5.1 INTRODUCTION

Catheterization in cardiac procedures is very common in medicine. The aim is to measure the arterial pressure or pressure gradient or the velocity of blood flow across the cross-section of an artery by injecting a dye in the X-ray examination of arterial network. In percutaneous transluminal coronary angioplasty a catheter with a balloon attached to the upstream tip of the catheter is used to clear the obstructions from the walls of a stenosed artery.

Catheter is a long narrow cylindrical tube of length of about 135cm. The diameter ranges from 1mm to 9mm depending on the site and mode of application in the arteries. It is inserted through a small incision at the appropriate place into a smaller blood vessel i.e., femoral artery close to the skin and threaded along it to the site of investigation. A small stain gauge pressure transducer, a sensing device to measure pressure or pressure gradient is positioned on the wall of the catheter. The transducer is usually situated at the tip of the catheter or some times towards the down stream end of the catheter. The diaphragm of the transducer serves as a spring and overshoots when it is displaced by a pressure signal. The measurement of arterial blood pressure / pressure gradient in routine clinical studies and experiments is usually done through a pressure transducer attached to the tip of the catheter (Gabe, 1972; Gang 1983, 1985). The velocity of blood or flow rate in arteries is measured through an electromagnetic or ultrasonic flow meter mounted at the
tip of the catheter (Mills, 1972; Hartley and Cole 1974; Cole and Hartley 1977). Various arterial diseases have been usually diagnosed by the method of X-ray angiography which essentially involves the insertion of a catheter into the blood vessels and passing a X-ray opaque dye through the catheter. Velocity of blood is measured on the basis of the rate of clearance of the dye after withdrawing the tip of the injecting catheter from the blood vessels. In coronary angioplasty, catheter is inserted through a constricted region and the balloon attached to the tip of the catheter is inflated such that it compresses and reduces the occlusion.

The insertion of a catheter into blood vessel changes the flow characteristics of blood. Frictional resistance to flow through the blood vessels increases and the pressure wave reflection at the tip of the catheter distorts the change of the pressure wave and hence the pressure or pressure gradient recorded by the transducer will differ from the corresponding pressure or pressure gradient in an uncatheterized artery.

The instantaneous aortic blood velocity with a catheter-tip pressure gauge was measured first by Fry et al. (1956), Womersely (1958) was the first to obtain the reflection coefficient of the pulse wave at the injections and at the tip of the catheter inserted in the arterial system through his study on the phenomenon of arterial pressure wave propagation.
Pieper in a series of papers (1964, 1966, 1968) studied the blood flow in pulmonary artery, coronary artery and to measure the left ventricular diameter using the catheter-tip blood flow meters. Bjorno and Patterson in a series of (1976a, b, 1977) conducted experimental investigations to understand the hemodynamic effects of catheterisation of blood vessels.

Kanai et al. (1970) studied the effect due to the reflection of pressure wave at the tip of the catheter and at the occluding point of the artery and the increase of the pressure wave attenuation produced by the insertion of a catheter into blood vessels. Their theoretical results are compared with the data obtained from experiments conducted on live dogs and reported large catheter induced errors. Mc Donald (1986) obtained theoretical results for the modification of pressure gradient in a femoral artery in the presence of catheters which were positioned coaxially and eccentrically with the artery. Their results indicated that the assumption that the rate of mass flow is same for both analyses carried with or without catheter. In a coaxial arrangement when the ratio of the radius of the catheter to that of artery is 0.6 a substantial increase in the pressure gradient is noticed. This is attributed to the assumption that the wall of the artery is rigid and the flow is fully developed. Further, if insertion of catheter in the artery results in a rate of mass flow with reduced amplitude and a reduced pressure gradient. Back (1994) studied the pressure drop, frictional resistance and wall shear-stress in a coronary artery in the
presence of a catheter and obtained the estimates for increased frictional resistance and pressure gradient due to catheterization. In all the above studies blood was modelled as a Newtonian fluid.

