Effect of a Time-Dependent Stenosis on Flow Through a Tube

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A common occurrence in the arterial system is the narrowing of arteries due to the development of atherosclerotic plaques or other types of abnormal tissue development. As these growths project into the lumen of the artery, the flow is disturbed and there develops a potential coupling between the growth and the blood flow through the artery. A discussion of the various possible consequences of this interaction is given. It is noted that very small growths leading to mild stenotic obstructions, although not altering the gross flow characteristics significantly, may be important in triggering biological mechanisms such as intimal cell proliferation or changes in vessel caliber. An analysis of the effect of an axially symmetric, time-dependent growth into the lumen of a tube of constant cross section through which a Newtonian fluid is steadily flowing is presented. This analysis is based on a simplified model in which the convective acceleration terms in the Navier-Stokes equations are neglected. Effect of growth on pressure distribution and wall shearing stress is given and possible biological implications are discussed.

Introduction

It is well known that, at various locations in the arterial system, stenoses may develop due to abnormal intravascular growths. For example, arteries may be narrowed by the development of atherosclerotic plaques and several investigators have suggested that this development is closely related to the hydrodynamics of blood flow through the artery [1–4].[1] Numerous other references can be found in the voluminous medical literature on atherosclerosis which refer to this aspect of the problem. It has been demonstrated experimentally [5, 6] that injury to the intima, even if it is minimal, can quickly lead to the development of gross lesions in the vicinity of the damaged area with a subsequent narrowing of the artery. Some investigators [7, 8, 9] have indicated that the initial injury to the artery may be due to localized turbulence and relatively large shearing stresses which occur at branches, or at any site at which the geometry changes in a relatively abrupt manner.

Although the specific reason for the initiation of a growth, which eventually projects into the lumen of the artery, is not known, it is clear that if such an event occurs the flow characteristics in the vicinity of the resulting protuberance may be significantly altered. There may now take place a "coupling" between the development of the stenosis and the corresponding change in the flow characteristics. It has been suggested that a localized change in pressure or shearing stress [10] may trigger certain biological mechanisms whereby the endothelial cells lining the arterial wall, and subendothelial cells, proliferate with a subsequent narrowing of the lumen. It has been demonstrated experimentally [11] that the narrowing of an artery by some external means can cause significant growths to develop at the artificially induced stenosis. In some cases cited, the vessel was almost completely occluded by the growth apparently induced by the stenosis.

As a stenosis develops there is the increased danger of complete occlusion due to the reduced lumen. In addition, there is an increased resistance to flow, with a possible corresponding reduction in flow to the particular vascular bed supplied by the artery. It is also known that at a stenosis a dilatation may develop downstream from the narrowed section (see, for example, reference [12]). Although the reason for poststenotic dilatation has not been definitely established, it has been attributed by some investigators to damage and weakening of the arterial wall by turbulence generated in the separated flow downstream from the narrowed lumen.

The following list summarizes the aforementioned important physiological effects that may be associated with the presence of a stenosis:

(*a*) Increased resistance to flow with possibly severe reduction in blood flow.
(*b*) Increased danger of complete occlusion.
(*c*) Abnormal cellular growth in the vicinity of the stenosis, thereby increasing the severity of the stenosis.
(*d*) Tissue damage leading to poststenotic dilatation.

It is the purpose of this paper to consider certain aspects of the fluid mechanics of the flow through a stenosis that are related to these effects.

Fluid Mechanics Phenomena

It is certainly true that in vivo vascular lesions leading to stenoses are not well-defined geometrical configurations. In general, complex three-dimensional flow patterns are developed near the stenosis which are virtually impossible to analyze. In some instances the stenosis is known to be more "collar like" with some degree of axial symmetry and, for the purpose of this paper, it is assumed that the stenosis is axially symmetric. It is envisioned that the size of the stenosis initially increases with

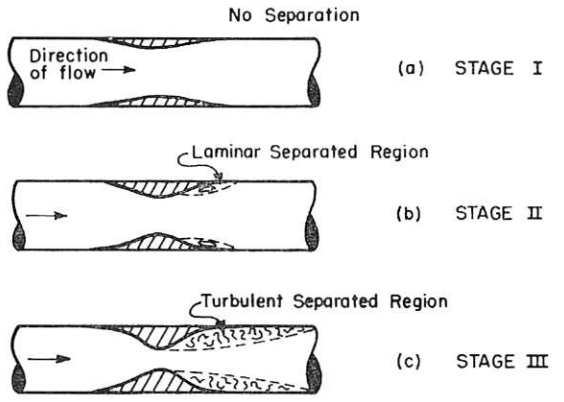

Fig. 1 Three stages of developing stenosis

[1] Numbers in brackets designate References at end of paper.

