CHAPTER VI

EFFECTS OF LOCAL VISCOSITY VARIATIONS ON FLOW AND DIFFUSION IN CAPILLARIES WITH MILD STENOSIS

INTRODUCTION

Systematic studies related to flow of blood vessels started towards the beginning of the present century. Quite a few analytical as well as experimental investigations with different perspectives have already been carried out over the years. Interest in studies of this particular domain of biomechanics has been magnified a great deal with the advent of the discovery that many cardiovascular diseases are closely associated with the flow conditions in the blood vessels. One major type of flow disorder results from the coarctation of the aorta in the arterial lumen, that is the development of the intra-vascular plaques on the internal surfaces of the arterial walls, narrows the artery and the disease is termed as 'stenosis', on the other hand, vascular plaques are generated when the nature of blood flow changes from its usual state to a disturbed flow conditions due to the presence of a stenosis in an artery.
Diagnosis of arterial diseases is usually carried out by the method of X-Ray angiography which essentially consists of inserting a catheter into the blood vessel and passing X-Ray opaque dye through the catheter. X-Ray cinemagraphy is used to map the arterial lumen and for the perfusion of the artery. As pointed out by Willcutt (1968), these angiographic methods of detecting arterial diseases involve considerable risk of morbidity. So any attempt to develop non-invasive techniques for detecting arterial diseases like stenoses is greatly appreciated. Such a technique involves the prediction of the rate of blood flow through the subject vessel and the degree of occlusion of the lumen.

Over a period of years, due to localized accumulation of material within or beneath the intima (i.e., the inner surface of the arteries), the deposits sometimes turn into atherosclerotic plaques, greatly reducing the arterial diameter. In this situation, the flow to the vascular bed is significantly disturbed. It has been seen through clinical and sub-clinical examinations that such a condition can lead to hemorrhage and local thrombosis. Although the exact mechanism of such deposits is somewhat unclear, it has been observed that certain parts of the human arterial tree like the carotid coronary and femoral arteries as well
as the bifurcation of the aorta at the iliac arteries are predisposed to deposits of this sort.

The mean flow velocity attains a maximum where the cross-sectional area is smallest. At this point the hydrostatic pressure is minimum, the pressure drop is partially recovered in the diverging area of the stenotic region. However, the pressure drop depends not only on the geometry of the region but also on the flow through it.

Human circulatory system is very complex and it is very difficult to incorporate all the physical and biological factors such as taperness, branchings, pulsatility, distensibility and the characteristics of the bio-fluid. Neglecting taperness and pulsatility in comparison to the changes induced by the stenosis and the assumption of rigid-walled symmetric constriction are reasonable approximations for the study of localised flow characteristics as considered by Young (1979).

It was indicated by May et.al (1963) as well as by Ekbl and Schwartz (1970) that higher resistance to flow is of particular importance when the stenosis takes a severe form. Pressure in the stenotic region was also discussed by Texon (1963). The effect of turbulence was examined by Wesolowski et.al (1965) and more recently by Lees and Dewey (1970), Roach (1972) and Nerem and Seed (1972). An attempt
towards a systematic study of the flow around a stenosis seems to be first initiated by Young (1968). The effect of flow separation was examined by Forrester and Young (1970) as well as Lee and Fung (1970), Bougher and Roach (1971), observed that isolated arterial strips undergo marked changes in elastic properties when vibrated at certain frequencies, they remarked that the broad frequency band width of fluctuating forces acting on the arterial wall down stream of a stenosis may be responsible for the development of post-stenotic dilatation. Alteration of the vessel wall structure, the associated potential for plaque formation and the wall dilation caused due to abnormal flow environments were studied by several investigators.

Low shear stress regions were examined by Merrill (1965) and Caro (1973) disorders of flow patterns due to the presence at stenosis were investigated by Fry (1973) and Giddens et.al (1976). They indicated that disturbances to the flow field could be generated due to fluctuation in the pulse rate, Reynolds number or vessel wall contour in physiologic conditions. Several other investigators carried out theoretical studies on various aspects of wall deformation and blood flow in arteries, [Young and Tsai (1973), Margon and Young (1974), Deshpande et.al (1976), Fernandez et.al (1976), Kandrapa Davis (1976), Kaimal (1981),
Shukla et al. (1980), Perkkio (1983), Tandon et al. (1982, 1986), Chaturani and Samy (1986), Bitoun and Bellet (1986), Misra and Singh (1983, 1984), Misra and Chakravarty (1986), considering it as a Newtonian and non-Newtonian fluid. Bugliarello and Sevilla (1970) have experimentally shown that blood flow through small diameter tubes has blunted velocity profiles and a cell free plasma layer near the tube wall. The fluid in this layer is not separate Newtonian and non-Newtonian fluid but it is actually the suspending the medium occurring near the wall forming the peripheral layer. Therefore the viscosity of the peripheral layer fluid must be identical to that of the suspending medium of the overall physiological fluid under consideration.

