Abstract

To evaluate the local hemodynamic effects of coronary artery balloon angioplasty, computational fluid dynamics was applied to representative stenoses geometry post-angioplasty (minimal lesion diameter \(d_m = 1.8\) mm which produced 64% mean area stenoses) based on a group of patients and measured values of coronary flow reserve (CFR) returning to a normal range (3.6 ± 0.3). The computations were at mean flow rates (\(\bar{Q}\)) of 50, 100, 150 and 170 ml/min. The study indicates changes in the hemodynamic conditions due to insertion of a guidewire, which can be used to determine the mean pressure drop (\(\Delta p\)) and fall in distal mean coronary pressure (\(\bar{p}_r\)), and thus give quantitative estimate of uncertainty expected in diagnosis of moderate lesions. The guidewire to minimal lesion diameter ratio is 0.26, causing tighter “artifactual” mean area stenoses of 65.5%. During hyperemia, \(\bar{p}_m\) dropped to 72 mmHg as compared to 75 mmHg under patho-physiological condition without guidewire. \(\bar{Q}_h\) (subscript h: hyperemia) decreased from 180 without guidewire to 170 ml/min with the guidewire present. Thus, there was a significant ~ 43% increase in \(\Delta p_h\) and a ~ 51% increase in the hyperemic flow resistance (\(\bar{R}_h = \Delta p_h/\bar{Q}_h\)) over the patho-physiological condition. This could cause an overestimation of the severity of the moderate stenoses. Transient and steady flow guidewire surface shear stress was 35–50% higher than corresponding values for arterial wall shear stress. The non-dimensional data given in tabular form may be useful in interpretation of clinical guidewire measurements for moderate lesions of similar geometry and size.

Keywords: Hemodynamics; Coronary stenoses; Mean pressure drop; Guidewire

1. Introduction

The estimation of stenosis severity from measured pressure gradients (e.g. DeBruyne et al., 1993; DiMario et al., 1993; Emanuelsson et al., 1993) and velocity (e.g. Cole and Hartley, 1977; Doucette et al., 1992; Kern et al., 1994; Segal, 1992) using a guidewire has been enhanced with the use of small diameter guidewires. Presently, a single guidewire is essential (DeBruyne et al., 2001; Pijls et al., 2002; Fearon et al., 2003; Siebes et al., 2004) for simultaneous evaluation of epicardial and microvascular disease.

However, the introduction of a guidewire causes an obstructive effect and creates an additional “artifactual” stenosis (e.g. Segal et al., 1992; Banerjee et al., 2003a, b). This is especially important for the diagnosis of moderate stenoses as measured pressure drops across them are relatively small and a slight change in the measured values will produce some uncertainty in assessing their severity. Glagov et al. (1987) have indicated that it may be difficult to estimate the flow blockage effect of coronary artery lesions till they occupy about 40% of the internal elastic...
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luminal area. Wilson and Laxson (1993) note that the specific effects of changes in hemodynamic conditions on $\Delta \tilde{p}$ measurements are not well described in the human coronary circulation. Additionally, an absolute measurement of $\tilde{Q}$ and coronary flow reserve (CFR) with guidewire is complicated by uncertainties such as lumen curvature or nearby branches, reverse flow, sample volume, etc. in velocity distributions.

Detailed analysis of the patho-physiological condition before and after Coronary Angioplasty (Banerjee et al., 2000, 2003a, b) drew important correlations with several in vitro and computational studies of stenotic models (e.g. Cho et al., 1983; Deshpande et al., 1976; Mates et al., 1978, Youند and Tasi, 1973) with respect to important physiological aspects of blood flow in stenosed arteries namely, $\Delta \tilde{p}$, wall shear stress and pressure drop coefficients. The purpose of this investigation was to evaluate current clinical measurements in a moderately stenosed coronary artery by determining the following translational hemodynamics with a guidewire present: (1) velocity profiles and pressure distributions; (2) wall shear stress distributions; (3) shear stress distributions at guidewire surface; (4) phasic and mean pressure drops to obtain the $\Delta \tilde{p} - \tilde{Q}$ relation; and (5) amount of shift in the $\Delta \tilde{p} - \tilde{Q}$ relation relative to patho-physiological flow.