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time and eventually "stabilizes" to some fixed geometrical configuration.

During the early development of the stenosis, denoted as Stage I, the flow is not expected to separate from the protuberance [Fig. 1(*a*)] and the effect on the gross flow characteristics, such as increased pressure drop across the stenosis, is small. As the growth continues into the lumen, a second stage (Stage II) is reached at which the flow separates and a laminar backflow develops [Fig. 1(*b*)]. However, the separated region is small and localized. As the stenosis continues to develop, the separated region extends downstream and turbulence develops in the separated region (Stage III) as illustrated in Fig. 1(*c*). It is assumed that, during the growth, the flow through the stenosis remains essentially constant, and the flow preceding the stenosis is laminar. Since the flow through the arteries is actually pulsatile, the designated stage of the stenosis would be the most severe stage reached during a cycle.

It is expected that, for a stenosis in Stage I or II, the most physiologically significant effect would be abnormal cell proliferation. A stenosis in Stage III could induce all the effects summarized at the end of the preceding section. To proceed beyond this qualitative description of the general flow characteristics, numerous assumptions and idealizations must be made.

Idealized Model

As a first approximation to this problem, it is assumed that the flow is laminar and steady, the artery is of constant diameter (preceding and following the stenosis), and the fluid is Newtonian with a constant density, ρ, and viscosity, μ. It is further assumed that the stenosis develops in an axially symmetric manner due to some abnormal growth over a length, $2Z_0$, of the artery as shown in Fig. 2. The rate of growth into the lumen is expected to be a function of time, t, and of the longitudinal coordinate, z. Specifically, it is assumed that the time rate of change of the radius R is given in the form

$$\frac{\partial R}{\partial t} = -\alpha_0 \left(1 + \cos \frac{\pi z}{Z_0}\right) e^{-t/\tau} \tag{1}$$

for $-Z_0 \leq z \leq Z_0$, and

$$\frac{\partial R}{\partial t} = 0 \tag{2}$$

for all other z. The parameter, τ, is the "time constant" for the stenotic growth and α_0 is a constant. Equation (1) can be integrated to give

$$R = R_0 - \tau\alpha_0 (1 - e^{-t/\tau}) \left(1 + \cos \frac{\pi z}{Z_0}\right) \tag{3}$$

where $R = R_0$ for $t = 0$. It is noted that as $t \to \infty$

$$(R_0 - R)_{z=0} \to \delta_m \to 2\tau\alpha_0$$

where δ_m is the maximum projection of the stenosis into the lumen. Thus equation (3) can be written as

$$R = R_0 - \frac{\delta_m}{2} (1 - e^{-t/\tau}) \left(1 + \cos \frac{\pi z}{Z_0}\right) \tag{4}$$

In dimensionless form,

$$\frac{R}{R_0} = 1 - \frac{\delta_m}{2R_0} (1 - e^{-t/\tau}) \left(1 + \cos \frac{\pi z}{Z_0}\right) \tag{5}$$

When $t = \tau$, the height of the growth at $z = 0$ is

$$\delta = 0.632\delta_m$$

The general shape of the stenosis based on the preceding assumptions is shown in Fig. 2. Obviously, the assumed growth characteristics are arbitrary and many other possibilities exist.

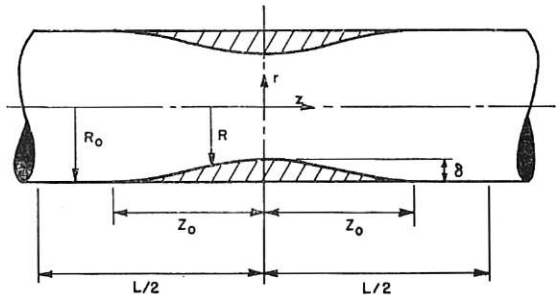

Fig. 2 Idealized geometry for stenosis

It should be noted that, although the stenosis varies with time, the variation is considered to be slow so that the flow can be assumed steady. Thus the fluid mechanics analysis depends only on the instantaneous condition of the stenosis.

With this idealized model, the flow through the stenosis is governed by the Navier-Stokes equations, which for axially symmetric flow reduce to

$$v_r \frac{\partial v_z}{\partial r} + v_z \frac{\partial v_z}{\partial z} = -\frac{1}{\rho}\frac{\partial p}{\partial z} + \frac{\mu}{\rho}\left(\frac{\partial^2 v_z}{\partial r^2} + \frac{1}{r}\frac{\partial v_z}{\partial r} + \frac{\partial^2 v_z}{\partial z^2}\right) \tag{6}$$

$$v_r \frac{\partial v_r}{\partial r} + v_z \frac{\partial v_r}{\partial z} = -\frac{1}{\rho}\frac{\partial p}{\partial r} + \frac{\mu}{\rho}\left(\frac{\partial^2 v_r}{\partial r^2} + \frac{1}{r}\frac{\partial v_r}{\partial r} + \frac{\partial^2 v_r}{\partial z^2} - \frac{v_r}{r^2}\right) \tag{7}$$

where v_r and v_z are the velocity components in the r and z-directions, respectively, and p is the pressure. In addition, the continuity equation

$$\frac{1}{r}\frac{\partial r v_r}{\partial r} + \frac{\partial v_z}{\partial z} = 0 \tag{8}$$

is required.