The viscosity of the peripheral layer, i.e., plasma viscosity has proved to be a very important parameters as compared to whole blood viscosity which depend upon the several other factors according Ditzel (1968). The main rheological changes in diabetic patients are an elevation of the plasma viscosity shown by Coagen et al (1961) and the development of diseases in the diabetic subjects is two or three times faster. Most recently Perkkio and Keskinen (1983) studied the effects of the viscosity-concentration dependence and of the concentration profile on blood flow through a vessel with stenosis. The flow resistance and
the wall shear stress have been found to be smaller than in the two-fluid model with constant viscosities. Kang and Eringen (1976) have also discussed the effects of local variation of the concentration of the suspended particles of the blood on the various parameters in micropolar fluid. Therefore, this chapter presents modifications on earlier work so as to consider the viscosity depending upon the concentration of undissolved cells. Since some parts of the human arterial wall have been recognised as favorite sites so far as the formation of stenosis is concerned, related studies of abnormalities in the arterial wall deserve special attention.

MATHEMATICAL MODELLING

Let us consider the flow of blood through a rigid circular tube of radius \( R(z) \). It is assumed that the stenosis develops in the arterial wall in axially symmetric manner, and depends on the axial distance \( z \) and the thickness of its growth. In such a case the geometry of the wall surface can be described as by Young (1968):

\[
\frac{R}{R_0} = 1 - \frac{\delta_s}{2R_0} \left[ 1 + \cos \frac{2\pi}{L_0} (z-d-L_0) \right] ; \quad d \leq z \leq L_0 + d
\]

\[= 1; \quad \text{elsewhere.} \quad (6.1)\]
where $L_0$ is the length of the stenosis, $d$ indicates its locations and $\delta_s$ is the maximum height of the stenosis. The flow is considered to be steady, fully developed and laminar. [Figure 6.1].

We consider a slow viscous flow of suspensions of cells. As a reasonable approximation for the viscosity function we assume

$$\mu = \mu_0 [1 + \alpha C]$$  \hspace{1cm} (6.2)$$

where $\mu_0$ is the viscosity of the plasma, $\alpha$ is a constant parameter ($0.0 \leq \alpha \leq 2.5$), and $C$ is the concentration of the undissolved cells, and is expressed as,

$$D\left(\frac{\partial^2 C}{\partial r^2} + \frac{1}{r} \frac{\partial C}{\partial r}\right) + m = 0$$  \hspace{1cm} (6.3)$$

where,

$D$ is the diffusion coefficient of the undissolved cells and $m$ represents the generation or degradation of cells.

The concentration equation for the solute is expressed as follows:

$$\frac{\partial C_1}{\partial t} + U \frac{\partial C_1}{\partial z} = D_1 \left(\frac{\partial^2 C_1}{\partial r^2} + \frac{1}{r} \frac{\partial C_1}{\partial r}\right)$$  \hspace{1cm} (6.4)$$
Fig. 6.1 Geometry of the artery with stenosis.
where, $C_1$ represents the concentration of the solute, $U$ is the axial velocity and $D_1$ is the diffusion coefficient for the solute under consideration in the blood.

The basic equation governing the flow of blood in the arterial system is given by (Young 1968)

$$0 = - \frac{dp}{dz} + \frac{1}{r} \frac{\partial}{\partial r} \left[ \mu \frac{\partial U}{\partial r} \right]$$

(6.5)

where $U$ is the axial velocity, $p$ is the fluid pressure and $\mu$ is the viscosity function.

