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COMPARISON BETWEEN CALCULATED PHYSIOLOGIC AND MEASURED PHASIC PRESSURE GRADIENTS ACROSS HUMAN STENOSES REMODELED BY CORONARY ANGIOPLASTY

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ABSTRACT

The coupling of computational hemodynamics to endovascular diagnostics in human coronary artery stenoses during angioplasty procedures was evaluated. Calculated mean pressure gradients were much lower for physiologic flow than measured mean pressure gradients. The narrower flow cross-section with the catheter present effectively introduced a tighter stenosis than the enlarged residual stenoses after balloon angioplasty; thus elevating the pressure gradient and reducing blood flow during the measurements.

INTRODUCTION

Quantitative methods to measure the hemodynamic consequences of various endovascular interventions including balloon angioplasty are of clinical importance. Catheters, measuring translesional pressure drops during balloon angioplasty procedures, can cause flow blockage effects. Wilson and Laxson (1993) note that the specific effects of changes in hemodynamic conditions on pressure gradient measurements are not well described in the human coronary circulation. In this investigation computational hemodynamics is used to acquire information on changes in flow, flow regime, phase lag and the magnitude of phasic and mean pressure gradients during such measurements, compared to physiologic flow (without the catheter present).

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. The patients had single-vessel, single-lesion coronary artery disease. Dimensions and shape of the coronary stenosis after angioplasty were obtained from biplanar angiograms. After PTCA the average minimal diameter increased to $d_m = 1.8$ mm ($A_m = 2.5 \pm 0.1$ mm^2) within treated lesions; initially $d_m = 0.95$ mm ($A_m = 0.7 \pm 0.1$ mm^2). Average proximal diameter $d_c = 3$ mm was relatively unchanged in the procedure producing a residual 40% mean diameter stenosis after PTCA. Dimensional data on the shape of a similar size lesion are from Back and Denton (1992). Balloon angioplasty both lengthened and widened the narrowest region of the stenosis with resulting $l_m = 3$ mm. Despite axial redistribution of plaque away from the narrowest region, the constriction length $l_c = 6$ mm and divergence length $l_r = 1.5$ mm were roughly unchanged by balloon dilatation. The mean pressure measurements by Wilson et al. (1988) were made with the angioplasty catheter $d_i = 1.4$ mm spanning the lesions.

The coronary flow waveform used in the flow simulations was obtained in our laboratory from *in vitro* calibration (Cho et al. 1983) and smoothing the fluctuating Doppler signal. The spatial average velocity across the flow $\bar{u}(t)$ needed for flow simulations, is similar to that from Doppler catheter measurements in the LAD and LCX of patients undergoing PTCA (Sibley et al. 1986). The ratio of peak systolic to peak diastolic velocity was 0.4. Ratios for the RCA often are larger than for the LAD and LCX.

The flow simulations were carried out by solving mass and momentum equations using a Galerkin finite element method. The Carreau model was used for shear rate dependent non-Newtonian viscosity of blood. In the proximal vessel the spatial velocity profile was taken to be the usual Poiseuille profile for physiologic flow, and the analogue profile with the catheter (assumed to lie concentrically in the lesion). The calculations were initiated a distance proximal to the lesion for numerical stability purposes, and extended a distance distal to the lesion to allow for separated flow reattachment processes in the distal vessel where pressures were measured near the catheter tip. The calculations were for two consecutive pulse cycles in order to compare them and to obtain accurate results; the results for the second pulse cycle are calculated in continuation of the first one. Heart rate was 75 beats/min (period of a heart beat T = 0.8 s) and the density of blood $\rho = 1.05$ gm/cm^3. The finite-element code FIDAP, Sun Ultrasparc2 computer was used. Reported pressure drops $\Delta p(t) = p_r - p_e$ are instantaneous pressure differences between the stenosis inlet and distal region, thereby including pressure recovery therein. These are referred to as pressure gradients in the literature.

RESULTS AND DISCUSSION

Values of calculated $\Delta p(t)$ are shown in Fig. 1 during the cardiac cycle at time average (mean) flow rates $\tilde{Q}$ of 10, 30 and 50 ml/min with the catheter present. The shape of Δp with time is remarkably similar to the waveform velocity $\bar{u}$. The largest peak values Δp_{p-t} occur at peak diastolic flow, and the secondary peak in Δp occurs at peak systolic flow. There is virtually no phase lag in the instantaneous $\Delta p(t) - \bar{u}(t)$, relation and the flow is quasi-steady. Examination of details of the velocity field (not shown here) indicates that unsteady inertial effects appear to be secondary in the relatively low mean hydraulic Reynolds number ($\tilde{\mathrm{Re}} = 14 - 69$) viscous dominated flow field during the cardiac cycle. The frequency parameter $\alpha_e = 1.2$ was relatively small. In this flow regime, time average (mean) $\Delta\tilde{p}$, shown in Fig. 1 by the dotted lines, are relatively large at nominal flow rates.