It is well known that these equations cannot be solved in general due to the nonlinearity of the connective acceleration terms. However, depending essentially on the size of the stenosis, certain terms in these equations are more important than others. It is therefore convenient to consider the case for a mild stenosis (Stage I) and for the more severe stenosis (Stages II, III) separately.

Analysis for Mild Stenosis

Equation (5), which defines the geometry of the stenosis, can be written

$$\frac{R}{R_0} = 1 - \frac{\delta}{2R_0}\left(1 + \cos \frac{\pi z}{Z_0}\right) \tag{9}$$

where

$$\delta = \delta_m(1 - e^{-t/\tau}) \tag{10}$$

and represents the instantaneous maximum height of the growth. During the initial development of the stenosis, $\delta/R_0 \ll 1$ and $v_r \ll v_z$, so that the Navier-Stokes equations can be simplified. The order-of-magnitude analysis given in the Appendix shows that, if the conditions

$$(a)\quad \mathrm{R_e}\frac{\delta}{Z_0} \ll 1 \quad (b)\quad \frac{\delta}{R_0} \ll 1 \quad (c)\quad \frac{R_0}{Z_0} \sim 0(1)$$

are satisfied, where $\mathrm{R_e}$ is the tube Reynolds number, then the governing equations can be approximated as

$$0 = -\frac{\partial p}{\partial z} + \mu\left(\frac{\partial^2 v_z}{\partial r^2} + \frac{1}{r}\frac{\partial v_z}{\partial r}\right) \tag{11}$$

$$0 = \frac{\partial p}{\partial r} \tag{12}$$

A stenosis satisfying these conditions will be denoted a mild stenosis. These are the same equations as obtained for flow through a tube of constant diameter and can be integrated to give

$$v_z = \frac{1}{4\mu}\frac{\partial p}{\partial z}(r^2 - R^2(z)) \tag{13}$$

i.e., the velocity distribution is parabolic at each cross section. This same approximation has been made in the analysis of flow through flexible tubes [13, 14]. It follows that

$$\frac{\partial p}{\partial z} = -\frac{8\mu}{\pi}\frac{Q}{R^4} \tag{14}$$

and the wall shearing stress, τ, is

$$\tau = \frac{R}{2}\frac{\partial p}{\partial z}$$

$$= -\frac{4\mu}{\pi}\frac{Q}{R^3} \tag{15}$$

where Q is the discharge through the tube.

The pressure drop ΔP across the stenosis between the cross sections $z = \pm L/2$ can be obtained from the integration of equation (14); i.e.,

$$\Delta P = \frac{8\mu Q}{\pi R_0^4}\int_{-L/2}^{L/2}\left(\frac{R}{R_0}\right)^{-4} dz \tag{16}$$

where R/R_0 is given by equation (9). Equation (16) can be expressed in the form

$$\frac{\Delta P}{Q} = \frac{8\mu}{\pi R_0^4} G_0$$

$$= \Lambda \tag{17}$$

where

$$G_0 = \int_{-L/2}^{L/2}\left(\frac{R}{R_0}\right)^{-4} dz \tag{18}$$

and Λ is defined as the "resistive impedance." For classical Poiseuille flow, in which $R = R_0$, it follows that $G_0 = L$, and

$$\Lambda_P = \frac{8\mu L}{\pi R_0^4} \tag{19}$$

The subscript, P, will be used throughout to denote Poiseuille flow. The ratio of the impedance with the stenosis to that without the stenosis is

$$\frac{\Lambda}{\Lambda_P} = \frac{G_0}{L} \tag{20}$$

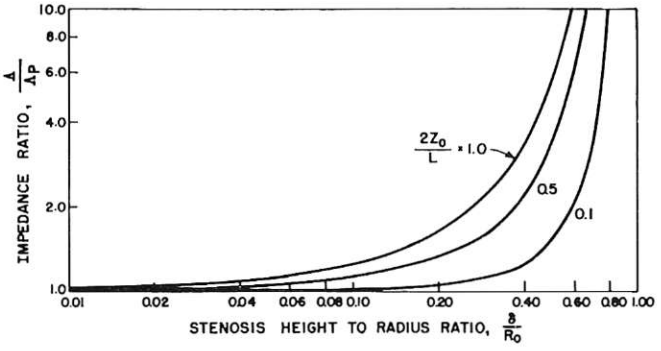

Fig. 3 Variation of impedance ratio with size of stenosis

The integral G_0 can be written

$$G_0 = L - 2Z_0 + \int_{-Z_0}^{Z_0}\left(\frac{R}{R_0}\right)^{-4} dz \tag{21}$$