To solve the above system of equations (6.3) to (6.5) the appropriate boundary conditions in mathematical form are:

$$\frac{\partial C_1}{\partial r} = 0 \quad \text{at} \ r = 0$$

$$D_1 \frac{\partial C_1}{\partial r} = V \cdot N \cdot C_1 \quad \text{at} \ r = R$$

$$\frac{\partial C_1}{\partial r} = 0 \quad \text{at} \ r = 0$$

$$D_1 \frac{\partial C_1}{\partial r} = V \cdot N \cdot C_1 \quad \text{at} \ r = R \quad (6.6)$$

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

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

$$P = P_0 \quad \text{at} \ z = 0$$

$$P = P_L \quad \text{at} \ z = L$$
where \( \mathbf{V} \) is the fluid velocity, \( N \) is the retention parameter (\( N \leq 1 \)), \( P_0 \), \( P_L \) are the pressures at \( z = 0 \) and \( z = L \) respectively.

The expression for the concentration \( C \) is obtained by solving equation (6.3) with the boundary conditions (6.6):

\[
C = \frac{m}{4DV} [V(R^2-r^2) - 2DR]
\]  

(6.7)

Substituting equation (6.7) in (6.2) we have

\[
\mu = \nu_0 [1 + \alpha \left( \frac{m}{4DV} (V(R^2-r^2) - 2DR) \right)]
\]  
or

\[
\mu = \nu_0 [A_1 r^2 + A_2]
\]  

(6.8)

where

\[
A_1 = - \frac{\alpha m}{4D}
\]

\[
A_2 = 1 + \frac{\alpha m}{4DV} (VR^2 - 2DR)
\]

Now on solving equation (6.5) with the boundary conditions (6.6), we get

\[
U = \left( - \frac{1}{2} \frac{dP}{dz} \right) \int_{r}^{R} \frac{rdr}{\mu(r)}
\]  

(6.9)

hence
\[ U = -\frac{1}{4A_2\mu_0} \left[ (R^2-r^2) - \frac{A_1}{2A_2} (R^4-r^4) \right] \frac{dP}{dz} \] (6.10)

The flow flux \( Q \), is defined as,

\[ Q = \int_0^R 2\pi r u dr \]

\[ Q = -\frac{\pi}{2A_2\mu_0} \left[ \frac{R^4}{4} - \frac{A_1 R^6}{3A_2} \right] \frac{dP}{dz} \] (6.11)

The pressure gradient is written as follows:

\[ \frac{dP}{dz} = -\frac{2Q}{\pi I(z)} \] (6.12)

where

\[ I(z) = \int_0^R \frac{r^3 dr}{\mu(r)} \] (6.13)

Integrating equation (6.12) and using the conditions (6.6)

\[ P_0 - P_L = \frac{2Q}{\pi} \int_0^L \frac{dz}{\pi(z)} \] (6.14)

The resistance to flow, \( \lambda \), is defined as follows (Young 1968):

\[ \lambda = \frac{P_0 - P_L}{Q} \] (6.15)

From equations (6.1), (6.14) and (6.15), \( \lambda \) is given by

\[ \lambda = \frac{2}{\pi} \left[ \frac{L - L_0}{I_0} + \int_{d+L_0}^L \frac{dz}{I(z)} \right] \] (6.16)
where

\[ I_0 = \int_0^R r^3 \frac{dr}{\mu(r)} \]  \hspace{1cm} (6.17)

The shearing stress at the wall can be defined as

\[ \tau_R = [\mu(r) \cdot \frac{du}{dr}]_{r=R(z)} \]  \hspace{1cm} (6.18)

which on using equations (6.9) and (6.14) gives the wall shear, \( \tau_s \) as follows

\[ \tau_s = \frac{R(z)}{\pi I(z)} \]  \hspace{1cm} (6.19)

Following Tandon et.al (1979) the apparent viscosity, \( \frac{\mu}{\mu_0} \), is defined as follows:

\[ \frac{\mu}{\mu_0} = \frac{1}{R^4 \left[ \frac{4A_1}{A_2} - \frac{A_2^2}{3A_2^2} \right]} \]  \hspace{1cm} (6.20)

