Analysis of pulsatile coronary pressure and flow velocity: looking beyond means
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Chapter 1

General introduction and aims
General introduction

Developments in measuring equipment technology have made possible the recording of intracoronary pressure and flow velocity in the catheterization laboratory with high accuracy and temporal resolution, using sensor-equipped guidewires. In the clinical practice however, for diagnostic and research purposes, only a small amount of the information included in the recorded signals is used, such as averages per beat, amplitudes, systolic/diastolic values and timing of events during the cardiac cycle.

Coronary blood flow and pressure vary during the heart cycle and their pulsatile character was acknowledged as early as 1689 by Scaramucci [1], who hypothesized that the coronary vessels are squeezed empty during ventricular contraction and are refilled during ventricular relaxation. This pulsatile nature is the result of both the pulsatility in the aortic pressure waveform and the compressing/releasing action of the beating heart on the intramural coronary vessels. The overall coronary blood flow profile in the epicardial vessels will in fact be determined not only by these two sources of pulsatility, but also by the intraluminal environment. For example, the presence of an epicardial stenosis and/or of atheromatous plaque and vessel wall stiffness are some of the local factors that also have an effect on the shape of the intracoronary waveforms.

This thesis deals with intracoronary pressure and flow velocity measurements derived in the catheterization laboratory from patients scheduled for elective balloon angioplasty and stenting of a single coronary artery. Pressure and velocity measurements were simultaneously acquired with a single, dual-sensor guidewire that incorporates a Doppler sensor at the tip and a pressure sensor 3cm distal to the tip (Volcano Corp., Rancho Cordova, CA). The small diameter of this wire (0.014-inch) allows for recordings distal to epicardial coronary stenoses to be made.

Aim and overview of the thesis

The general aim of this thesis is to report on techniques that seek to extract information about the cardiac and coronary environment from the phasic content of intracoronary waveforms. The value of the proposed techniques is assessed by comparing findings derived from them with the results obtained from more conventional, clinically used methods. We hypothesized that it is possible to unravel from the contours of the intracoronary waveforms information about the components of the cardiovascular system that have an effect on these contours.

A short description of cardiac mechanics and coronary physiology and pathophysiology is given in Chapter 2.

In Chapter 3 we present a technique for quantifying the pulsatile content of intracoronary pressure and flow velocity and demonstrate that it is possible to derive an index of stenosis severity that is based on the dynamic rather than the static component of these signals.

In Chapter 4 a new technique for derivation of wave speed from simultaneous pressure and velocity recordings was tested under different hemodynamic conditions. Our interest in wave speed stems not only from its relation to vessel wall properties, but also from its close connection to Wave Intensity Analysis (WIA).
In Chapter 5 we applied WIA to intracoronary pressure and flow velocity and investigated how changes in the coronary wave intensity contour during the heart cycle are related to changes in proximal (epicardial) and distal (microcirculatory) conditions in the coronary circulation.

In Chapter 6 we investigated whether the well-established beneficial effects of alpha-receptor blockade after percutaneous coronary intervention could be attributed to changes in the timing of cardiac contraction, as assessed from changes in diastolic time fraction.

In Chapter 7 we developed a model that predicts perfusion distribution over the ventricular wall. This chapter is incorporated in the thesis because it underlines the importance of epicardial hemodynamic measurements to be combined with information obtainable from perfusion distribution measurements, like MRI or other imaging modalities, in order for an accurate account of perfusion throughout the myocardial wall to be obtained.

A synthesis of our findings and general discussion is presented in Chapter 8.

Chapter 9 includes a more in-depth description of the theoretical basis of Wave Intensity Analysis and a short review of in vivo applications in the cardiovascular system.

References