The last integral in equation (21) is essentially of the form

$$\int_{-Z_0}^{Z_0}\left(\frac{R}{R_0}\right)^{-4} dz = \int_{-Z_0}^{Z_0}\frac{dz}{\left(a - b\cos\frac{\pi z}{Z_0}\right)^4} \tag{22}$$

where

$$a = 1 - \frac{\delta}{2R_0}$$

$$b = \frac{\delta}{2R_0}$$

and can be integrated by means of the theory of residues. The resulting equation for the resistive impedance ratio is

$$\frac{\Lambda}{\Lambda_P} = 1 - 2\frac{Z_0}{L} + 2\frac{Z_0}{L}$$

$$\times\left\{\frac{1}{b^4(\beta^2 - 1)^2}[1 - 12\gamma + 30\gamma^2 - 20\gamma^3]\right\} \tag{23}$$

where

$$\beta = \frac{a}{b}$$

and

$$\gamma = \frac{-\beta + \sqrt{\beta^2 - 1}}{2\sqrt{\beta^2 - 1}}$$

The impedance can now be determined as a function of the ratios $2Z_0/L$ and δ/R_0. The results are shown in Fig. 3. The curve labeled $2Z_0/L = 1.0$ shows directly the influence of the stenosis on the resistance. It is noted that, for a δ/R_0 equal to 0.1, the impedance has increased over that for a constant-diameter tube by approximately 25 percent. The curve labeled $2Z_0/L = 0.1$ is illustrative of the fact that, if the resistance over a long segment of artery is considered, the effect of the stenosis is very small until a certain value of δ/R_0 is exceeded. Beyond this critical value of δ/R_0, the presence of the stenosis rapidly becomes significant. It should be emphasized that, for the mild stenosis under consideration, the change in the actual pressure at a point in the artery due to the stenosis will still be small in comparison to the mean arterial pressure.

It follows from equation (15) that the ratio of the shearing stress at the wall of the stenosis to that for the corresponding Poiseuille flow is

$$\frac{\tau}{\tau_P} = \left(\frac{R}{R_0}\right)^{-3} \tag{24}$$

The variation in the wall shear at the midpoint of the stenosis, τ_0 (which represents the maximum variation), is given by the equation

$$\frac{\tau_0}{\tau_P} = \frac{1}{1 - 3\left(\frac{\delta}{R_0}\right) + 3\left(\frac{\delta}{R_0}\right)^2 - \left(\frac{\delta}{R_0}\right)} \tag{25}$$

The variation in the shear ratio with δ/R_0 is shown in Fig. 4. It is clearly noted from this figure that, for a given rate of flow, the wall shearing stress increases rapidly as the stenosis increases in size. For $\delta/R_0 = 0.1$, the wall shear is approximately 37 percent higher than the corresponding "normal" shear; for $\delta/R_0 = 0.2$, the increase is approximately 95 percent. If cellular growth or

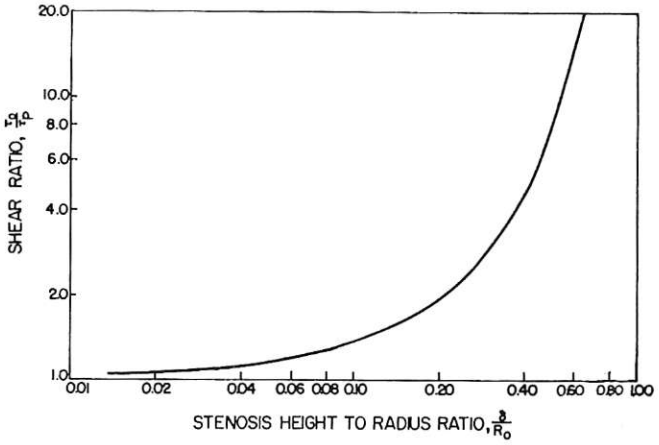

Fig. 4 Variation of shear ratio with size of stenosis

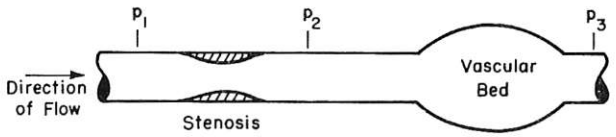

Fig. 5 Schematic representation of developing stenosis in artery supplying vascular bed

autoregulation is sensitive to changes in the wall shearing stresses, it is apparent that the presence of even a mild stenosis will be significant.