We solve equation (6.4), using the following non-dimensional quantities

\[ t_1 = \frac{t}{t}, \; \overline{t} = \frac{t}{U}, \; \overline{\tau} = \frac{z - Ut}{L}, \; \eta = \frac{r}{R_0} \] \hspace{1cm} (6.21)

\[ \overline{C}_1 = \frac{C_1}{C_0}, \; \overline{R}' = \frac{R}{R_0} \]

hence, equation (6.4) becomes,
\( \frac{1}{t} \cdot \frac{\partial \bar{c}_1}{\partial t} + \nu \frac{\partial \bar{c}_1}{\partial \bar{\eta}} = \frac{D_1}{R_0} \left( \frac{\partial^2 \bar{c}_1}{\partial \eta^2} + \frac{1}{\eta} \frac{\partial \bar{c}_1}{\partial \eta} \right) \) \hspace{1cm} (6.22)

To solve this equation, we use the following non-dimensional boundary conditions,

\[
\frac{\partial \bar{c}_1}{\partial \eta} = 0 \quad \text{at } \eta = 0 \tag{6.23}
\]

\[
\bar{D}_1 \frac{\partial \bar{c}_1}{\partial \eta} = V \cdot N \cdot \bar{c}_1 \quad \text{at } \eta = R/R_0
\]

If the Taylor's longitudinal condition is valid in this problem, the partial equilibrium may be assumed at any cross-section of the artery and the variation in \( \bar{c}_1 \) with \( r \) is obtained from equation (6.22) and is of the form

\[
\left( \frac{\partial^2 \bar{c}_1}{\partial \eta^2} + \frac{1}{\eta} \frac{\partial \bar{c}_1}{\partial \eta} \right) = \frac{R_0^2}{\bar{D}_1 L} \cdot \frac{\partial \bar{c}_1}{\partial \xi} \hspace{1cm} (6.24)
\]

where

\[
\nu = U - \bar{U} \hspace{1cm} (6.25)
\]

and

\[
\bar{U} = \frac{2}{(R')^2} \int_0^{R'} \eta u \, d\eta \]

\[
\bar{U} = \frac{R_0^4 (R')^2}{4 \mu_0 A_2 L} \frac{dP}{d\xi} \hspace{1cm} (6.26)
\]

To solve equation (6.24), we use the boundary conditions (6.23), hence, we get
The volumetric rate at which the solute is transported across a section of the artery of unit breath is

\[ M = 2\pi R_0^2 \int_0^{R'} \overline{C}_1 \nu \eta \, d\eta \]  

(6.29)

we get on integration

\[ M = \frac{R_0^4 \overline{U}}{D_1 L^2} \left[ \frac{0.0182 \pi R_0^2 (R')^6}{A_2 \mu_0} + \frac{0.0088 \pi R_0^4 (R')^6}{A_2 \mu_0} \right] L \\
- \frac{2\pi A_4 (R')^2}{R_0^2} + \frac{A_5 \pi (R')^4}{2A_2 \mu_0} \left( \frac{\delta C_1}{\delta \xi} \right) L^2 \]  

(6.30)

where,

\[ A_4 = \frac{(R')}{2A_2 \mu_0 L^2} \frac{d\overline{P}}{d\xi} \left[ \frac{10 \nu \cdot N(R')}{144} - \overline{D}_1 \right] \]

\[ A_5 = \frac{1}{L^2} \frac{d\overline{P}}{d\xi} \left[ \frac{10 \nu \cdot N}{144} - \overline{D}_1 \right] \]

Following Taylor (1953), we assume that the variations
of $\bar{c}_1$ with $\eta$ are small compared with those in the longitudinal direction and if $\bar{c}_{1m}$ is the mean concentration over a section, $\frac{\partial \bar{c}_1}{\partial \xi}$ is indistinguishable from $\frac{\partial \bar{c}_{1m}}{\partial \xi}$ so that (6.30) may be written as

$$M = \frac{R_0^4 \bar{c}_1}{D_1 L^2} \left[ \frac{0.0182 \pi R_0^2 (R')^6}{A_2 \mu_0} + \frac{0.0088 \pi R_0^4 (R')^6}{A_2 \mu_0} \right] + \frac{2 \pi A_2 (R')^2}{R_0^2} + \frac{A_5 \pi (R')^4}{2 A_2 \mu_0} \left( \frac{L}{2} \right)^2 \frac{\partial \bar{c}_{1m}}{\partial \xi}$$

