Combined CFD/MRI analysis of blood flow in human left ventricle.

Seyed Saeid Khalafvand.

2013

Seyed Saeid Khalafvand. (2013). Combined CFD/MRI analysis of blood flow in human left ventricle. Doctoral thesis, Nanyang Technological University, Singapore.

<https://hdl.handle.net/10356/55149>

<https://doi.org/10.32657/10356/55149>

Combined CFD/MRI Analysis of Blood Flow in Human Left Ventricle

Seyed Saeid Khalafvand

School of Mechanical and Aerospace Engineering
Nanyang Technological University

A thesis submitted to the Nanyang Technological University in
partial fulfilment of the requirement for the degree of

Doctor of Philosophy

2013

Abstract

The study is to simulate blood flow processes in a human left ventricular (LV) via a combination of computational fluid dynamics (CFD) and magnetic resonance imaging (MRI). Cardiac MR images from normal and patient subjects are segmented and transformed to generate time-resolved two-dimensional (2D) and three-dimensional (3D) moving grids for blood flow using the arbitrary Lagrangian-Eulerian (ALE) formulation of the Navier-Stokes equations. The numerical solution of the Navier-Stokes equations yields pressure gradient in LV which is well correlated with the acceleration and deceleration of the diastolic and systolic flow. The complexity of transient spiral flow produced by the LV dilation and contraction is difficult to analyze and graphically presented. Thus 2D and 3D models of LV have been used to capture the main characteristics of blood flow in LV. The numerical simulation is performed for (i) 2D model of three normal and three heart failure subjects without valve leaflets (ii) 2D model of LV with and without mitral and aortic valve leaflets for a normal case (iii) 3D model of LV for a normal case (iv) 3D model of LV for a patient case before and after surgery. The results of the 2D modeling for three normal and three heart failure cases without valve leaflets show that the number of vortices and pressure difference

between basal and apical for normal cases is higher than abnormal cases. The effect of leaflets on fluid patterns during diastole and systole are assessed. The flow patterns are highly altered with presence of valve leaflets. The work-energy equation is well used to quantify the energy transfer from the contraction and dilation of the ventricle to the pulsating flow processes. The results show that the valve leaflets can not change the rate of energy transfer from LV in comparison with LV without valve leaflets. Furthermore, the pressure and vorticity contours in LV associated with net pressure and shear stress on leaflets are derived. The results show low shear stress on leaflets during diastole and systole. In a 3D model of LV, the 3D flow processes are analyzed by calculating the Lagrange stream function on a sequence of longitudinal planes. The net rate of energy transfer from the wall motion to the blood flow in the ventricle is primarily contributed by the rate of kinetic energy. For the normal subject, the work done by shear stresses and the dissipation of energy are rather small. The results of energy characteristics show an optimal filling and ejection for a normal case. Moreover, the kinematic, dynamics and energy characteristics of blood flow in a LV before and after surgery are quantified. All mentioned characteristics are improved for after surgery LV. The Theoretical and numerical assessment of LV blood flow might provide a basic understanding of fluid mechanics of normal and abnormal ventricular dilation and contraction.

Acknowledgements

First and foremost, I would like to thank my family for their love, support, inspiration and advice. I cordially confess that I am richly blessed to have my parents who are always there for me. I would like to thank my father for giving me strength and my mother for her unwavering love in my life. I also would like to thank my brother, Ghobad, for his support from an early age to present. Ghobad, with no doubt, I would never had started PhD, if you did not support me. Utmost thanks go to the most special person in my life who she was my best friend and eventually is my wife, now. Since the first day, she tried to show me the real life via the true love. Ghazal, truly speaking, there is no word I can find to thank you.

Next, I wish to express my sincere appreciation to my thesis supervisor Associate Professor Eddie Yin-Kwee Ng for his constant guidance, encouragement, patience and motivated me to do the best in my research although he was very busy. Thank you for being patient with me in every step. I am also thankful to my co-supervisor Dr. Zhong Liang (National Heart Center, Singapore) for providing the medical data of my thesis and his useful suggestions in a number of aspects of the work.

