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PHASIC VARIATIONS AND MAGNITUDE OF PRESSURE RECOVERY DISTAL TO HUMAN CORONARY ARTERY STENOSES DURING ANGIOPLASTY

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ABSTRACT

Distal pressure recovery coefficients ($c_{pr} = kc_{p_{min}}$) were determined from pulsatile hemodynamic computations for the 32 patient group of Wilson et al. 1988 during coronary angioplasty in conjunction with quantitative angiography and measurements of coronary flow reserve (CFR). Before angioplasty, values of the factor $k(t)$ ranged 2-4 times high than a reference value, $c_{p_{min}}$=0.18, and varied during the flow acceleration and deceleration phases of the cardiac cycle. After angioplasty, values of $k(t)$ ranged from 0.8-1.4, roughly the same magnitude as $c_{p_{min}}$=0.46, and also varied phasically.

INTRODUCTION

This investigation is concerned with the complex process of pressure recovery in the divergent stenotic region and distal vessel of flow limiting human coronary lesions. The results of our detailed pulsatile hemodynamic computations in conjunction with previously reported clinical data in a patient group of Wilson et al. 1988 before and after coronary angioplasty, contribute to an improved understanding of flow regimes and flow separation and distal flow disturbances as observed in the patient group of Kajiya et al. 1987 before coronary bypass surgery. These flow disturbances are of the type observed earlier by Back and Rosche, 1972, and are associated with spatial separated shear-layer wave instabilities that depend primarily on the degree to which lesions narrow vessel lumens and Reynolds number. The phasic variations and magnitude of pressure recovery are reported for the patient group of Wilson et al. 1988 for which the disturbances led to the periodic formation of vortical flow cells along the distal vessel wall during the (phase shifted) systolic phase where Reynolds numbers were higher.

METHODS

Dimensions and shape of the coronary stenoses before and after percutaneous transluminal balloon coronary angioplasty PTCA were obtained from biplanar angiograms (Wilson et al. 1988; Back and Denton 1992). Balloon angioplasty both widened and lengthened the narrowest region of the stenoses (d_m = 0.95 to 1.8 mm, l_m = 0.75 to 3 mm). Despite axial redistribution of the plaque away from the narrowest region, overall stenosis shape and constriction (l_c = 6 mm) and divergence (l_r = 1.5 mm) lengths were roughly unchanged by balloon dilation. Average values of proximal and distal diameter were $d_e = d_i$ = 3 mm, and average percentage area stenosis changed from 90% to 64% with angioplasty. Measurements of CFR with a 3F pulsed doppler ultrasound catheter positioned proximal to the lesions (with minimal blockage) increased from 2.3 ± 0.1 to 3.6 ± 0.3 in the procedures; 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 $\overline{u}(t)$ used in the flow simulations was obtained in our laboratory from in-vitro 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.

A finite element method (FEM) was used to solve the unsteady Navier-Stokes equations for pulsatile non-Newtonian blood flow (Carreau model) so that the initiation, growth, and damping of flow fluctuations can be computed in principle. Both velocity and pressure were calculated simultaneously; calculations time steps varied between 1×10^{-4} to 1×10^{-5} s. Computations were for mean flow rates ($\tilde{Q}$) of 50 ml/min (typical of basal values in a coronary vessel of 3 mm size) and at 75 and 100 ml/min (the limit of our converged solutions) for the native lesions. The structure of the distal flow field was not known for the extrapolated hyperemic condition (CFR = 2.3) but was likely to be in an early stage of turbulent flow development during the peak systolic phase. Proximal mean flow Reynolds numbers ($\tilde{\mathrm{Re}}_e$) ranged from 100-200 and 100-300, respectively, before and after angioplasty;

he frequency parameter $\alpha_e = 2.25$. Steady flow calculations at the same mean flow rates were made for reference purposes.