Dash *et al.* (1996) studied the changed flow pattern in a narrow artery in the presence of a catheter modelling blood as a Casson fluid. They presented the estimates for the increase in the frictional resistance in large blood vessels where the effect of yield stress can be neglected. It was shown that when the ratio of catheter radius to vessel radius varies in the range 0.7 the frictional resistance increased by a factor ranging from 3 to 33. For the same range of catheter radii, in small blood vessels where yield stress is significant the frictional resistance is increased by a factor of 7-121 when yield stress is as small as 0.05. It is shown that increase in size of the catheter and yield stress the frictional resistance increased substantially. Sankar and Hemalatha (2007) studied the pulsatile flow of Herschel Bulkley Fluid through catheterised arteries using perturbation method. They studied the effect of catheterization on the flow characteristics i.e., pressure drop, wall shear stress and frictional resistance to flow modelling blood as a Herschel Bulkley Fluid and obtained the results for power law fluid, Newtonian fluid and Bingham fluid as particular cases.

In this chapter the changed flow pattern in an artery in the presence of a catheter under the influence of a periodic body acceleration is studied. Blood is
modelled as a Newtonian fluid. The flow is studied under the action of a periodi
periodic pressure gradient produced by the heart and an external body acceler
acceleration. The results are used to obtain the estimate for the increase in fric
frictional resistance in an artery due to catheterization and the reduction in fric
frictional resistance due to the application of periodical body acceleration.

Section 5.2 deals with the mathematical formulation of the problem. Section 5.3
deals with the method of solution. The effects of catheter radius and amplitude of
the body acceleration on the velocity distribution, frictional resistance and wall shear stress are discussed in section 5.4. The concluding remarks are given in section 5.5.

5.2 MATHEMATICAL FORMULATION

Fig 5.1 shows the schematic diagram of the annular geometry. The radius of the outer tube is ‘a’ and that of the inner tube is ‘ka’ with k < 1. The flow in the annulus is assumed to be fully developed, steady and laminar. Assume that the flow is subjected to periodic body acceleration and the flowing blood is modelled as a Newtonian fluid.

The equations of motion governing the fluid flow is given by

\[ \rho \frac{\partial \bar{w}}{\partial t} = -\frac{\partial \bar{p}}{\partial z} + \rho G + \mu \nabla^2 \bar{w} \]  

(5.1)
where \( V^2 = \frac{1}{r} \frac{\partial}{\partial r} \left( r \frac{\partial}{\partial r} \right) \), \( \tilde{z} \) denotes the axial co-ordinate, \( \tilde{t} \) is time, \( \tilde{p} \) is pressure, \( \tilde{w} \) is the axial velocity, \( \rho \) is the density of blood, \( \mu \) is the co-efficient of viscosity of blood.

The pressure gradient at any \( \tilde{z} \) may be represented as follows.

\[
-\frac{\partial \tilde{p}}{\partial \tilde{z}} = A_0 + A_1 \cos (\omega_p \tilde{t}) \tag{5.2}
\]

where \( A_0 \) is steady component of the pressure gradient, \( A_1 \) is amplitude of the fluctuating component and \( \omega_p = 2 \pi f_p \), \( f_p \) is the pulse frequency. Both \( A_0 \) and \( A_1 \) are functions of \( \tilde{z} \), \( f \) being the heart pulse frequency.

Again the body acceleration \( G \) is given by

\[
G = a_0 \cos \left( \omega_b \tilde{t} + \phi \right), \quad t \geq 0 \tag{5.3}
\]

where \( a_0 \) is the amplitude of body acceleration, \( \omega_b = 2 \pi f_b \), \( f_b \) is the body acceleration frequency in Hz, \( \phi \) is the lead angle.