In this study, the effect of the guidewire on the hemodynamics of flow will be brought out in comparison to the patho-physiological condition after angioplasty without the guidewire (Banerjee et al., 2000). The ratio of guidewire size ($d_i = 0.46 \text{ mm}$) to minimal lesion diameter ($d_m = 1.8 \text{ mm}$) was 0.26. With the guidewire spanning the residual lesion, the tighter “artificial” mean area stenoses was 65.5% compared to 64% mean area stenoses from the patient angiograms. $Re_c$ varied from 90 to 300 (based on hydraulic diameter, $d_H = d_o - d_i$) and mean flow rate, $\tilde{Q}$, from 50 to 170 ml/min; the Womersley number was 1.90.

2. Methods

The in vivo data set of Wilson et al. (1988) in a 32 patient group undergoing percutaneous transluminal balloon coronary angioplasty (PTCA) was used. Additional dimensional data on the lesion shape were used from a similar stenosis described by Back and Denton (1992) and further described in Banerjee et al. (2000). CFR increased from 2.3 ± 0.1 to 3.6 ± 0.3 following angioplasty; mean aortic pressure ($\tilde{p}_a$), measured at the entrance of the left main coronary artery, decreased from 89 ± 3 to 84 ± 3 mmHg (Wilson et al., 1988). Since in the Wilson et al. (1988) measurements the Doppler crystal was embedded on the side of the catheter proximal to the tip, the sample volume was in the gap between the proximal vessel and catheter wall, a more stable flow region (e.g. Fig. 3 at $z = 0.0 \text{ cm}$ for hyperemic flow with guidewire).

In this analysis, the guidewire was presumed to lie concentrically in the residual lesion (Fig. 1). The concentric configuration of the guidewire within the lesion may give the largest pressure drop (Back et al., 1996; Back, 1994). The coronary flow waveform used in this analysis was obtained from in vitro calibration (Cho et al., 1983) and smoothing of the fluctuating Doppler signal (Sibley et al., 1986), and without any phase shifting for the proximal LAD (Segal, 1992). The ratio of peak systolic to peak diastolic velocity was 0.4. In Fig. 2, the peak diastolic velocity $\tilde{u}_{t-1}$ corresponds to a normalized velocity of 1.0, so that the ratio of mean to peak velocity $\tilde{u}/\tilde{u}_{t-1}$ is 0.537 as shown by the dashed line.
In the flow analysis, the residual composite lesion was assumed to have a smooth, rigid plaque wall and round concentric shape with mean proximal diameter $d_o$ (Fig. 1). Plaque geometry was presumed to remain rigid and the same for hyperemic conditions (e.g., Glagov et al., 1987; Vita et al., 1989). The continuity and momentum equations were solved by Galerkin finite element method (Baker, 1983; FIDAP Manual, 2003). The Carreau model was used for shear rate dependent non-Newtonian viscosity of blood (Cho and Kensey, 1991). Further details of computational method can be found in Banerjee et al. (1999, 2000, 2003a, b). Our computational modeling methods have been previously validated by extensive comparison with published experimental data (e.g., Cho et al., 1983; Yound and Tasi, 1973), and with the same moderate stenosis without guide wire (Banerjee et al., 2000). In the Reynolds number, a kinematic viscosity of $0.035 \text{cm}^2/\text{s}$ was used, a value near the asymptote in the Carreau model for blood (Cho and Kensey, 1991).

In the proximal vessel, the spatial velocity profile in the annular gap was initially taken to be the analogous Poiseuille flow relation for the axial velocity $u$:

$$u/2\bar{u} = [(1 - (r/r_o)^2) \ln(r_o/r_i) + (1 - (r_i/r_o)^2) \ln(r/r_o)] / [(1 + (r_i/r_o)^2) \ln(r_o/r_i) - (1 - (r_i/r_o)^2)]$$  \hspace{1cm} (1)

Zero velocity was specified on the guidewire and plaque wall. A stress free boundary condition was specified at the outlet. Adequate distal length was ensured for accurate determination of pressure drops due to the lesion and for the convergence of the calculations. The calculations were done at $\bar{Q}$ of 50 ml/min (typical of basal physiological values in a coronary vessel of 3 mm size (Back et al., 1977)), 100, 150 and hyperemic $\bar{Q}$ of 170 ml/min. Numerical data is reported for the second cycle for $\bar{Q} = 50$; 100 and 150 ml/min, and for the fifth cycle for $\bar{Q} = 170$ ml/min to fully capture the phasic variations in pressure drops. Heart rate was 75 beats/min and blood density was 1.05 g/cm$^3$. Simpler steady flow computations at the mean flow rates were also made for comparison with the more difficult pulsatile flow computations.