It is of interest to consider the changes that may occur during the development of a mild stenosis. An idealization of a segment of the vascular system is shown in Fig. 5. The artery containing the stenosis supplies blood to a particular vascular bed and it is assumed that the total pressure drop across the artery and the vascular bed, $p_1 - p_3$, is essentially constant. The total pressure drop can be expressed as

$$p_1 - p_3 = p_1 - p_2 + p_2 - p_3 = (\Lambda_{12})Q + (\Lambda_{23})Q \tag{26}$$

since

$$\frac{p_1 - p_2}{Q} = \Lambda_{12} \tag{27}$$

and

$$\frac{p_2 - p_3}{Q} = \Lambda_{23} \tag{28}$$

The impedance, Λ_{23}, represents the effective impedance of the vascular bed. It is assumed that

$$\Lambda_{23} = K(\Lambda_{12})_P$$

where $(\Lambda_{12})_P$ is the impedance of the artery supplying the vascular bed based on Poiseuille flow (no stenosis) and K is a constant. It is known that the pressure drop across the vascular bed is large in comparison to the pressure drop in a normal artery so that $K \gg 1$.

Equation (26) can now be written as

$$\frac{(\Lambda_{12})_P Q}{p_1 - p_3} = \left[\frac{\Lambda_{12}}{(\Lambda_{12})_P} + K\right]^{-1} \tag{29}$$

The first term in brackets is the impedance ratio for the stenosis as given in Fig. 3. To illustrate the manner in which the discharge, Q, may be altered with respect to time by the development of a stenosis, a specific example will be considered. Let a stenosis be specified for which the maximum height-radius ratio, δ_m/R_0, is 0.2, so that

$$\frac{\delta}{R_0} = 0.2\,(1 - e^{-t/\tau}) \tag{30}$$

In addition, let $2Z_0/L = 1.0$ and $K = 10.0$. The variation in the discharge parameter, the left side of equation (29), can now be plotted versus the dimensionless time, t/τ (Fig. 6). This illustrates the variation of Q versus time for a particular system since all other parameters are assumed constant. The small, and gradual, variation in discharge with time is noted. This result demonstrates that the development of a mild stenosis will have only a slight effect on the flow rate. The change may be completely negated by autoregulation of the impedance of the vascular bed in which K may change to maintain a constant discharge.

Although the theoretical results discussed in this section are only valid for a mild stenosis, i.e., $\delta/R_0 \ll 1$, the general form for the variation in the impedance ratio shown in Fig. 3 is expected to be similar for larger ratios. It is also anticipated that the potentially variable impedance of the vascular bed can compensate for the changing impedance due to the stenosis only to some critical value of K. Beyond this point, the discharge will begin to be significantly affected by the changing stenosis so that, for a more severe stenosis, the variation in discharge with time may appear, as illustrated in Fig. 6. This must be considered an approximate result for $\delta_m/R_0 = 0.8$. These general trends have been observed experimentally [15, 16].

The expression for the maximum wall shearing stress in the stenosis can be written as

$$\frac{\tau_0(\Lambda_{12})_P \pi R_0^2}{4\mu(p_1 - p_3)} = \left[\frac{\Lambda_{12}}{(\Lambda_{12})_P} + K\right]^{-1} \left(\frac{R}{R_0}\right)^{-3} \tag{31}$$