The boundary conditions are given by

\[
\tilde{w} = 0 \quad \text{at} \quad \tilde{r} = k a \quad \text{and} \quad \tilde{w} = 0 \quad \text{at} \quad \tilde{r} = a \tag{5.4}
\]
Let us introduce the following non-dimensional variables:

\[ w = \frac{\bar{w}}{w_0}, \quad r = \frac{\bar{r}}{a}, \quad t = \frac{\bar{t}}{w_p} \]

Using non-dimensional variables, eq (5.1) reduces to

\[ \alpha^2 \frac{\partial w}{\partial t} = p(t) + \frac{1}{r} \frac{\partial}{\partial r} \left( r \frac{\partial u}{\partial r} \right) \quad (5.5) \]

where \( \alpha^2 = \frac{\omega_p \rho R^2}{\mu} \), \( \alpha \) is called Womersley frequency parameter.

\[ p(t) = D_1 + D_2 \cos(t) + D_3 \cos(\omega t + \phi) \text{ and} \]

\[ D_1 = \frac{A_0 R^2}{\mu u_0}; \quad D_2 = \frac{A_1 R^2}{\mu u_0}; \quad D_3 = \frac{\rho A_0 R^2}{\mu u_0}; \quad \omega = \frac{\omega_b}{\omega_p} \]

The boundary conditions in non-dimensional form is

\[ w = 0 \text{ at } r = k \quad \text{and} \quad w = 0 \text{ at } r = 1 \quad (5.6) \]

### 5.3 METHOD OF SOLUTION

To solve the eq (5.5) associated with boundary conditions, we employ perturbation technique. Considering the Womersley parameter to be small, the velocity \( w \) can be expressed in the following form

\[ w(z,r,t) = w_0(z,r,t) + \alpha^2 w_1(z,r,t) + \ldots. \quad (5.7) \]
Substituting the expression of \( w \) which is given in equation (5.7) in (5.5) and equating the constant term and terms of \( \alpha^2 \) we get

\[
\frac{\partial}{\partial r} \left( r \frac{\partial w_0}{\partial r} \right) = -r p(t) \tag{5.8}
\]

\[
\frac{\partial w_u}{\partial t} = \frac{1}{r} \frac{\partial}{\partial r} \left( r \frac{\partial w_1}{\partial r} \right) \tag{5.9}
\]

The corresponding boundary conditions are

\[
w_0 = 0 \quad , \quad w_1 = 0 \quad \text{at} \quad r = k \quad \text{and} \tag{5.10a}
\]

\[
w_0 = 0 \quad , \quad w_1 = 0 \quad \text{at} \quad r = 1 \tag{5.10b}
\]

Integrating the equation (5.8) twice with respect to \( r \) and using the boundary condition (5.10a), we obtain the expression for \( w_0 \) as

\[
w_0 = \frac{p(t)}{4} \left[ 1 - r^2 - (1-k^2) \frac{\log(r)}{\log(k)} \right] \tag{5.11}
\]

Substituting eq (5.11) in (5.9), integrating twice with respect to \( r \) and using the boundary condition (5.10b), we get \( w_1 \) expression as

\[
w_1 = \frac{p(t)}{4} \left\{ \frac{r^2}{4} - \frac{r^4}{16} - \frac{1-k^2}{\log(k)} \left( \frac{r^2}{4} \log(r) - \frac{r^2}{4} \right) \right\} + C_1 \log(r) + C_2 \tag{5.12}
\]
where

\[ C_1 = \frac{p'(t)}{4} \left( \frac{(1-k^2)}{\log(k)} \left[ \frac{3}{16} \left( 1 - \frac{k^2}{2} \right) + \left( \frac{1-k^2}{4} \log(k) \right) \left( \frac{2}{3} - \frac{k^2}{4} \right) \right] \right) \]

\[ C_2 = -\frac{p'(t)}{4} \left( \frac{3}{16} \left( 1 - \frac{k^2}{2} \right) \right); \quad p'(t) = -D_2 \sin t - D_3 \sin (\omega t + \phi) \omega \]

The expression for velocity \( w \) can be obtained from equations (5.7), (5.11) and (5.12).