3. Results

The origin $(r = 0, z = 0)$ is 0.3 cm proximal to the lesion (Fig. 1); $z = 1.05$ cm is the mid-point of the throat segment; $z = 1.5$ and 4.5 cm are distal to the lesion. The pulsatile data are denoted by time instants I, II, III and IV in the coronary flow waveform (Fig. 2), where values of $u/\bar{u}_{p-1}$ are 0.50, 0.99, 0.75 and 0.25, respectively.
chosen to give about equal incremental changes in normalized velocity. Point I ($t = 1.05, 3.45$ s) is near the beginning of diastole. Point II is nearly peak diastolic flow ($t = 1.2, 3.6$ s); point III ($t = 1.38, 3.78$ s) and point IV ($t = 1.51, 3.91$ s) are during the deceleration phase in diastole. Although the steady state calculations do not include the unsteady effects of blood flow during the cardiac cycle, they nevertheless show characteristics similar to pulsatile variables in some regions of the flow field.

3.1. Velocity profiles along the lesion

The velocity profiles, during the acceleration and deceleration phases of the cardiac cycle and along the length of the lesion, were similar to patho-physiological condition (Banerjee et al., 2000), and with increasing $\bar{Q}$ (Figs. 3 and 4). The velocity profiles have zero axial velocity at $r = 0.023$ cm. Proximal to the lesion, velocity profiles were skewed towards the guidewire (Back et al., 1996), and thus produce a higher shear stress ($\tau_i$) on the guidewire surface than on proximal vessel wall ($\tau_w$) (Fig. 5). The corresponding peak systolic flow rates ranged from $Q_{p,s} = 93.1$ to 316.6 ml/min and peak Reynolds numbers from $Re_{ep} = 163$ to 555 as $\bar{Q}$ increased from 50 to 170 ml/min (Figs. 3 and 4). Distal to the throat, flow separation and reattachment occurs along the lesion wall, indicated by negative axial velocities and $\tau_w < 0$ near the lesion wall. This flow separation in the distal vessel could play a significant role in underestimation of true flow reserve in moderate stenoses (Porenta et al., 1999).

3.2. Arterial wall and guidewire shear stress distributions (reference steady and pulsatile flow)

In Figs. 5 and 6, steady and pulsatile $\tau_w$ distributions for different flow rates were similar to $\tau_w$ distributions in patho-physiologic flow (Banerjee et al., 2000). During the cardiac cycle, $\tau_w$ and $\tau_i$ varied considerably at times I, II, III and IV even though the basic nature of the flow separation zone varied little during the cardiac cycle and

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Fig. 3. Velocity profiles at different locations along the stenosis at various times during the cardiac cycle for $\bar{Q} = 50$ ml/min.
with increasing $\dot{Q}$. Moreover, the pulsatile $\tau_w$ and $\tau_i$ appears to be principally quasi-steady.

Due to the skewness of the velocity profile (peak closer to the guidewire surface), $\tau_i$ in the proximal vessel is larger than $\tau_w$ along the arterial wall. Proximal to the divergent section, $\tau_i$ distributions at different flow rates were similar to $\tau_w$ distributions. However, $\tau_i > 0$ distal to the throat as there was no flow separation along the guidewire surface. Table 1 lists some $\tau_{wm}$ values for steady and pulsatile flow. The values of $\tau_{im}$ are higher ($\sim 35-50\%$) as compared to $\tau_{wm}$ due to a higher shear rate ($\dot{\omega}_{/r}$) at the guidewire surface (Table 1). Once again the quasi-steady nature of the flow field is evident from $\tau_i$ distributions for $Q$ and $\dot{Q}$.

