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ELEVATED MEAN TRANSLESIONAL PRESSURE GRADIENTS AND FLOW LIMITATION IN SIGNIFICANT HUMAN CORONARY STENOSES BEFORE ANGIOPLASTY

Rupak K. Banerjee
Biomedical Industry Team
Fluent Incorporated
Lebanon, NH 03766

Lloyd H. Back
Jet Propulsion Laboratory
California Institute of Technology
Pasadena, CA 91109

Martin R. Back
Division of Vascular Surgery
University of South Florida
Tampa, FL 33606

Young I. Cho
Department of Mechanical Engineering & Mechanics
Drexel University
Philadelphia, PA 19104

ABSTRACT

Blood flow was simulated in patients (Wilson et al. 1988) with physiologically significant coronary lesions (minimal lesion size d_m = 0.95 mm; 68% mean diameter stenosis; measured coronary flow reserve (CFR) of 2.3) by using computational hemodynamics. Computed mean pressure gradients $\Delta\tilde{p}$ were about 9 mm Hg for basal flow, and flow limiting values about ~ 35 mm Hg. Mean hyperemic flow rates were about 115 ml/min, and distal mean coronary pressures ~ 55 mm Hg, a level known to cause ischemia in the subendocardium (Brown et al. 1984), and consistent with the occurrence of angina in the patients.

INTRODUCTION

This paper reports on the coupling of detailed hemodynamic computations to clinical data in patients with physiologically significant coronary lesions. The *in vivo* data set of Wilson et al. (1988) was used. The flow simulations are for physiologic conditions without diagnostic catheter sensors present within the native lesions that elevate the pressure gradient and reduce blood flow during measurements. Results of the simulations at basal and hyperemic flow conditions give numerical data on the pulsatile velocity profiles along the lesions; phasic and mean wall shear stress; pressure drop distributions and translesional pressure gradients; and phase relations. The flow regime was basically laminar in the narrow mean flow Reynolds number range $\tilde{\mathrm{Re}}_e \equiv 100 - 230$; the frequency parameter α_e= 2.25.

Here we show the physiological relation between mean translesional pressure gradient, flow and distal coronary pressure. The wall shear stress, not measurable in coronary lesions, determines the frictional contribution to pressure drop along the lesion. Relatively high levels of shear stress calculated in the narrow throat region will be reported subsequently.

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 with unstable angina pectoris or stable angina. Dimensions and shape of the coronary stenosis before angioplasty were obtained from biplanar angiograms. The average minimal diameter d_m = 0.95 mm ($A_m = 0.7 \pm 0.1$ mm^2). Average proximal diameter was d_e = 3 mm, producing a 68% mean diameter stenosis before PTCA. Dimensional data on the shape of a similar size lesion are from Back and Denton (1992). Before angioplasty, the length of the constriction region was l_c = 6 mm, the narrow throat length was l_m = 0.75 mm, and the divergence length l_r = 1.5 mm. Average distal diameter $d_r \simeq d_e$. Measurements of coronary flow reserve (CFR) by Wilson et al. (1988), with a 3F pulsed Doppler Ultrasound Catheter ($d \simeq$ 1.0 mm) with tip positioned proximal to the coronary lesions (with minimal blockage) was 2.3 $\pm$0.1 in the abnormal range for the patient group before angioplasty.

Accurate quantification of volumetric blood flow rates in stenotic vessels is difficult to determine clinically. Patients with abnormalities that might affect the vasodilator capacity of the arteriolar vasculature were excluded from their study. Mean arterial pressures $\tilde{p}_a = 89 \pm 3$ mm Hg were measured in the coronary ostium. The large measured mean pressure gradient of 56 mm Hg at basal flow using the angioplasty catheter was induced by flow obstruction (Wilson et al. 1988).

The coronary flow waveform used in the flow simulations was obtained in our laboratory from *in vitro* calibration (Cho et al. 1983), smoothing the fluctuating Doppler signal, and phase shifting the normal pattern for the proximal LAD. The spatial average velocity across the flow $\bar{u}(t)$ needed for flow simulations, is similar to that from Doppler catheter measurements in patients where normal peak diastolic flow is reduced by significant lesions (e. g., Wilson et al. 1988; Segal (*Cardioimetrics*, 1992). The ratio of relative peak diastolic to peak systolic velocity was 0.4 .