Pressure Recovery Coefficients

In the usual way, pressure recovery is defined as $\Delta p_r = p_r - p_1 = \Delta p_1 - \Delta p$, where Δp_1 is the pressure drop to the throat exit, and Δp is the overall pressure drop across the stenoses and distal region. To place the pulsatile hemodynamic analysis in perspective, values of Δp_r are scaled on instantaneous the dynamic pressure in the throat region $(0.5\rho\overline{u}_m^2)$, to give a pressure recovery coefficient

$$c_{pr} = \Delta p_r / 0.5\rho\overline{u}_m^2 = kc_{pr\infty} \quad (1)$$

Here the reference datum value $(c_{pr\infty})$ is the high Reynolds number limit $(\mathrm{Re} \to \infty)$ across a sudden expansion of area ratio $(\kappa_r = A_m / A_r)$, ignoring wall friction $(\tau_w \to 0)$ and unsteady flow effects $[(\partial\overline{u}/\partial t) = 0]$

$$c_{pr\infty} = 2\kappa(1-\kappa); \quad \kappa_r = \kappa_e = \kappa = A_m / A_e \quad (2)$$

From conservation principles, flow momentum decreases distally and viscous dissipation (turbulence at high Re) occurs, increasing the internal energy of a fluid at the expense of pressure energy. Values for the lesion after angioplasty (κ = 0.360) $c_{pr\infty}$ = 0.461, and before angioplasty (κ =0.100) $c_{pr\infty}$ =0.180, a much lower value. These relations were used in an elementary way to determine the pressure recovery factor k in eq.1 from the flow computations.

RESULTS AND DISCUSSION

The pressure recovery factor $k(t)$ is shown in Fig.1 as a function of the proximal Reynolds number $\mathrm{Re}_e(t)$ on a log-log plot before and after PTCA at various times in the coronary flow waveform for which values of the normalized velocity $\overline{u}/\overline{u}_{p-i}$ are given in the table in Fig.1. Values of k_s for the reference steady flow calculations (cross points), decrease with an increase in Re_e as noted by Banerjee et al. [3] after PTCA, with an average value of $k_s \simeq 1.2$. This trend is also observed before PTCA for the reference steady flow calculations, but the average value of k_s is larger being about 2.0. The pulsatile values of $k(t)$ vary at different times in the cardiac cycle. After PTCA, values of $k(t)$ near peak diastolic flow ($\overline{u}/\overline{u}_{p-i}$ = 0.99) lie below k_s, as also is the case for acceleration phase ($\overline{u}/\overline{u}_{p-i}$ = 0.35). However, during the deceleration phase ($\overline{u}/\overline{u}_{p-i}$ = 0.70) values of $k(t)$ lie above k_s after PTCA. The pulsatile values of $k(t)$ before PTCA were much higher than after PTCA. For peak (phase shifted) systolic flow ($\overline{u}/\overline{u}_{p-i}$ = 1.0) average values of $k \simeq 3.3$ before PTCA, presumably due to the formation of vortical flow cells along the distal vessel wall, which enhance pressure recovery. At other times in the cardiac cycle values of $k(t)$ were also relatively large before PTCA as seen in Fig.1, being generally higher than k_s, but varied during the flow acceleration ($\overline{u}/\overline{u}_{p-i}$ = 0.50) and deceleration ($\overline{u}/\overline{u}_{p-i}$ = 0.25 and 0.75) phases which tend to decrease and increase Δp_r, respectively.

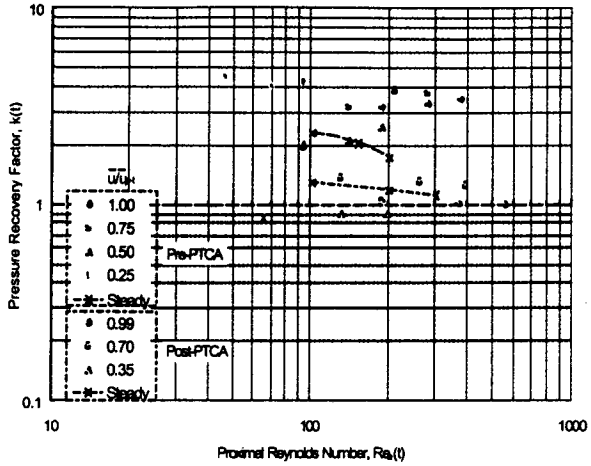

Figure 1. Variation of the pressure recovery factor k(t) with proximal Reynolds number $\mathrm{Re}_e(t)$ before and after PTCA.

The results shown in Fig.1 indicate the complex distal pulsatile hemodynamics with significant stenotic lumen reduction. Remodeling of the stenoses by angioplasty leads to more orderly pulsatile flow patterns, absent of flow instabilities as indicated by the hemodynamic computations. These results may be useful in simpler modeling of hemodynamics of diseased epicardial coronary vessels of similar size and shape in terms of using a pressure recovery coefficient, $c_{pr} = kc_{pr\infty}$, distal to the throat where flow separation and reattachment processes occur.

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