

## PREFACE

Cardiac pumping is dependent on cardiac perfusion. Hence, it is only natural that we address both cardiac perfusion and pumping in this book. We have gone one step further in also considering assisted perfusion by coronary bypass surgery and myocardial regeneration by means of stem cells transformed into implantable cardiomyocytes. The book is hence divided into three sections:

- (1) Cardiac Perfusion,
- (2) Cardiac Pumping Characteristics,
- (3) Assisted Perfusion and Pumping, and Myocardial Repair.

Section I on Cardiac perfusion starts out with the chapter on physiomics of coronary microcirculatory perfusion, which supplies nutrients to the heart myocardium for its contraction. The following chapter deals with the phenomenon of myocardial inhomogeneity, and provides an answer to this enigma as the basis of providing cardiac functional reserve. The next chapter is on quantification of cardiac perfusion and function, using nuclear cardiac imaging. The final chapter in this section is a synthesis of cardiac perfusion and pumping. It analyses left-ventricular (LV) pumping in terms of intra-ventricular blood flow velocity and pressure distributions, and also computationally depicts the distribution of pressure and flow velocities in discrete regions of the heart.

Section II is on how cardiac pumping is initiated by myocardial contraction, causing stresses and strains in the myocardium. More importantly, the mechanism of how myocardial contraction causes LV torsion is also discussed. The LV myocardial fibers are spirally wound inside its wall. Thereby, when they contract, the LV twists and then unwinds during relaxation. While this LV torsion is an end-product of LV myocardial fibers geometry and contraction, it can also serve as an index of contractility. Finally, in order for LV to contract or to depict its inability to contract adequately for adequate blood outflow, we have developed indices to assess its contractility, in the form of some intrinsic indices that correlate well with the traditional contractility index of  $(dP/dt)_{\max}$ . In doing so, we have also addressed a hitherto unexplained phenomenon of LV suction during its early filling state. The LV sarcomere is still contracting (albeit decreasing

its contractile force and shortening velocity) during early filling. It is this mechanism that causes LV suction (and resulting LV pressure decrease), before the left atrium starts to contract and cause LV filling.

Cardiovascular disease results in *in vivo* bioelectrical abnormalities, blocked coronary vessels and consequently myocardial infarcts. Hence, it is but natural that this book also deals (in Section III) with methods of augmented myocardial perfusion by coronary bypass surgery, mechanical circulatory support in the form of cardiac-assist devices, and myocardial regeneration by means of implantable three-dimensional cardiomyocyte constructs converted from embryonic stem cells. In this regard, this section discusses in detail the reasons for blocked distal coronary graft-occluded vessel anastomosis, the mechanical analysis of circulatory-support ventricular-assist systems, and in particular of an axial blood pump. The last two chapters deal with the basis and prospects of tissue engineering and novel approaches to cell transplantation following the conversion of embryonic stem cells into cardiomyocyte scaffolds for implantation.

We hope that this book can serve as a major reference resource in *cardiology* and *cardiac surgery*, as well be employable as a course text for a course on this topic in a biomedical engineering program. With these aims in mind, the individual chapters deal in adequate rigor with both theory and clinical applications.

Thank you.

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