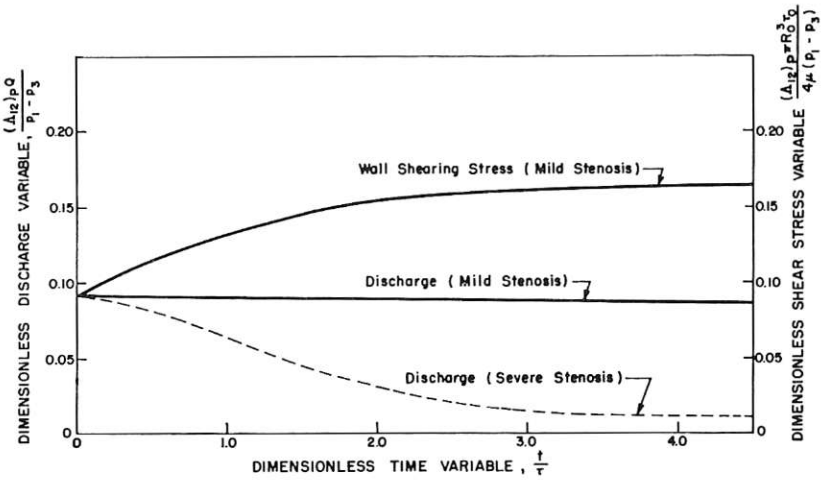

Fig. 6 Variation in wall shearing stress and discharge with time

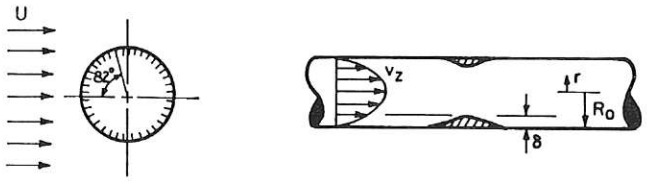

Fig. 7 Nomenclature for characterizing separation phenomenon

The dimensionless shear is plotted versus the dimensionless time variable in Fig. 6 for the case $\delta_m/R_0 = 0.2$, $2Z_0/L = 1.0$, and $K = 10.0$. It is observed that the shear increases rapidly with time although the discharge is decreasing very slowly. For $t = \tau$, the shear has increased approximately 50 percent whereas the discharge has decreased by approximately 2 percent.

Prediction of Separation

The analysis given in the preceding section is based on the condition that viscous forces are much larger than inertial forces. For steady flow, inertial effects are due to the convective acceleration terms in the Navier-Stokes equations. It is clear that, as the size of the stenosis increases, or as the Reynolds number increases, the importance of the inertial terms increases and they can no longer be neglected. Unfortunately, if these terms are retained, the resulting equations are nonlinear and cannot be readily solved. No solution to this problem which takes into account the nonlinear terms is available. Two important effects due to inertial forces are: (*a*) Lowered pressure at narrowed section of stenosis due to the Bernoulli effect; and (*b*) separation. It is thought that separation may play an important role in the development of the stenosis. In the separated region near the wall, the flow direction will actually be reversed from the main stream direction, thereby leading to a complete reversal in the direction of the shearing stresses acting on the cells lining the artery. For pulsating flow, it is entirely possible that at a given location along the wall the shearing stress will vary with time and alternate in direction. The importance of this phenomenon in cellular processes is unknown but conceivably important.

A rough approximation of the conditions under which separation may occur can be made from an analysis given by Goldstein [18]. It is known that, for flow around a solid body such as an infinitely long circular cylinder, the condition for separation is characterized by the Reynolds number Ua/ν, where U is the characteristic uniform approach velocity [Fig. 7(*a*)], a is a characteristic length (either radius or diameter of the cylinder), and ν the kinematic viscosity. For small Reynolds numbers, the separation point is near the rear stagnation point. As the Reynolds number increases, the separation point moves forward and stabilizes at approximately 82 deg, measured from the forward stagnation as shown in Fig. 7(*a*). For a cylinder in a uniform stream, the critical Reynolds number for the initial separation, as computed from a numerical analysis [17], is approximately 5 (where the characteristic length is the radius). At a Reynolds number of approximately 25, well-defined vortexes have developed behind the cylinder and extend downstream to form a wake.

As outlined in Goldstein, the characteristic velocity for the protuberance is assumed to be given by the equation

$$v_\delta = 2U\left\{1 - \left(1 - \frac{\delta}{R_0}\right)^2\right\}$$

and

$$v_\delta \cong 4U\left(\frac{\delta}{R_0}\right) \tag{32}$$

for small values of δ/R_0. This result is based on the following assumptions: (*a*) The velocity distribution preceding the obstruction is parabolic; and (*b*) the appropriate velocity to characterize separation is the velocity upstream from the obstruction at a position corresponding to the top of the obstruction. The Reynolds number

$$(\mathrm{R_e})_\delta = \frac{\delta v_\delta}{\nu} = 4\left(\frac{\delta}{R_0}\right)^2 \frac{UR_0}{\nu} \tag{33}$$

is now defined. It is assumed that, when this Reynolds number reaches some critical value, $\mathrm{R_{crit}}$, separation will occur. Thus, the condition for separation is

$$\frac{\mathrm{R_{crit}}}{4} = \left(\frac{\delta}{R_0}\right)^2 \mathrm{R_e} \tag{34}$$

The value of $\mathrm{R_{crit}}$ must be determined experimentally. As noted previously, separation apparently starts at approximately 5 for a circular cylinder in a uniform stream. Limiting conditions for δ/R_0 and $\mathrm{R_e}$ for various values of $\mathrm{R_{crit}}$ are given in Fig. 8. The approximate nature of this analysis should be recognized and values obtained from Fig 8 can only be used as rough estimates for predicting separation. It is apparent from this figure that, even for a mild stenosis, separation may occur at a relatively small Reynolds number. For example, for $\delta/R_0 = 0.1$, the limiting value of the pipe Reynolds number, $\mathrm{R_e}$, is approximately 130 (based on $\mathrm{R_{crit}} = 5$).

