Proceedings of IMECE'03
2003 ASME International Mechanical Engineering Congress & Exposition
Washington, D.C., November 16-21, 2003
IMECE2003-43138

GUIDEWIRE DIAGNOSTICS IN RESIDUAL CORONARY ARTERY STENOSES AFTER ANGIOPLASTY

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ABSTRACT

Detailed pulsatile hemodynamic computations were conducted in conjunction with previously reported clinical data in a group of patients with clinically significant coronary lesions remodeled by angioplasty. The hemodynamic effects of a small guidewire pressure sensor (d_i = 0.46mm) was evaluated, and indicated a moderate increase in hyperemic mean flow resistance, relatively small flow blockage, and elevation of the hyperemic mean pressure gradient by about 30% above the patho-physiological value in the 64% mean area stenosis with minimal diameter d_m = 1.8 mm. The ratio d_i/d_m was 0.26 or about ¼.

INTRODUCTION

This study gains insight on the hemodynamic effects of small guidewire catheter sensors in measuring mean trans-stenotic pressure gradients $\Delta\tilde{p}$ across residual or remodeled coronary artery stenoses. Detailed pulsatile hemodynamic computations were made in conjunction with previously reported clinical data in a group of patients with clinically significant coronary lesions remodeled by angioplasty (Banerjee et al., 2000). The purpose of this study is to ascertain changes in pulsatile hemodynamic conditions due to the insertion of a guidewire catheter (d_i = 0.46 mm) assumed to lie concentrically across the lesions to directly determine the mean pressure gradient ($\Delta\tilde{p}$) and fall in distal mean coronary pressure ($\tilde{p}_r$).

METHODS

For the 32 patient group of Wilson et al. (1988) [minimal remodeled lesion diameter d_m = 1.80 mm; proximal and distal vessel diameter d_e = 3 mm; 64% mean area stenosis; proximal (with minimal flow blockage) measured Coronary Flow Reserve (CFR) of 3.6 in the normal range] the diameter ratio of guidewire catheter to minimal lesion (d_i/d_m) was 0.26, thus producing an "artifactual" mean area stenosis of 65.5%.

The patients had single-vessel, single-lesion coronary artery disease with angina. For the native lesions, d_m = 0.95 mm; 90% mean area stenosis; CFR = 2.3 in the abnormal range. Patients with abnormalities that might affect the vasodilator capacity of the distal arteriolar vasculature were excluded from the study. Measured mean arterial pressure ($\tilde{p}_a$) in the (induced by repeated doses of papavarine) coronary ostium decreased from 89 to 84 mmHg in the procedure.

Details on the shape of the remodeled lesions; coronary flow waveform used in the flow analyses (typical of the proximal LAD), and computational method are given by Banerjee et al (2000) for patho-physiological flow, and by Banerjee et al (1999) with a larger catheter (d_i = 1.4 mm) in the flow.

RESULTS AND DISCUSSION

The results of the computations indicated a variable shift in the $\Delta\tilde{p}-\tilde{Q}$ relation due to guidewire-induced increases in mean flow resistances, $\tilde{R}=\Delta\tilde{p}/\tilde{Q}$. Both flow resistances increased with mean flow rate, $\tilde{Q}$, due to non-linear $\Delta\tilde{p}-\tilde{Q}$ relations, but the relative increase was much less with the guidewire spanning the lesion. For hyperemic flow, $\tilde{R}_{h-guidewire}$ was about 30% higher than $\tilde{R}_h$.

For patho-physiological flow, in the absence of a guidewire, the pressure gradient was 1.1 mmHg for basal flow of 50 ml/min (typical for a coronary vessel of 3 mm size). If the guidewire were present the pressure gradient of 2.2 mmHg would be about twice as large at this same flow rate, although actual values are not known.

For patho-physiological hyperemic flow $\tilde{Q}_h$ = 180 ml/min (CFR = 3.6) in the patients, the pressure gradient, $\Delta\tilde{p}_h \simeq 7.4$ mmHg. The estimated hyperemic flow conditions, if the guidewire were present and a vasodilator administered, indicated relatively small flow blockage of about 4% ($\tilde{Q}_{h-guidewire} \simeq 173$ ml/min). The elevated hyperemic pressure gradient $\Delta\tilde{p}_{h-guidewire} \simeq 9.5$ mmHg would amount to about a 30% increase above the patho-physiological value, or by a couple of mmHg.

The results of the complete set of guidewire computations will be reported at the conference: 1) to show the method, and to refine estimated hyperemic flow values; and 2) to discuss the nature of the coupled velocity and pressure fields during the cardiac cycle, and altered flow regime due to the presence of the guidewire.

ACKNOWLEDGMENTS

This work is supported by National-Scientific Development Grant (National-SDG) from the American Heart Association (AHA grant reference number: 033527N).

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