{dU_2}{dr} - k_n^2 U_2 = - iy_n R_e^* \Omega(r) + 2\sigma(inw - b) R_e^* \qquad (11)$$

where $\beta = \frac{dU_1(r)}{dt}$, $\sigma = \frac{dU_2(r)}{dt}$

Equation (10) and (11) are Bessel's type of differential equations. Hence, an assumption of solutions to this set of equations in the form of Bessel's functions will be rightly justified. Thus, we have the general solutions to these equations as:

$$X = \alpha_1 J_1(iy_n r) + \alpha_2 J_1(ik_n r) \qquad (12)$$

$$Y = \beta_1 J_0(iy_n r) + \beta_2 J_0(ik_n r) \qquad (13)$$

Substituting equation (12) into equation (10), we get that:

$$\frac{d\Omega(r)}{d\,r} = -[(inw - b)^2\alpha_1^{\,1} J_1(iy_n r) + 2R_e^{*}\beta\,(inw - b)]$$

Solving this equation, we have:

$$\Omega(r) = -i[\{\frac{(inw - b)^2\alpha_1^{\,1} J_0(iy_n r)}{y_n}\} + 2rR_e^{*}\beta\,(inw - b)] \qquad (14)$$

Similarly, substituting equation (13) into equation (11), we get:

$$\beta_1^{\,1}(inw - b)^2 J_0(iy_n r) = iy_n\,\Omega(r) + 2\sigma(inw - b) \qquad (15)$$

But since the deformation is finite, we demand that the expressions for $\mathbf{U_1(r)}$ be finite. Hence, we require that $\alpha_2 = \beta_2 = 0$ for these expressions to be finite. Substituting these new forms of these expressions in equation (7), we have a complete expression for the radial and axial deformation as well as the mean negative stress as:

$$U_R = \alpha_1^{\,1} \sum_{n=0}^{\infty} J_1(iy_n R)\, e^{imat - iy_n z} \qquad (16)$$

$$U_Z = \beta_1^{\,1} \sum_{n=0}^{\infty} J_0(iy_n R)\, e^{imat - iy_n z} \qquad (17)$$

$$\Omega = -\sum_{n=0}^{\infty} i[\{\frac{(inw - b)^2\alpha_1^{\,1} J_0(iy_n R)}{y_n}\} + 2RR_e^{*}\beta\,(inw - b)]\, e^{imat - iy_n z} \qquad (18)$$

where we have replaced r by R, a more general expression for the radius of the artery.

RESULTS AND DISCUSSIONS

We had earlier looked at a situation where we have a full-grown stenosis in the artery. We discovered that in this case, the radial deformation induced by the pulsatile motion never brought the already deformed parts (stenotic region) to the normal level or state (t=0) of the artery (depending on the height of the stenosis). In this very case, we saw that the stenotic region dilates less but contracts more than the non-stenotic region. This can be seen in fig. 1 and fig. 2.

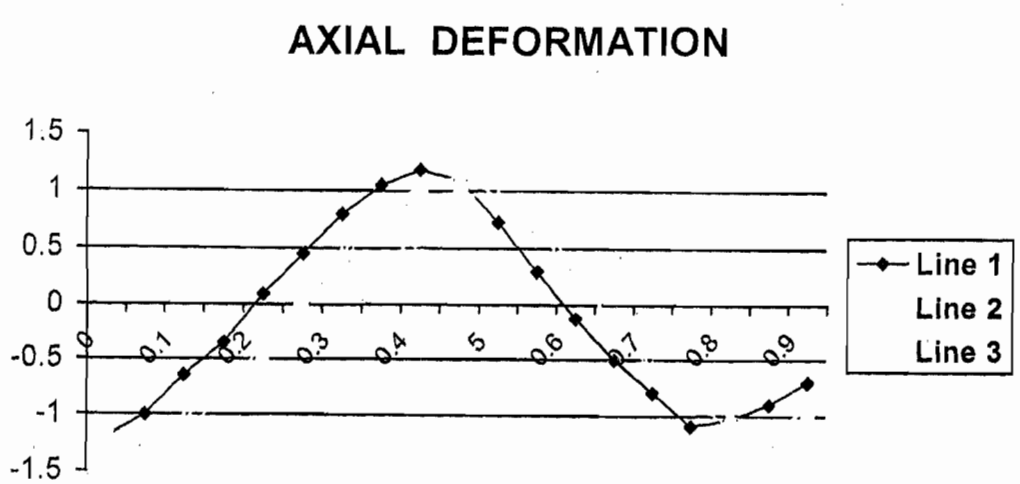


FIG. 2. TIME (t)