This shows that $C_{1m}$ is dispersed relative to a plane which moves with the velocity $\bar{U}$ exactly as though it were being diffused by a process which obeys the same law as molecular diffusion but with a relative diffusion coefficient $D^*$, called Taylor-diffusion coefficient, where

$$D^* = \frac{R_0^4 \bar{U}}{D_1} F_0$$

and

$$F_0 = \frac{1}{L^2} \left[ \frac{0.0182 \pi R_0^2 (R')^6}{A_2 \mu_0} + \frac{0.0088 \pi R_0^4 (R')^6}{A_2 \mu_0} \right] L + \frac{2 \pi A_4 (R')^2}{R_0^2} + \frac{A_5 \pi (R')^4}{2 A_2 \mu_0} \left( \frac{L}{2} \right)^2$$

The fact that no material is lost in the process is expressed by the continuity equation for $\bar{c}_{1m}$ namely
Fig. 6.2 Variation of apparent viscosity ($\mu/\mu_0$) with $z/L_0$ for different values of $\delta_s/R_0$.
where \( \frac{\delta}{\delta t} \) represents differentiation with respect to time at point where \( \xi \) is constant. Equation (6.33), using (6.26) becomes

\[
\frac{\delta C_{lm}}{\delta t} = D^* \frac{\delta^2 C_{lm}}{\delta \xi^2}
\]

which is the equation governing the longitudinal dispersion. Equation (6.34), using (6.26) can also be written in the form

\[
D^* = \frac{dp}{d\xi} \frac{R_0^6}{D_1} F_1
\]

where

\[
F_1 = -\frac{(R')^2}{8A_2 \mu_o L^3} \left[ \frac{0.0182 \cdot \pi \cdot R_0^2 (R')^6}{A_2 \cdot \mu_o} + \frac{0.0088 \cdot \pi \cdot R_0^4 (R')^6 \cdot L}{A_2 \cdot \mu_o} \right]
\]

\[
- \left( \frac{2 \cdot \pi \cdot A_4 \cdot (R')^2}{R_0^2} + \frac{A_5 \cdot \pi \cdot (R')^4}{2 \cdot A_2 \cdot \mu_o} \right) L^2 \right]
\]

RESULTS

Cardiovascular diseases are responsible for about fifty percent deaths. A knowledge of Apparent viscosity, Resistance
Fig. 6.3 Variation of apparent viscosity $(\mu/\mu_0)$ with $\delta_S/R_0$ for different $\alpha$.  

$z/L_0 = 0.5$, $m = 1$
Fig. 6.4 Variations of apparent viscosity ($\mu/\mu_0$) with $\delta_s/R_o$ for different $m$. 

- $z/L_0 = 0.5$, $\alpha = 1.0$
- $m = 3$
- Curves 1, 2, and 3 represent different values of $m$. 
to flow and wall shearing stress are therefore very important factors in locating various arterial diseases just at the developing stage. In this chapter, an attempt has been made to obtain the Apparent viscosity, Resistance to flow and shearing stresses for blood flow through growing stenosis.

The Apparent viscosity has been represented in figures 6.2, 6.3 and 6.4 for different parametric values. It may be observed that the apparent viscosity increases as the stenosis grows and remains constant outside the stenotic regions. Increase in the parameter $\alpha$ increases the viscosity $\mu$. It is seen from the figure 6.3 that increase in $\alpha$ increases the apparent viscosity as the stenosis develops. The effect of variations of $m$ representing the generation or degradation of the cells on apparent viscosity has been depicted in figure 6.4. Increasing values of $m$ represent growth of new cells i.e., generation. This, in turn, increases the viscosity. The effect of increasing values of $\delta_s/R_0$ is also obvious because narrowing the arteries would increase concentration of the suspended cells.

Resistance to flow depending on various parameters involved has been depicted in figures 6.5, 6.6 and 6.7. One may observe that the resistance to flow is larger in stenotic
Fig. 6.5 Variation of flow resistance ($\lambda$) with $L_0/L$ for different $\delta_s/R_0$. 

Flow Resistance ($\lambda$)

$\delta_s/R_0 = 0.25$

0.20

0.15

0.10

0.05

$L_0/L$

0 0.2 0.4 0.6 0.8 1.0
Fig. 6.6 Variation of resistance to flow (\(\lambda\)) with \(s_s/R_0\) for different \(\alpha\).
regions as compared to the non-stenotic regions. The resistance to flow increases with the growth of stenosis and with the increasing values of the parameters $\alpha$ and $m$. It may be observed that when $\alpha$ or $m$ increases the viscosity increases and this, in turn, increases the resistance to flow. Variation of resistance to flow with developing stenosis is similar to that obtained by Shukla et.al (1980) and Misra and Chakrvarty (1986).

