Computational Cardiovascular Mechanics
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Modeling and Applications in Heart Failure
The ultimate aspiration of this book is to promote the application of computational cardiovascular mechanics models to clinical medicine. Patient-specific computational models can aid medical diagnosis and help design optimal treatment for cardiovascular disease. At present, however, most computational biomechanics modeling is still in the research and development stage. Further, the progress that has been made remains largely unrecognized in the clinic.

The objective of this book is, therefore, to present a comprehensive perspective on computational modeling of cardiovascular mechanics from both solid mechanics and fluid dynamics points of view. Since clinical translation of these models is the ultimate objective, we focus on applications in heart failure as this is an area that can particularly benefit from mechanical modeling. With our limited objective, this book does not claim to be a compendium or handbook of current information on the selected topics nor a review of literature; rather, it is largely works of the editors and their associates with a balanced point of view. A comprehensive bibliography is not provided; the list of references is limited to items quoted in the text.

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Introduction

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Abstract  Heart failure (HF) is a huge health and economic problem in the United States. As a consequence, a number of innovative device-based and surgical HF therapies have been introduced in the last 10 years. Examples include the total artificial heart, ventricular assist device, surgical ventricular remodeling, passive constraint, and resynchronization therapy. In general these therapies aim to either increase cardiac output or reverse left ventricular remodeling. However, a number of problems remain unsolved including the effect of therapy on left ventricular stress and function and intraventricular and intra-device flow and thrombus formation.

Mathematical modeling of the cardiovascular system using the finite element (FE) method has become both more powerful and easy to use. FE models of the heart now incorporate constitutive laws based on myocardial architecture that mimic the passive anisotropic nonlinear nature of the myocardium that can simulate active contraction. Inverse solutions now allow the calculation of myocardial material properties and stress. Computational fluid dynamics (CFD) studies can calculate endothelial shear stress and predict thrombosis and hemolysis. The FE method will play an increasing role in the understanding of cardiovascular pathology and in the design of therapies for HF.

The significance of HF as an economic and clinical problem will be discussed. Current device therapy will be discussed with an emphasis on problems that remain to be solved. Next, the chapter outlines the four basic biomechanics modeling steps. We conclude by introducing the chapters to follow.
1 Heart Failure

Heart failure (HF) is an increasing health and economic problem in the United States. For instance, one in eight deaths has HF mentioned on the death certificate. The number of “total mention” deaths from HF was 284,365 in 2004 with HF listed as the underlying cause in 57,120 [58]. Patients with HF use an increasing amount of health care resources. From 1979 to 2005, hospital discharges for HF rose from 400,000 to 1,084,000 [58]. The estimated direct and indirect cost of HF in the United States for 2008 is $34.8 billion [58].

HF is either caused by decreased left ventricular (LV) end-systolic elastance (systolic HF) or decreased diastolic compliance (diastolic HF) [1]. The most common cause of systolic HF is myocardial infarction (MI) secondary to coronary artery disease (~two-thirds of all HF). The annual incidence of MI is 920,000 [58]. Most current therapies, and all those discussed in this book, are designed to treat systolic HF.

The consequences of significant post-MI ventricular remodeling are substantial [2]. An increase in LV size after MI is one of the most important adverse prognostic findings [3–5, 55], and relatively small changes (25 cc) in end-systolic volume are associated with exponential increases in mortality [6]. Although the current incidence of LV aneurysm is unknown, Visner reported an incidence of 22% in 1986 [7] and Benediktsson reported an incidence of 5.3% in 1991 [8]. The percentage of patients with LV aneurysm who develop congestive heart failure (CHF) is 29% [9].

2 Cardiac Surgery

The last 50 years have seen an explosion of device- and engineering-related progress in cardiac surgery and the treatment of HF. Fig. 1 shows some of the landmark events and publications [10] that have occurred since the initial use of cardiopulmonary bypass by Gibbon in 1953 [11].