The non-dimensional flow rate is given by

\[ Q = Q_0 + \alpha^2 Q_1 \quad (5.13) \]

where \( Q_0 = 8 \int_k^1 w_0 r \, dr \)

\[ = p(t) \left[ \frac{1}{2} - k^2 \left( 1 - \frac{k^2}{2} \right) + \left( \frac{1-k^2}{4} \log(k) \right) \left( \frac{1}{2} - k^2 \left( \log k - \frac{1}{2} \right) \right) \right] \quad (5.14) \]

and

\[ Q_1 = 8 \int_k^1 w_1 r \, dr \]

\[ = \frac{p'(t)}{4} \left\{ \frac{5}{12} - \left( \frac{k^2}{4} - \frac{k^4}{16} \right) + \left( \frac{1-k^2}{2} \log(k) \right) \left( \frac{5}{4} + k^4 \left( \log(k) - \frac{5}{4} \right) \right) \right\} \]

\[ - 4 C_1 \left[ k^2 \left( \log(k) - \frac{1}{2} \right) + \frac{1}{2} \right] + 4 C_2 (1 - k^2) \quad (5.15) \]
The wall shear stress is given by

$$\tau_w = \frac{\partial w_0}{\partial r} + \alpha^2 \frac{\partial w_i}{\partial r} \quad \text{at} \quad r = 1 \quad (5.16)$$

By substituting velocity expressions (5.11) and (5.12) in (5.16) we get

$$\tau_w = \frac{p(t)}{2} \left[ -1 + \frac{(k^2 - 1)}{2 \log(k)} \right] + \alpha^2 \frac{p'(t)}{4} \left[ \frac{1}{4} + \frac{(1-k^2)}{4 \log(k)} \right] + C_1$$

The frictional resistance per unit length ($F_r$) of the artery can be defined as

$$F_r = \frac{dp/dz}{Q} \quad (5.17)$$

where $dp/dz$ is the pressure gradient and $Q$ is the flow rate.

**5.4 RESULTS AND DISCUSSION**

The objective of this analysis is to understand the change in flow pattern due to the insertion of a catheter in an artery, the rate of flow and frictional resistance, shear stress and to study the effect of body acceleration on the flow characteristics. Blood is modelled as a Newtonian fluid. The results are discussed in large and small arteries viz aorta, femoral, carotid and coronary arteries. The relevant data is presented in table 4.1 in chapter 4.

Fig 5.2 (a-d) depicts the axial velocity distribution for different values of the amplitude of the body acceleration when $k = 0.5$, $\alpha = 0.1$and $t = 0.5$. The presence of body acceleration increases the velocity in all the arteries. Increase
in the amplitude of the body acceleration further increases the velocity. It is observed that the effect of body acceleration is significant in large arteries. In aorta the effect of body acceleration is felt throughout the cross-section of the artery. When the amplitude of the body acceleration is 0.3 the peak value of velocity is found to be more than twice of that in the absence of body acceleration. In femoral artery the effect of body acceleration is negligible in the vicinity of the boundaries. The peak value of velocity in femoral artery increases from 0.1605 to 0.1839 when the amplitude of body acceleration changes from 0.1 to 0.3. In carotid artery the effect of body acceleration is seen in the mid region of the annulus and it is not significant as in the case of aorta and femoral. In coronary artery the effect of body acceleration is very negligible.

Fig 5.3 (a-d) describes the velocity variation for different sizes of the catheters when \( t = 0.1, \alpha = 0.5 \) and \( a_0 = 0.1 \). The values of the radius of catheter in the range 0.3 – 0.6 are widely considered in coronary angioplasty procedure. As the size of the catheter increases the velocity decreases. When the size of the catheter is 0.3, the peak velocity is reduced by two times of the value corresponding to the case when \( k = 0.1 \). When \( k = 0.5 \) the reduction factor in the peak velocity is 4. A similar trend is noticed in the other arteries also.