Discussion and Summary

The development of a stenosis in an artery can obviously create many serious problems and, in general, disrupt the normal function of the circulatory system. In this paper, certain aspects of the fluid mechanics of flow through an axially symmetric

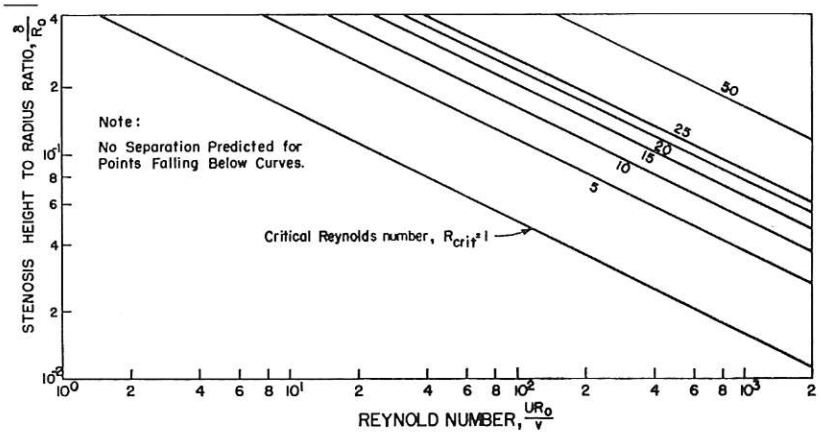

Fig. 8 Variation of separation Reynolds number with size of stenosis

stenosis are considered. Flow through a tube of nonconstant cross section presents an extremely difficult fluid mechanics problem which in general has not been solved. As an initial step in the analysis of this problem, an approximate solution that is valid for a mild stenosis, having a specific, time-dependent, geometrical configuration, is considered in detail. Since the flow is treated as quasi-steady, in the sense that flow unsteadiness due to the time-dependent boundary condition is neglected, the growth of the stenosis plays no role in the fluid mechanics analysis. However, it should be noted that, although the growth rate, as characterized by the parameter, τ, is not important to the fluid mechanics of the problem, the rates at which variables such as pressure and shearing stress are changing may play an important role in certain cellular processes. By formulating the problem in terms of a time-dependent stenosis, these rates may be computed.

Frequently in the study of hemodynamics, the flow is considered to be steady as was done in this paper. Since the flow is actually pulsatile, this represents a serious approximation that may lead to erroneous conclusions [19]. The steady-flow assumption is only valid for the case in which the ratio of the inertial forces (due to the pulsating flow) to the viscous forces is small. The index for this ratio is the dimensionless parameter $R_0\sqrt{\omega/\nu}$, where ω is the characteristic frequency of the pulsations. It is expected that, if $R_0\sqrt{\omega/\nu}$ is of the order of unity, then the flow can be treated as quasi-steady. This condition is usually only satisfied in the smaller arteries. Therefore, in addition to the limiting conditions on the Reynolds number and stenosis geometry previously given, this additional restriction must also be imposed in order for the results presented to be applicable to the flow of blood in the arteries.

It is clear that the prediction of the flow characteristics in a stenosis is complicated, and numerous simplifying assumptions are required to establish a tractable model. The results presented in this paper represent an initial step in the analysis of this interesting problem.

Acknowledgments

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APPENDIX

The Navier-Stokes equations, equations (6) and (7), and the continuity equation, equation (8), are expressed in dimensionless form by means of the transformations

$$r = R_0\bar{r} \qquad z = Z_0\bar{z} \qquad p = \rho U^2\bar{p}$$
$$v_r = \frac{U\delta}{Z_0}\bar{v}_r \qquad v_z = U\bar{v}_z \tag{35}$$

where U is a characteristic velocity in the z-direction. With these transformations, the dimensionless space variables and the velocity components have an order of magnitude of approximately unity or less. Symbolically, this order of magnitude will be denoted, 0(1). If the functions for $\bar{v}_r$ and $\bar{v}_z$ are smooth and continuous, the derivatives of the functions will also have an order of magnitude of unity [20]. The transformed equations of motion and the continuity equation become

$$\left(\frac{\delta}{R_0}\right)\bar{v}_r\frac{\partial\bar{v}_z}{\partial\bar{r}} + \bar{v}_z\frac{\partial\bar{v}_z}{\partial\bar{z}} = -\frac{\partial\bar{p}}{\partial\bar{z}} + \left(\frac{\mu}{\rho UR_0}\right)\left(\frac{Z_0}{R_0}\right)\left\{\frac{\partial^2\bar{v}_z}{\partial\bar{r}^2} + \frac{1}{\bar{r}}\frac{\partial\bar{v}_z}{\partial\bar{r}} + \left(\frac{R_0}{Z_0}\right)^2\frac{\partial^2\bar{v}_z}{\partial\bar{z}^2}\right\} \tag{36}$$