The number of routine cardiac surgical and interventional cardiology procedures has increased dramatically over the last 50 years. For instance, there were 699,000 open heart operations performed in 2005 [58]. An estimated 1,265,000 percutaneous coronary interventions (PCI) and 469,000 coronary bypass (CABG) procedures were performed in the United States in 2005 [58]. Finally, 106,000 valve prostheses were implanted in 2005 [58].

Survival after cardiac transplantation has gradually increased since the first human cardiac transplant by Barnard in 1967. In 2001, time to 50% survival after heart transplantation in an adult had increased to 10.3 years [12]. However, the need for surgical therapy for HF has outstripped demand. It is estimated that 40,000 people aged 65 or older may benefit from cardiac transplantation. Of those awaiting transplantation, 59% have a non-ischemic cardiomyopathy while 41% have an ischemic cardiomyopathy [57]. Although heart transplantation is increasing in
frequency, scarcity of donors limited the number of heart transplants performed in the US to 2,192 [58]. In 2001, 8.5% of those on the transplant waiting list died awaiting transplant [57].

3 New Devices and Operations

As a consequence, a number of innovative device-based and surgical HF therapies have been introduced, many in the last 10 years. Examples include mechanical circulatory support (total artificial heart, ventricular assist device (VAD)), surgical ventricular remodeling, passive constraint, and resynchronization therapy. In general these therapies aim to either increase cardiac output or reverse LV remodeling.

3.1 Mechanical Circulatory Support

In 1969, Cooley used an artificial heart as a bridge to transplantation but the patient died shortly after second operation [13]. In 1982, DeVries implanted a Jarvik-7 total artificial heart developed at the University of Utah by Jarvik, Kolff, and colleagues [14]. The patient lived for 112 days. Since those early days, there has been tremendous progress. In 2004, the REMATCH trial showed that treatment of end-stage HF with a VAD was a viable therapy [15]. Currently, a large number of VADs and artificial heart devices are under development for use as bridge to transplant, bridge to recovery, or destination therapy. Recent innovations include continuous flow VAD (Fig. 2) and VADs with hydraulic and magnetic rotor suspension.
3.2 Surgical Remodeling

A number of surgical procedures have been proposed to stop or reverse ventricular remodeling. In general, these procedures are designed to either decrease stress on the myocardium or to constrain the heart so that further enlargement cannot occur.

Although previously described by Cooley [16] and others, in 1984, Dor popularized the use of an endoventricular circular patch plasty ("Dor procedure") to restore LV size and shape by excluding the infarcted anterior LV wall and interventricular septum in patients with ischemic cardiomyopathy and either akinetic or dyskinetic anterior wall (Fig. 3) [17].

In 2009, the NIH sponsored Surgical Treatment for Ischemic Heart failure (STICH) trial found no survival benefit for surgical ventricular remodeling [18]. The STICH report noted that there may be specific subgroups of patients who might benefit from the combined procedure. However, at this time, the future of the Dor procedure and other surgical remodeling operations is unclear.
Also worth noting was the description in 1996 by Batista of a new surgical therapy for HF in patients with dilated cardiomyopathy (DCM) and end-stage CHF in which a viable “slice” of lateral LV wall was resected [19]. Because of problems with diastolic dysfunction and ventricular arrhythmias the Batista operation has largely been abandoned.

### 3.3 Passive Constraint

Passive LV constraint devices made of woven polyester fabric (Acorn Corcap TM Cardiac Support Device (CSD)) [20, 21] or nitinol (Paracor) [22] have been designed to prevent further ventricular remodeling. Passive LV restraint with Acorn and Paracor is arguably the least invasive and most likely to be applied to patients with ventricular remodeling and/or heart failure after myocardial infarction. Acorn and Paracor devices have not to date received FDA approval (Fig. 4).

![Fig. 4 Acorn cardiac support device (Jacket)](image)