In Fig 5.4 (a-d) the variation of velocity in one time cycle is shown. It is noticed that the velocity in aorta reduces throughout the cross section of the
annular region. The velocity decreases with increase in t and it is zero throughout the cross-section at $t = 90^0$ and it becomes negative at $t = 135^0$ and $180^0$ indicating a back flow. In femoral artery the velocity reduces with time upto $t = 135^0$ and then increases in the remaining time cycle. No back flow is noticed. In carotid a similar trend as that of femoral artery is noticed assuming almost similar values for $t = 135^0$ and $180^0$. In femoral and carotid arteries the variation of velocity with respect to time is not much in the vicinity of the boundaries of the vessel. In coronary the effect of time on the variation of velocity is felt away from the boundaries.

In the absence of body acceleration the flow rate in aorta decreases with time in the first half cycle assuming a minimum value at $t = 180^0$ and then increases in the second half of the time cycle. However, the change in flow rate is not very significant. In the presence of body acceleration the flow rate becomes harmonic. The flow rate increases with increase in the amplitude of the body acceleration. In the femoral artery, in the absence of body acceleration the behaviour is similar to that of the aorta. In the presence of body acceleration it is oscillatory. An exactly similar trend is noticed in carotid artery also. But in the coronary artery (Fig 5.5d) the influence of body acceleration is not appreciable.

Fig 5.6 (a-d) describes the variation of frictional resistance with catheter size. It is noticed that the frictional resistance increases with increase in the size
of the catheter in all the arteries. It is observed that the presence of the body acceleration reduces frictional resistance considerably in aorta. In femoral and carotid arteries though the frictional resistance reduces with increase in the amplitude of the body acceleration, it is not as significant as in aorta. In coronary artery there is no impact of body acceleration on the frictional resistance.

Table 5.1 shows the values of frictional resistance in aorta, femoral and carotid arteries. The range from 0.3 to 0.6 for the size of the catheter is significant in coronary angioplasty procedures (Back, 1994). Clinical investigations revealed that the frictional resistance is found to increase with increase in the catheter size.

The presence of body acceleration is found to reduce frictional resistance from 29.161 to 10.758 when $k = 0.6$ in aorta, when $a_0 = 0.49$. When $a_0 = 0.98$ this value is reduced by five times of that case when $a_0 = 0$. In femoral (carotid) the reduction factor is in the range 3.645 to 16.379 (4.340-19.448) when $k$ is in the range from 0.3 to 0.6. Therefore, it is suggestible to give external accelerations by properly timing with respect to heart beat (Arntzenius et al. 1970) to the patients undergoing clinical procedures which involve catheter insertions with in the required limits so as to reduce the increased resistance due to the insertion of a catheter. In coronary artery there is a negligible reduction in this value when $a_0 = 0.98$. 

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Fig 5.7 (a-d) describes the variation of shear stress versus time. In the absence of body acceleration the wall shear stress is negative and increases in the first half cycle and then reduces in the other half cycle with its maximum at \( t = 180^0 \). The presence of body acceleration increases the wall shear stress and further increases with the amplitude of body acceleration. This is due to the increased flow rate in the presence of body acceleration. The behaviour of wall shear stress versus time is reversal to that of the flow rate. In all the other arteries the same behaviour is noticed.