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. The calculations were initiated a distance proximal to the lesion to allow the pulsatile non-Newtonian blood velocity profile to develop before the stenosis inlet, and extended a distance distal to the lesion to allow for separated flow reattachment processes in the distal vessel. The calculations were for two consecutive pulse cycles in order to compare them and to obtain accurate results. Heart rate was 75 beats/min (period of a heart beat T = 0.8 s) and the density of blood ρ = 1.05 gm/cm^3. The finite-element code FIDAP, Sun Ultrasparc2 computer was used, which predicts measured pressures in vitro in a stenotic casting and model, and in vivo in a dog femoral artery within about 10%. 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 for physiologic flow during the cardiac cycle at time average (mean) flow rates $\tilde{Q}$ of 50, 75, and 100 ml/min. The shape of Δp with time is generally similar to the waveform velocity $\bar{u}(t)$. Inertial effects and momentum changes are significant and there is a phase lag in the $\Delta p - \bar{u}$ relation. Time average (mean) $\Delta\tilde{p}$ are shown by the dotted lines in Fig. 1.

The results of the computations are shown in Fig. 2 by the mean $\Delta\tilde{p} - \tilde{Q}$ relation, and fall in distal mean coronary pressure, $\tilde{p}_r = \tilde{p}_e - \Delta\tilde{p}$ with $\tilde{p}_e \cong \tilde{p}_a$, since mean flow resistances of proximal vessels were negligible compared to the large values across the stenoses which increased with flow rate. Mean pressure gradients $\Delta\tilde{p}$ were about 9 mm Hg for basal flow, and flow limiting values ~ 35 mm Hg , consistent with the measurements of abnormal CFR of 2.3. Distal mean coronary pressures $\tilde{p}_{rh}$ for hyperemic flow $\tilde{Q}_h$~115 ml/min, were in the range of 55 mm Hg known to cause ischemia in the subendocardium (Brown et al. 1984). The relative steepness of the $\Delta\tilde{p} - \tilde{Q}$ relation (Fig. 2) implies that variations of $\tilde{Q}_h$ in the patients were only moderately sensitive to variations of critical values of $\Delta\tilde{p}_h$ across the lesions.

These results show how computational fluid dynamics (CFD) in conjunction with clinical data in patients may be used to estimate critical hemodynamic variables before angioplasty; namely, hyperemic blood flow rates, translesional pressure gradients, and thus distal coronary pressures, difficult to measure accurately in patients. Progress on determining two points on the nearly linear maximal vasodilatation-distal perfusion pressure curve, CFR- $\tilde{p}_{rn}$ before and after angioplasty in the patients will be reported subsequently in connection with deducing by extrapolation the critical zero-flow pressure $\tilde{p}_{ro}$ important in assessing perfusion in different transmural layers of the left ventricle as shown by animal studies (e.g., Bache and Schwartz, 1982).

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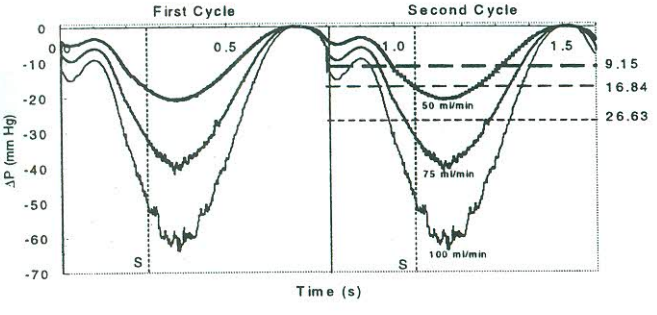

Fig. 1 Pulsatile pressure drop $\Delta p(t)$ across the stenosis during the cardiac cycle before angioplasty for physiologic flow rates $\tilde{Q}$ = 50, 75 and 100 ml/min.

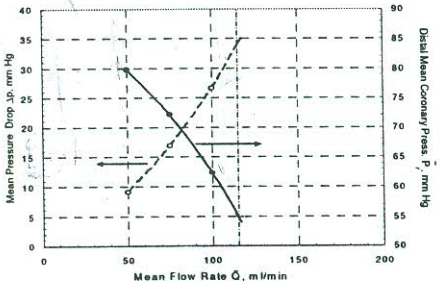

Fig. 2 Time (mean) pressure drop-flow rate relation $\Delta\tilde{p} - \tilde{Q}$, for physiologic flow before PTCA. The fall of distal mean coronary pressure $\tilde{p}_r$ is shown on the right y-axis.