A similar plot is shown in Fig. 2 for physiological flow at mean flow rates in the normal range including hyperemic response. For these calculations, $\tilde{Q} = 50$ - 200 ml/min, corresponding $\tilde{\mathrm{Re}}$ were 100 - 400, and $\alpha_e = 2.25$. In the physiologic flow regime for residual stenoses, common in angioplasty procedures, inertial effects and momentum changes are significant, and there is a phase lag in the $\Delta p - \bar{u}$ relation. However, most importantly, phasic pressure gradients are substantially lower in magnitude.

The results of these computations are shown in Fig. 3 by the mean $\Delta\tilde{p} - \tilde{Q}$ relation for physiologic flow, and for flow with the catheter present to make pressure measurements. The curves are vastly different. From these curves, the pressure measurements, and separate measurements of coronary flow reserve by Wilson et al. (1988) with the Doppler catheter ($d = 1.0$ mm) tip positioned proximal to the coronary lesions after PTCA (with minimal blockage) we deduce the following. For resting flow estimated flow reduction with the catheter present was about 0.4 of basal flow, and for hyperemia, about 0.2 of elevated flow at corresponding inferred measured values of $\Delta\tilde{p}$ of 10 and 20 mm Hg. For physiologic flow, mean pressure gradients were probably only about 1 mm Hg for basal flow, and about 8 mm Hg for hyperemic flow. More work is needed to evaluate hemodynamic interactions with smaller catheter based sensors evolving in clinical use for diagnostic purposes in native and remodeled stenoses.

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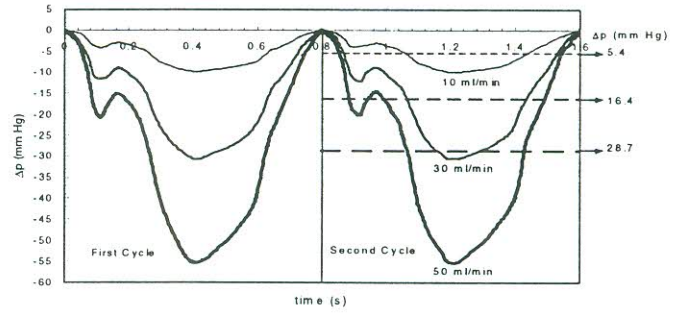

Fig. 1 Pulsatile pressure drop $\Delta p(t)$ across the stenosis during the cardiac cycle after angioplasty with the catheter present; mean flow rates $\tilde{Q}$ = 10, 30 and 50 ml/min.

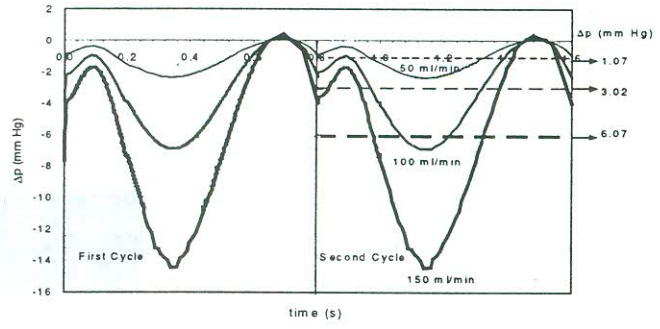

Fig. 2 Pulsatile pressure drop $\Delta p(t)$ across the stenosis during cardiac cycle after angioplasty for physiologic mean flow rates (without the catheter) $\tilde{Q}$ = 50, 100 & 150 ml/min.

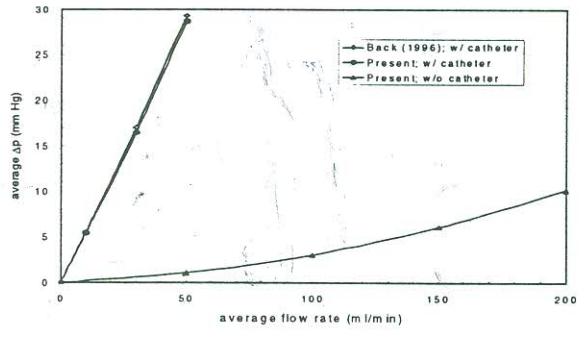

Fig. 3 Time (mean) pressure drop-flow rate relation, $\Delta\tilde{p} - \tilde{Q}$, for physiologic flow and with the catheter present after angioplasty.