5.5 CONCLUSIONS

The present mathematical model reveals some salient features in the flow pattern, flow rate, frictional resistance to flow and wall shear stress due to the presence of a catheter subjected to periodic body acceleration. Blood is modelled as a Newtonian fluid. The velocity and flow rate are enhanced in the presence of body acceleration and further increased with increase in amplitude of body acceleration in the aorta, femoral and carotid arteries. The effect of body acceleration in the coronary artery is very meager. Depending on the size of the catheter in the range 0.3 to 0.6 (widely used in coronary angioplasty procedures) the frictional resistance in large vessels increases by a factor 6.576 – 29.161. However, in aorta in the presence of body acceleration the frictional resistance is found to be reduced considerably in the range 2.381 – 10.758 for the same range of values of catheters when \( a_0 = 0.49 \) in aorta. In
smaller diameter vessels carotid, the frictional resistance is decreased in the range 5.228 – 23.334 for the variation of the size of the catheter. Hence, body acceleration can be used to reduce the frictional resistance that arise due to the insertion of a catheter.
Table 5.1 Frictional resistance

<table>
<thead>
<tr>
<th>k</th>
<th>Aorta B = 0</th>
<th>Aorta B = 0.49</th>
<th>Aorta B = 0.98</th>
<th>Femoral B = 0</th>
<th>Femoral B = 0.49</th>
<th>Femoral B = 0.98</th>
<th>Carotid B = 0</th>
<th>Carotid B = 0.49</th>
<th>Carotid B = 0.98</th>
<th>Coronary B = 0</th>
<th>Coronary B = 0.49</th>
<th>Coronary B = 0.98</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.5</td>
<td>15.866</td>
<td>5.826</td>
<td>3.568</td>
<td>15.866</td>
<td>11.389</td>
<td>8.882</td>
<td>5.866</td>
<td>12.677</td>
<td>10.555</td>
<td>5.866</td>
<td>15.586</td>
<td>15.315</td>
</tr>
</tbody>
</table>
Fig. 5.2 (a,b) Variation of axial velocity for different values of body acceleration $a_0$

when $k = 0.5$, $\alpha = 0.1$, $t = 0.5$ in (a) aorta (b) femoral
Fig. 5.2 (c,d) Variation of axial velocity for different values of body acceleration $a_0$
when $k = 0.5$, $\alpha=0.1$, $t=0.5$ in (c) carotid (d) coronary
Fig. 5.3 (a,b) Variation of axial velocity for different sizes of the catheters when $a_0 = 0.1$, $\alpha = 0.5$, $t = 0.1$ in (a) aorta (b) femoral
Fig. 5.3 (c, d) Variation of axial velocity for different sizes of the catheters when

\( a_0 = 0.1, \alpha = 0.5, t = 0.1 \) in (c) carotid (d) coronary
Fig. 5.4 (a, b) Variation of axial velocity for different values of time when $k=0.1$, $a_0 = 0.1$, $\alpha=0.5$ in (a) aorta (b) femoral
Fig. 5.4 (c, d) Variation of axial velocity for different values of time when $k=0.1$, $a_0 = 0.1$, $\alpha=0.5$ in (c) carotid (d) coronary
Fig. 5.5(a, b) Variation of flow rate with time for different values of body acceleration $a_0$ when $k=0.5$, $\alpha = 0.5$ in (a) aorta (b) femoral
Fig. 5.5 (c, d) Variation of flow rate with time for different values of body acceleration $a_0$ when $k = 0.5$, $\alpha = 0.5$ in (c) carotid (d) coronary
Fig. 5.6 (a, b) Variation of frictional resistance with catheter radius $k$ for different values of body acceleration when $t = 0.5$, $\alpha = 0.5$ in (a) aorta (b) femoral
Fig. 5.6 (c, d) Variation of frictional resistance with catheter radius $k$ for different values of body acceleration when $t = 0.5$, $\alpha = 0.5$ in (c) carotid (d) coronary
Fig. 5.7 (a, b) Variation of wall shear stress with time for different values of body acceleration when \( k = 0.5, \alpha =0.5, t=0.1 \)in (a) aorta (b) femoral
Fig. 5.7 (c, d) Variation of wall shear stress with time for different values of body acceleration when \( k = 0.5, \alpha = 0.5, t = 0.1 \) in (c) carotid (d) coronary.
Fig 5.1 Schematic diagram of catheterized artery