3.3. Pressure distributions (reference steady and pulsatile flow)

The variation of the pressure distribution, $p - p_e$, along the arterial wall is shown in Fig. 5 as a reference datum for $Q = 50, 100, 150$ and $170$ ml/min. Pressure differences are with reference to $p_e$ upstream of the lesion inlet ($z = 0$). The general shape of pulsatile $p - p_e$ distribution (Fig. 7) along the stenosis was similar to patho-physiologic flow (Banerjee et al., 2000), and reference steady flow computations, though the magnitudes were higher.

Overall pressure drop $\Delta p = p_e - p_i$ increased appreciably with flow rate (Fig. 8(a) and Table 2). Values of $\Delta p_i$ increased from 1.75 to 9.12 mmHg for a 3.4 fold increase in $Q$. Like shear stress distributions, there were wide variations in magnitude of pulsatile $p - p_e$ distribution (Fig. 7) at time I, II, III and IV during the cardiac cycle. At time II, pressure drop to the minimal throat value $\Delta p_i$ increased from 4.9 mmHg at 50 ml/min to 33 mmHg at 170 ml/min. Applying Bernoulli equation at peak diastole for inviscid flow, $\Delta p_{i\infty}$ was 1.5 and 17 mmHg at flow rate $Q_{p-1}$ of 93.1 and 316.6 ml/min, respectively. Thus, contribution of wall and guidewire friction and more precise determination of net
momentum changes in the computations considerably increase $D_{p1}$ (Back et al., 1996, 1997).

Distal to the lesion, flow separation takes place and the flow requires some length to once again fully develop to non-Newtonian pulsatile blood (Fig. 7). This length varies nearly linearly with increasing $\bar{Q}(t)$, and increasing $\dot{Q}$. The flow recovery lengths at time II, measured from the throat exit, ranged from 0.79 cm at $\dot{Q} = 50$ ml/min to 2.63 cm at $\dot{Q} = 170$ ml/min. Thus, at hyperemic flow, the sensor element on the side of a pressure guidewire probably should be located $\approx 15 \times d_m$ in a straight, unbranched vessel distal to a moderate stenosis.

3.4. Phasic and mean flow pressure distributions

At $\dot{Q} = 50$, 100, 150 and 170 ml/min, the instantaneous peak diastolic pressure drops $\Delta p_s(= p_r - p_a)$ were about 3.9, 10.5, 19.5 and 23.5 mmHg, respectively (Fig. 8(a)). $\bar{p}$, obtained by integrating over the cardiac cycle, increased from 1.96 mmHg at basal to 10.6 mmHg at hyperemic flow (Table 2). Mean distal pressure ($\bar{p}_d$) is given by $\bar{p}_d = \bar{p}_a - \Delta \bar{p} - \Delta \bar{p}_c$, where $\bar{p}_a = 84$ mmHg (the measured coronary ostium value) and $\Delta \bar{p}_c$ is the estimated pressure drop in the larger vessels distal to the coronary ostium to just proximal to the stenosis due to wall friction. This drop is about $\Delta \bar{p}_{ch} = 1.3$ mmHg at $\dot{Q}_c = 170$ ml/min. This value $\Delta \bar{p}_{ch}$ for hyperemic flow is scaled to lower values of $\dot{Q}$ by $\Delta \bar{p}_c = \Delta \bar{p}_{ch}(\dot{Q} / \dot{Q}_c)$. In clinical use of pressure guidewires for hyperemic flow condition and for a discrete lesion, the assumption is often made that $\bar{p}_{rh} = \bar{p}_a - \Delta \bar{p}_h$, i.e. $\Delta \bar{p}_{ch} \approx 0$, where $\bar{p}_a$ = mean aortic pressure.

