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EFFECT OF ENDOVASCULAR DIAGNOSTIC CATHETERS ON HUMAN CORONARY ARTERY LESION FLOW COEFFICIENTS

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ABSTRACT

Lesion flow coefficients ($\tilde{c}$) were determined from computational hemodynamics for the 32 patient group of Wilson et al. (1988) during coronary angioplasty in conjunction with quantitative angiography and measurements of the coronary flow reserve (CFR). The effect of the catheter insertion across lesions reduce blood flow during measurements, and also decrease ($\tilde{c}$) due to larger viscous effects. The flow computations may be useful in interpretation of catheter measurements in the clinical setting for lesions of similar size, particularly for flow limiting hyperemic conditions.

INTRODUCTION

Measurements of coronary flow reserve (CFR) and translesional pressure drops, $\Delta\tilde{p}$, (referred to as pressure gradients in the medical literature) are often made in conjunction with quantitative angiography to assess the physiologic significance of lesions in patients with coronary artery syndromes (eg. Wilson and Laxon 1993). Efforts to directly measure pressure losses by catheters spanning lesions introduce a tighter 'artifactual' stenosis thus elevating the pressure drop and reducing blood flow during measurements. In this investigation, detailed pulsatile hemodynamic computations for physiologic flow in conjunction with measurements by Wilson et al. (1988) in a 32 patient group are used; along with simulations with catheters present in lesions of patients, to determine changes in flow coefficients in a similar manner as for fluid flow measuring devices.

METHODS

Dimensions and shape of the coronary stenoses (Table 1) before and after percutaneous transluminal coronary balloon angioplasty (PTCA) were obtained from biplanar angiograms (Wilson et al. 1988; Back and Denton 1992). In Table 1 d_e, d_m, d_r are proximal, throat, and distal diameters; and l_e, l_m, l_r are constriction, throat, and divergence lengths, respectively. Average percentage area stenosis changed from 90% to 64% with angioplasty. Measurements of CFR with a pulsed dopplar ultrasound catheter proximal to the lesions (with minimal blockage) increased from 2.3 to 3.6 in the procedure; mean arterial pressures $\tilde{p}_a$, measured in the coronary ostium, decreased. The patients had single-vessel, single-lesion coronary artery disease with angina. Patients with abnormalities that might affect the vasodilator capacity of the arteriolar vasculature were excluded from the study.

The spatial average coronary velocity waveform $\tilde{u}(t)$ used in the flow simulations was obtained in our laboratory from invitro calibration, smoothing the fluctuating Doppler signal, and phase shifting the normal pattern for the proximal LAD, consistent with Doppler measurements in patients where normal peak diastolic velocity is reduced by significant lesions.

Detail of the computational method is given by Banerjee et al. (1999). Pressure differences $\Delta p(t)$ between the stenosis inlet and distal region, including pressure recovery in the separated flow reattachment processes in the distal vessel, were integrated over the cardiac cycle to obtain the mean pressure drops, $\Delta\tilde{p}$. Distal mean coronary pressures $\tilde{p}_r = \tilde{p}_e - \Delta\tilde{p}$ were computed with $\tilde{p}_e \simeq \tilde{p}_a$ since mean flow resistances of proximal vessels were estimated to be small compared to the larger values across the stenoses. Computations were for a range of mean flow rates, $\tilde{Q}$, consistent with measurements of CFR and flow limitation for physiological flow; mean Reynolds numbers $\tilde{\mathrm{Re}}_e$ ranged from 100-230, and 100-360, respectively, before and after PTCA; the frequency parameter $\alpha_e =$ 2.25. The computations with catheters in the flow mentioned in the text were made at lower flow rates due to the elevated pressure drops ($\Delta\tilde{p}$) computed.

LESION FLOW COEFFICIENTS

The time mean pressure drop ($\Delta\tilde{p}$) normalized by the mean dynamic pressure in the throat region ($0.5\rho\tilde{\bar{u}}_m^{\,2}$) ie., the pressure drop

	A_m, mm^2	d_m, mm	% Area Stenosis	l_m, mm	CFR	$\tilde{P}_a$, mmHg	$\Delta\tilde{P}_h$, mmHg	$\tilde{P}_{rh}$, mmHg	$\tilde{c}_h$
Before PTCA	0.7 ± 0.1	0.95	90	0.75	2.3 ± 0.1	89 ± 3	~34	~55	0.83
After PTCA	2.5 ± 0.1	1.8	64	3	3.6 ± 0.3	84 ± 3	8	74 - 76	0.55

Table 1: Lesion dimensions, shape, and hemodynamics before and after PTCA. Values of d_e=d_r= 3 mm; l_c= 6 mm; l_r= 1.5 mm.

coefficient ($\tilde{c}_{\Delta p} = \Delta\tilde{p} / 0.5\rho\tilde{\bar{u}}_m^2$), was inverted to give the time mean $\tilde{Q} - \Delta\tilde{p}$ relation in a familiar form as:

$$\tilde{Q} = \tilde{c}\,[(A_m - A_i)/(1-\kappa)]\,(2\Delta\tilde{p}/\rho)^{0.5} \qquad (1)$$

where $\tilde{c} = (1-\kappa)/(\tilde{c}_{\Delta p})^{0.5}$ is the lesion flow coefficient computed from the flow simulations and $\kappa = (A_m - A_i)/(A_e - A_i)$, the throat to the proximal lesion flow cross-sectional area ratio in the presence of a catheter $(d_i; A_i)$ in the lesions. For physiological flow, $\kappa = (A_m / A_e)$ and $A_i = 0$ in eq. 1.