$$0\left(\frac{\delta}{R_0}\right) \quad 0\left(\frac{\delta}{R_0}\right) \qquad 0\left(\frac{\mu}{\rho UR_0}\frac{Z_0}{R_0}\right)\left\{0(1) \quad 0(1) \quad 0\left(\frac{R_0^2}{Z_0^2}\frac{\delta}{R_0}\right)\right\}$$

$$\left(\frac{\delta}{R_0}\right)^2\bar{v}_r\frac{\partial\bar{v}_r}{\partial\bar{r}} + \left(\frac{\delta}{Z_0}\right)\left(\frac{R_0}{Z_0}\right)\bar{v}_z\frac{\partial\bar{v}_r}{\partial\bar{z}} = -\frac{\partial\bar{p}}{\partial\bar{r}} + \left(\frac{\mu}{\rho UR_0}\right)\left(\frac{\delta}{Z_0}\right)\left\{\frac{\partial^2\bar{v}_r}{\partial\bar{r}^2} + \frac{1}{\bar{r}}\frac{\partial\bar{v}_r}{\partial\bar{r}} + \left(\frac{R_0}{Z_0}\right)^2\frac{\partial^2\bar{v}_r}{\partial\bar{z}^2} - \frac{\bar{v}_r}{\bar{r}^2}\right\} \tag{37}$$

$$0\left(\frac{\delta^2}{R_0^2}\right) \qquad 0\left(\frac{\delta}{Z_0}\frac{R_0}{Z_0}\right) \qquad 0\left(\frac{\mu}{\rho UR_0}\frac{\delta}{Z_0}\right)\left\{0(1) \quad 0(1) \quad 0\left(\frac{R_0^2}{Z_0^2}\right) \quad 0(1)\right\}$$

$$\left(\frac{\delta}{R_0}\right)\frac{1}{\bar{r}}\frac{\partial \bar{r}\,\bar{v}_r}{\partial \bar{r}} + \frac{\partial \bar{v}_z}{\partial \bar{z}} = 0 \qquad (38)$$

$$0\left(\frac{\delta}{R_0}\right) \qquad 0\left(\frac{\delta}{R_0}\right)$$

The estimated orders of magnitude of the various terms in these equations are shown immediately under the equations. From equation (36), it is noted that, if

$$\frac{1}{\mathrm{R_e}}\frac{Z_0}{R_0} \ll \frac{\delta}{R_0}$$

or

$$\mathrm{R_e}\frac{\delta}{Z_0} \ll 1$$

where $\mathrm{R_e} = UR_0/\nu$, then the inertial terms are much smaller than the viscous terms. Furthermore, if

$$\left(\frac{R_0}{Z_0}\right)^2\left(\frac{\delta}{R_0}\right) \ll 1$$

then the last of the viscous terms in equation (36) is negligible in comparison with the first two. For

$$\frac{R_0}{Z_0} \sim 0(1)$$

it follows that this condition is satisfied if

$$\frac{\delta}{R_0} \ll 1$$

An inspection of the equation of motion for the r-direction, equation (37), reveals that

$$\frac{\partial \bar{p}}{\partial \bar{r}} \ll \frac{\partial \bar{p}}{\partial \bar{z}}$$

and the variation of the pressure in the r-direction may be neglected in comparison to the variation in the z-direction. The continuity equation indicates that, if

$$\frac{\delta}{R_0} \ll 1$$

then

$$\frac{\partial \bar{v}_z}{\delta \bar{z}} \ll 1$$

Therefore, with the conditions specified, i.e.,

$$\mathrm{R_e}\frac{\delta}{Z_0} \ll 1 \qquad \frac{R_0}{Z_0} \sim 0(1) \qquad \frac{\delta}{R_0} \ll 1$$

the governing equations, rewritten in terms of the original variables, are

$$0 = -\frac{\partial p}{\partial z} + \mu\left(\frac{\partial^2 v_z}{\partial r^2} + \frac{1}{r}\frac{\partial v_z}{\partial r}\right) \qquad (39)$$

$$0 = -\frac{\partial p}{\partial r} \qquad (40)$$

$$0 = \frac{\partial v_z}{\partial z} \qquad (41)$$

The boundary conditions are

$$v_z = 0 \quad \text{at} \quad r = R$$

and

$$\frac{\partial v_z}{\partial r} = 0 \quad \text{at} \quad r = 0$$