Table 2 gives the pulsatile flow results of the guidewire flow analysis after angioplasty. $\Delta \bar{p}(= \Delta \bar{p} / 0.5 \rho \bar{u}_w^2)$ ranged from 1.9 to 4; the mean pressure drops ($\Delta \bar{p}$) were roughly 20% higher than the reference steady flow values ($\Delta p_s$). $\bar{p}$ dropped from $\approx 82$ to $\approx 72$ mmHg over the mean flow rate range (Fig. 8(a)). Although the shape of $\Delta \bar{p}$ is similar to inlet velocity $\bar{u}$ (Fig. 2), there is some phase lag in the $\Delta \bar{p}(t) - \bar{u}(t)$ relation, similar to
patho-physiologic flow (Banerjee et al., 2000). At the beginning of the systolic phase i.e. \( u' \leq 0 \); the phase angle is \( /C0 \) and the phase shifted \( Dp \) is positive (rise in pressure). \( /C0 y p \) at the local systolic peak is about 4.5; but at peak diastolic flow, there is virtually no phase lag in the \( Dp(t)/C0 u(t) \) relation. Thus, the phase angle varied during the cardiac cycle, exhibiting hysteresis effects in the \( Dp(t)/C0 u(t) \) relation.

### 3.5. Pressure drop and pressure recovery

Fig. 8(b) shows magnitude of axial pressure drops \( \Delta p(t) \) as a function of \( Q(t) \) on a log–log basis for all \( Q \) at time instants (I, II, III and IV) during the cardiac cycle. The values of \( \Delta p \) (shown by open points), at the throat exit, lie on a nearly linear curve on a log–log basis, \( \Delta p \propto Q^n \), with \( n \simeq 1.51 \). The points

<table>
<thead>
<tr>
<th>( Q ) (ml/min)</th>
<th>( \frac{\tau_{wm}}{\tau_{si}} )</th>
<th>( \frac{\tau_{sm}}{\tau_{si}} )</th>
<th>( \tau_{wm} ) (dynes/cm²)</th>
<th>( \tau_{sm} ) (dynes/cm²)</th>
<th>( \frac{\tau_{wm}}{\tau_{sm}} )</th>
<th>( \frac{\tau_{wm}}{\tau_{si}} ) min</th>
<th>Reattachment length at time II, in cm from ( z = 0 )</th>
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<tr>
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</tr>
</tbody>
</table>

- steady flow.

Table 1

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Fig. 6. Arterial wall shear stress \( \tau_w \) and guidewire shear stress \( \tau_i \) along the stenosis and distal vessel for pulsatile flow \( \hat{Q} = 50 \) and 170 ml/min.
corresponding to reference values ($\Delta p_{\infty}$) for steady flow ($Q = 50, 100, 150$ and $170$ ml/min) fall on the same curve as the pulsatile computations.

The values of $\Delta p_{\infty}$, $\Delta p_1$, and $\Delta p$ can prove useful in defining pressure drop and recovery coefficients (Back et al., 1996; Banerjee et al., 2003a, b). In the asymptotic limit ($Re \to \infty$) for the lesions with a guidewire $\kappa = (d_1^2 - d_1^2)/(d_2^2 - d_2^2) = 0.345$; we get $c_{pr_{\infty}} = 0.452$, $c_{A\Delta p_{\infty}} = 0.881$, $c_{A\Delta p_1} = 0.430$. These coefficients are summarized in Table 3 at peak diastolic flow for all $Q$. Pressure drop coefficient, $c_{Ap}(=\Delta p/0.5\rho\bar{u}_0^2 = c_{Ap_1} - c_{pr})$, generally decreased with increasing $Re$ across stenoses (and distal vessel) since the variation of $\Delta p$ with $Q$ is less than $\bar{Q}^2$, the asymptotic high $Re$ limit. The pressure drop to the throat ($c_{Ap_1}$) ranged from 3.33 to 1.97, which is higher than the Bernoulli value ($c_{Ap_{\infty}}$), and the pressure recovery ($c_{pr}$) increased from 0.21 to 0.31 relative to $c_{Ap_1}$ (Table 3). Pressure recovery factor ($k = c_{pr}/c_{pr_{\infty}}$) decreased with increasing $\bar{Q}$ similar to patho-physiological condition (Banerjee et al., 2000). $c_{Ap}$ ranged from 2.3 to 1.2, implying that the pressure head loss ($\Delta p/\rho$) was of the order of one to two throat velocity heads ($\bar{u}_0^2/2$) (Table 3). The values of $k(=c_{pr}/c_{pr_{\infty}})$ for $Q = 50, 100, 150$ and $170$ ml/min are 1.66, 1.56, 1.52 and 1.58. $k$ for steady flow rate $Q = 50, 100, 150, 170$ ml/min are 1.52, 1.36, 1.27 and 1.25, respectively. The throat dynamic pressure (Table 3) was relatively small at peak diastole for $Q = 170$ ml/min, being 20 mmHg as compared to 46 mmHg at $Q = 60$ ml/min in a significant coronary artery stenosis (Banerjee et al., 2003a, b).