In the normalization procedure, the asymptotic behavior ($\tilde{c} \to 1$) in the high Reynolds number limit ie., $\mathrm{Re}_m \to \infty$ where wall friction becomes negligible, follows from the asymptote, $c_{\Delta p_\infty} \to (1-\kappa)^2$, which gives the net mean flow momentum change between the stenosis inlet and the throat exit ($c_{\Delta p_{1\infty}} = 1-\kappa^2$) from the Bernoulli equation, and for a sudden expansion in the divergent segment and distal vessel $(d_r \simeq d_e)$ where pressure recovery $[c_{p_\infty} = 2\kappa(1-\kappa)]$ occurs ie., $c_{\Delta p_\infty} = c_{\Delta p_{1\infty}} - c_{p_\infty}$. For coronary lesions, mean flow Reynolds numbers in the throat region $[\tilde{\mathrm{Re}}_m = (4\tilde{Q}/\pi\nu d_m)(1 + d_i/d_m)^{-1}]$ based on hydraulic diameter $(d_m - d_i)$ are relatively low and wall frictional effects are important. Consequently, values of $\tilde{c}_{\Delta p}$ lie above $c_{\Delta p_\infty}$, and values of $\tilde{c}$, conversely, lie below one.

RESULTS AND DISCUSSION

Values of $\tilde{c}$ are shown in Fig. 1 as a function of $\tilde{\mathrm{Re}}_m$ on a log-log plot before and after PTCA by solid curves for physiological flow, and by dashed curves (lower $\tilde{\mathrm{Re}}_m$) with catheters present in the lesions. For physiological flow after PTCA, the $\tilde{c}_{\Delta p} \propto \tilde{\mathrm{Re}}_m^{-n}$ relation can be approximated with the exponent $n \simeq 0.5$ for which $\Delta\tilde{p} \propto \tilde{Q}^{1.5}$; the $\tilde{c} \propto \mathrm{Re}_m^{m}$ relation had an exponent $m \simeq 1/4$, and an average value of $\tilde{c} \simeq 0.5$ over the range of CFR of 3.6. With the angioplasty catheter $(d_i = 1.4$ mm; $d_i/d_m = 0.78)$ in the flow after PTCA to measure pressure gradients the $\Delta\tilde{p} - \tilde{Q}$ relation was close to linear, so that $n \simeq 1$ and $m \simeq 0.5$. In this lower $\tilde{\mathrm{Re}}_m$ flow regime due to catheter induced flow blockage, momentum changes and pressure recovery were relatively small, and the flow was viscous dominated with an average value of $\tilde{c} \simeq 0.2$.

For physiological flow before PTCA, the flow regime was more controlled by momentum changes, and the average value of $\tilde{c} \simeq 0.75$ over the range of CFR of 2.3 was about 50% higher than after PTCA. With the guide wire catheter ($d_i = 0.46mm; d_i/d_m = 0.48$) in the flow, viscous effects were more important (average value $\tilde{c} \simeq 0.53$) nd significant flow blockage occurred. Since distal arteriolar reserve is exhausted in the subendocardium when pressure distal to the lesions ($\tilde{p}_r$) fell below ~ 55 mmHg (eg. Brown et al 1984), translesional pressure gradients, $\Delta\tilde{p}_h = \tilde{p}_e - \tilde{p}_r$ ~ 34 mmHg will become flow limiting in the absence or in the presence of catheters. For hyperemic maximal flow limitation, flow through the stenoses with the guide wire present ($\tilde{Q}_{gh}$) would be reduced below physiological values ($\tilde{Q}_h$) from eq. 1 by the factor (0.52) amounting to about 50% flow blockage, using the appropriate values of $\tilde{c}$ and flow cross-sectional areas. Moreover, by using eq. 1, hyperemic flow rates ($\tilde{Q}_h$) can be estimated directly for obstructive lesions, which in this case gives $\tilde{c}_h \simeq 0.83$ from Fig. 1 for the 90% area stenosis. The four sets of hemodynamic flow computations shown in Fig. 1 provide a basis for determining lesion flow coefficients. Estimates of volumetric flow rates $\tilde{Q}$ from eq. 1 require iteration since $\tilde{\mathrm{Re}}_m \propto \tilde{Q}$. These results may be useful in interpretation of catheter measurements in the clinical setting for lesions of similar size.

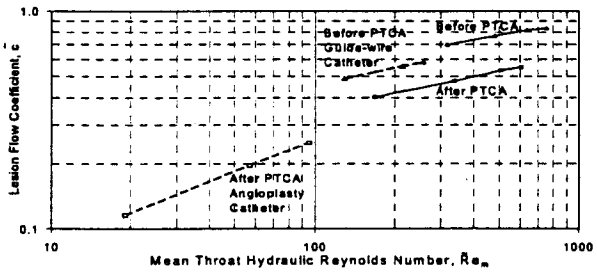

Figure 1: Lesion flow coefficient, $\tilde{c}$, vs. throat mean flow Reynolds number, $\tilde{\mathrm{Re}}_m$, based on hydraulic dia. $(d_m - d_i)$. Physiologic flow before & after PTCA (solid curves) & with catheters (dashed curves).

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