Variations in wall shearing stress with the developing stenosis for different values of the parameters are presented in the figures 6.8 and 6.9. As the stenosis grows, the wall shearing stress increases in the stenotic regions. The results for increasing values of $\alpha$ are similar to those for resistance to flow.

Figures 6.2, 6.5 and 6.8 show that the curves are symmetric in the stenotic region about an axis along which the constriction of the arterial lumen is maximum. The apparent viscosity, resistance to flow and wall shearing stress become higher and higher till the constriction of the vessel wall attains its maximum and gradually diminishes towards the end of the stenotic region. The symmetry in the curves in the stenotic region may be attributed to be due to the assumed symmetry in the geometrical configuration of the stenosis. These results are similar to those obtained by Misra and Chakravorty (1986).
Fig. 6.7 Variation of resistance to flow ($\lambda$) with $\delta_s/R_0$ for different $m$. 

$\alpha = 1.0, \, z/L_0 = 0.5$
Fig. 6.8 Variation of shearing stress ($T_s$) with $z/L_0$ for different values of $\delta_s/R_0$. 

[Graph showing the variation of shearing stress ($T_s$) with $z/L_0$ for different values of $\delta_s/R_0$.]
Table 6.1
Variation of Axial Diffusivity Coefficients with the Parameter $\alpha$

<table>
<thead>
<tr>
<th>$\alpha$</th>
<th>$F_0 \times 10^7$</th>
<th>$F_1 \times 10^8$</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.1</td>
<td>0.3076492</td>
<td>0.4296865</td>
</tr>
<tr>
<td>0.2</td>
<td>0.2752104</td>
<td>0.3719138</td>
</tr>
<tr>
<td>0.3</td>
<td>0.1376367</td>
<td>0.0595819</td>
</tr>
<tr>
<td>0.4</td>
<td>0.1113996</td>
<td>0.0287105</td>
</tr>
<tr>
<td>0.5</td>
<td>0.1002790</td>
<td>0.0183993</td>
</tr>
<tr>
<td>0.6</td>
<td>0.0941325</td>
<td>0.0133088</td>
</tr>
<tr>
<td>0.7</td>
<td>0.0875386</td>
<td>0.0105041</td>
</tr>
<tr>
<td>0.8</td>
<td>0.0840581</td>
<td>0.0086157</td>
</tr>
<tr>
<td>0.9</td>
<td>0.0828691</td>
<td>0.0072943</td>
</tr>
<tr>
<td>1.0</td>
<td>0.00919072</td>
<td>0.0063199</td>
</tr>
<tr>
<td>1.1</td>
<td>0.0081113</td>
<td>0.0055727</td>
</tr>
<tr>
<td>1.2</td>
<td>0.008044</td>
<td>0.0049820</td>
</tr>
<tr>
<td>1.3</td>
<td>0.00798</td>
<td>0.0045036</td>
</tr>
<tr>
<td>1.4</td>
<td>0.000786</td>
<td>0.00410645</td>
</tr>
<tr>
<td>1.5</td>
<td>0.000629</td>
<td>0.0037766</td>
</tr>
<tr>
<td>1.6</td>
<td>0.000601</td>
<td>0.00034941</td>
</tr>
<tr>
<td>1.7</td>
<td>0.000887</td>
<td>0.0003250</td>
</tr>
<tr>
<td>1.8</td>
<td>0.000962</td>
<td>0.000303</td>
</tr>
<tr>
<td>1.9</td>
<td>0.000940</td>
<td>0.0002852</td>
</tr>
<tr>
<td>2.0</td>
<td>0.000409</td>
<td>0.0000169</td>
</tr>
</tbody>
</table>
Axial diffusivity (Taylor-diffusion coefficient) for the solute particles in the stenotic region as described by the equations (6.32) and (6.35) has been observed to depend on viscosity. The variations of the functions $F_0$ and $F_1$ has been presented in the table 6.1. As viscosity increases either through $\alpha$ or $m$, the values of $F_0$ and $F_1$ (effective dispersion coefficient) decrease very rapidly with viscosity.
Fig. 6.9 Variation of wall shearing stress ($\tau_s$) with $\delta_s/R_0$ for different $\alpha$.