3.6. Increase in the mean pressure drop with the presence of the guidewire

In Fig. 9(a), $\Delta \bar{p}_h$ increased from $\sim 7.4$ mmHg at $\dot{Q}_h = 180$ ml/min for patho-physiological flow (Banerjee et al., 2000) to 10.58 mmHg at $\dot{Q}_h = 170$ ml/min in the guidewire flow analysis. An estimate for hyperemic flow rate $Q_h$, and mean flow resistance $\dot{R}_h$ with and without guidewire was made as follows (Banerjee et al., 2003a,b):

$$\dot{Q}_h = \frac{\dot{P}_h - \dot{P}_\infty}{\dot{R}_h + \dot{R}_v} = \frac{\dot{P}_{h0} - \dot{P}_{\infty}}{\dot{R}_v}$$

and

$$\dot{R}_h = \Delta \bar{p}_h/\dot{Q}_h.$$  (2)

The minimal distal bed hyperemic mean flow resistance ($\dot{R}_v$) was about 0.30 mmHg/ml/min for the Wilson et al. (1988) patient group. With and without (Banerjee et al., 2000) guidewire, $\dot{R}_v$ was 0.062 and 0.041 mmHg/ml/min,
respectively. This ~ 51% increase in $R_h$ produced nearly ~ 43% increase in $\Delta P_h$, and this would lead to an overestimation of the hemodynamic severity of the lesion. Both with and without the guidewire, $R_h$ increased with $Q$ due to the non-linear $\Delta P - Q$ relation for the residual lesions and the tighter “artifactual” stenoses with the guidewire spanning the lesions. Meuwissen et al. (2002) noted that the measurement of

\begin{table}[h]
\centering
\begin{tabular}{|c|c|c|c|c|c|c|c|c|c|c|}
\hline
\multirow{2}{*}{$Q$ (ml/min)} & \multirow{2}{*}{$Re_q$} & \multirow{2}{*}{$Re_m$} & \multirow{2}{*}{dynamic} & \multirow{2}{*}{wall shear} & \multirow{2}{*}{mean} & \multirow{2}{*}{coronary} & \multirow{2}{*}{distal} & \multirow{2}{*}{$\frac{\Delta P}{\Delta P}$} & \multirow{2}{*}{Stenosis} \\
 & & & pressure & stress & pressure & drop & drop & resistance & \\
\hline
50 & 88 & 134.1 & 0.483 & 96 & 1.96 & 1.75 & 81.7 & 4.06 & 1.12 & 0.0392 \\
100 & 175 & 268 & 1.932 & 199 & 5.05 & 4.27 & 78.1 & 2.61 & 1.18 & 0.0505 \\
150 & 263 & 402 & 4.347 & 312 & 9.04 & 7.59 & 73.8 & 2.08 & 1.19 & 0.0603 \\
170 & 298 & 456 & 5.583 & 361 & 10.58 & 9.12 & 72.1 & 1.89 & 1.16 & 0.0622 \\
\hline
\end{tabular}
\caption{Results of guidewire flow analysis after PTCA}
\end{table}

Fig. 8. (a) Overall pressure drop $\Delta P$ across the stenoses and distal vessel during the cardiac cycle for $Q = 50, 100, 150$ and $170$ ml/min. (b) Variation of magnitude of axial pressure drops $\Delta P_1(t)$ and $\Delta P(t)$ with flow rate $Q(t)$ over the mean flow rate range ($Q = 50, 100, 150$ and $170$ ml/min) at time points (I, II, III, IV) during the cardiac cycle.
stenosis resistance could be useful in the diagnosis of moderate lesions through combined velocity and pressure measurements in stenosed arteries.