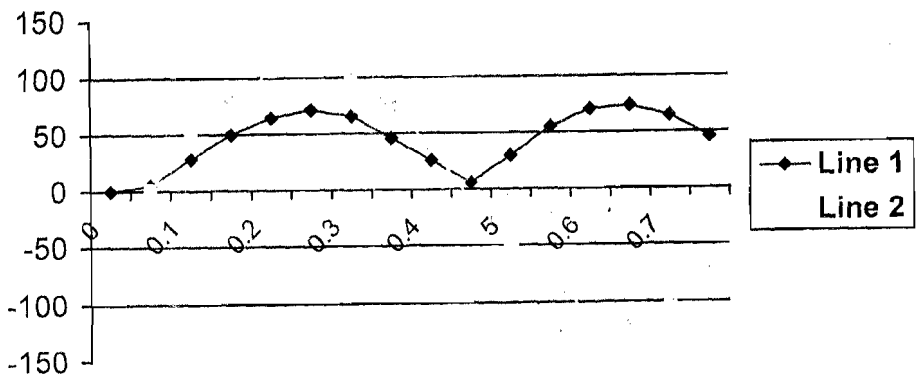


FIG. 3. TIME (t)

Line 1 = Without Stenosis Line 2 = With Stenosis

Line 3 in Fig. 2 shows the actual deformation done to the artery by the in-growth due to this disease (called stenosis) as the time intervals are subtracted from the actual joint effect. This further explains what we said above that the actual deformation done to the artery at the stenotic region due to the pulsatile motion of the blood is not the same as that done to the regions of the artery without stenosis. The dilation level in the non-stenotic regions is higher than that in the stenotic regions while the contraction or depression in the stenotic regions is deeper than what obtains in the non-stenotic regions. This quite agrees with real life expectation and observation. The joint effect of the deformation due to the pulsatile motion of the blood and that of the disease (stenosis) at the first flow time shows that the artery at the stenotic region dilated. However, line 2 in Fig. 2 shows that even though it dilated, it did not even produce the same level of dilation. Also it takes a lesser time for the arterial parts without stenosis to attain a maximum dilation than those with stenosis. This goes to confirm our earlier report of the existence of contraction in the stenotic region before dilation. This explains why there is time lag in attaining maximum dilation. From Katiyar and Mbah (1996) we find that the effect of full-grown stenosis on the start off of the pulsatile motion of blood is more glaring than that of gradually growing stenosis. In the time-dependent stenosis, the fear of back flow, turbulent flow, etc at the start of the pulsatile motion is not actually possible as it takes some time for the stenosis to attain the maximum height where these problems are usually experienced. Also it is shown that the level of deformation on the walls of the artery at such a region is less compared to that on the walls of the artery with time-dependent stenosis though as time progresses, the deformation caught up with one another. This point is where the time-dependent stenosed artery must have attained the maximum height.

In the case of axial deformation, we find that just like in the radial deformation, the level of dilation of the constricted region is less than that of the un-constricted regions while the level of contraction (depression) is more in the constricted region than that of the un-constricted region. In the time-dependent stenosed artery, we found the deformation of the wall starts off rather gradually than that of the artery without stenosis. The artery with full-grown stenosis will contract first before it starts dilating. However, we saw that with time, the wall of the artery with time-dependent stenosis dilates and contracts at equal rate with those of the walls with full-grown stenosis. We also saw that more time is taken for the arteries to dilate than to contract. However, this behaviour of the arterial wall is most pronounced in the stenotic regions. From all these analysis, it appears that pulsatile flow of blood induces more of axial deformation on the wall of the artery. Hence we may conclude that the pulsatile flow of blood principally induces axial deformation such that the radial deformation is only barely possible because of the elastic nature of the artery.

It was also observed that the stenotic regions of the artery absorb more stress than the non-

stenotic regions. Clear explanation for this is not known but we suspect that the stenotic region is already under stress and it might not be able to absorb much more stress.

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