For post-PTCA without guidewire, \( c_{\Delta p} \) (\( = \Delta p/\rho \bar{u}^2 \)) varies from 19.6 to 10.4 as compared to 34.2 to 16 for post-PTCA with guidewire at comparable values of \( \bar{R}_{e} \) (Fig. 9 (b)). Likewise for Poiseuille flow with and without guidewire in a straight tube without stenoses (Back et al., 1996), \( c_{\Delta p} \) varies from 13.7 to 4 and 7.1 to 2, respectively at similar values of \( \bar{R}_{e} \) (Fig. 9(b)). These values are indicative of an additive effect of stenotic and guidewire resistance (\( 10.4 + 4 \approx 16 \)) on computed pressure drop.

On the linear maximal vasodilation-distal perfusion pressure curve (CFR-\( \tilde{\bar{p}}_{rh} \)) curve (Banerjee et al., 2003a, b), \( \tilde{\bar{p}}_{rh} \) and CFR for the physiological flow after PTCA with the guidewire was located (Fig. 10). Extrapolation of the nearly linear CFR- \( \tilde{\bar{p}}_{rh} \) curve towards its origin gave a \( \tilde{\bar{p}}_{ro} \) of \( \sim 20 \text{ mmHg} \). This value of \( \tilde{\bar{p}}_{ro} \) is close to a value of \( \sim 18 \text{ mmHg} \) known to cause subendocardium ischemia in dog hearts (Bache and Schwartz, 1982). \( \tilde{\bar{p}}_{ro} \) of \( \sim 20 \text{ mmHg} \) should be reasonable as Wilson et al. (1988) did not include patients with prior myocardial infarction (MI) or microvascular disease, who could have a higher \( \tilde{\bar{p}}_{ro} \) (Tanaka et al., 2003).

### 4. Discussion

Combined flow and pressure measurements have become necessary to distinguish the hemodynamic severity of the epicardial lesions from any prevalent microvascular disease in patients. Fearon et al. (2003) have shown discrepancies are associated with CFR measurements using guidewire, due to measurements being made distal to the lesion where flow separation occurs. Steady flow pressure drops were about 10–20%

<table>
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<th>( \dot{Q} ) (ml/min)</th>
<th>( Q_p ) (ml/min)</th>
<th>( Re_{ep} )</th>
<th>Peak throat ( c_{Ap} )</th>
<th>( c_{Ap1} )</th>
<th>( \bar{k} )</th>
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<th>( c_{\Delta p1} )</th>
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</table>

![Fig. 9. (a) \( \Delta \bar{p} \) vs. \( \dot{Q} \) for physiologic flow after angioplasty with and without guidewire. The fall of \( \bar{p} \) is shown on the right \( y \)-axis. (b) \( c_{Ap} \) vs. \( \bar{R}_{e} \) for physiologic flow after angioplasty with and without guidewire, and \( c_{Ap} \) vs. \( \bar{R}_{e} \) for Poiseuille flow in a straight tube with and without guidewire at comparable values of \( \bar{R}_{e} \).](image)
lower than mean pressure drop in a pulsatile flow scenario for the same mean flow rate. Therefore, steady flow pressure drops do not give an accurate estimate of physiological pressure drops and shear stresses (Table 2). The present study focuses on pressure measurement using guidewire. While the moderate stenosis itself produces a pressure drop, \( \sim 43\% \) increase in the \( \Delta \bar{p}_h \) and \( \sim 51\% \) increase in \( \bar{R}_h \) over the pathophysiological condition was observed under hyperemic conditions due to guidewire presence. The non-dimensional pressure data given in Tables 2 and 3 may be useful in diagnosis of moderate lesions of similar shape and size in clinical settings. For instance, the values of pressure recovery factor \( k \) show a decreasing trend with increasing flow rate for both the patho-physiologic condition (Banerjee et al., 2000) and the present study with guidewire presence. The earlier studies on significant coronary artery lesions (mean area stenoses \( \sim 90\% \)) with and without guidewire (Banerjee et al., 2003a,b) show an increasing trend for \( k \) with increasing flow rate. This similarity may prove useful in defining a “guidewire correction factor” using pressure recovery coefficients combined with flow resistances particularly for flow limiting hyperemic conditions, which would compensate for the obstructive effect of the guidewire.

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**References**