I would like to express my profound appreciation and sincere thanks

to Professor Tin-Kan Hung. He was beside me in every step of doing this project during my PhD attachment in University of Pittsburgh (PA,USA). He was a real knowledgeable person. Like a father for his child, he taught me how to properly conduct scientific research, be a good scientist and eventually an inventor. I am very grateful to his sharp, constructive and goal-oriented guidance on this project especially when I might have been deviated from the main objective.

I will also not forget my friends in NTU who have provided me joyful moments in Singapore.

Finally, I would like to thank A*STAR and NTU for their financial support in SINGA scholarship, all the staff and students of MAE for their friendship.

Contents

Contents	v
List of Figures	ix
List of Tables	xvii
Nomenclature	xvii
1 General Introduction	1
1.1 Introduction	1
1.2 Motivation	2
1.3 Heart physiology	2
1.4 Cardiac cycle	4
1.5 Objective and outline of this study	5
2 Literature Review	8
2.1 Introduction	8
2.2 Experimental studies	9
2.3 Numerical methods	10
2.3.1 Generic models	11

CONTENTS

2.3.2	Geometry prescribed CFD/MRI method	13
2.4	Summary	14
3	Methodology	16
3.1	Introduction	16
3.2	MRI data acquisitions and image processing	16
3.3	CFD modeling	19
3.3.1	Geometry reconstruction and grid generation	19
3.3.2	Governing equations	21
3.3.3	CFD simulation and boundary conditions	23
3.4	Summary	27
4	Two-Dimensional Modeling of LV for Normal and Abnormal Cases Without Valve Leaflets	28
4.1	Introduction	28
4.2	Biodata for cases	29
4.3	Grid generation	29
4.4	Initial and boundary conditions	31
4.5	Results	31
4.5.1	Temporary LV volume and velocities for inlet and outlet	32
4.5.2	Velocity distribution in normal and MI subjects	34
4.5.3	Pressure drop in normal and MI subjects	39
4.6	Discussion	42
4.6.1	Analysis of LV vortex, velocity and pressure distributions	42
4.6.2	Limitations	43
4.6.3	Clinical implications	43

CONTENTS

4.6.4	Conclusion	44
5	Two-Dimensional Modeling of LV with Valve Leaflets for a Normal Case	46
5.1	Introduction	46
5.2	Methodology	50
5.3	Results and discussions	52
5.3.1	Diastolic flow development	52
5.3.2	Systolic blood flow	64
5.3.3	Pressure difference	77
5.3.4	Energy characteristics of diastolic and systolic flow	78
5.3.5	The effect of valve leaflets on fluid patterns	85
5.4	Conclusion	89
6	Three-Dimensional Modeling of LV for a Normal Case	90
6.1	Introduction	90
6.2	Methodology	91
6.3	Results and discussions	93
6.3.1	3D diastolic flow	94
6.3.2	Systolic flow during ventricular contraction	106
6.3.3	Pressure difference	113
6.3.4	Energy characteristics of diastolic and systolic flow	115
6.4	Conclusion	120
7	Three-dimensional Modeling of Blood flow in a Heart Failure Case Before and After Surgery	122

CONTENTS

7.1	Introduction	122
7.2	Patient	124
7.3	Results	124
7.3.1	Pre-surgery results	125
7.3.1.1	Flow patterns during diastole	127
7.3.1.2	Flow patterns during systole	134
7.3.1.3	Pressure difference	138
7.3.1.4	Energy characteristics	139
7.3.2	Post-surgery results	140
7.3.2.1	Flow patterns during diastole	142
7.3.2.2	Flow patterns during systole	149
7.3.2.3	Pressure difference	154
7.3.2.4	Energy characteristics	154
7.4	Discussion	155
7.5	Conclusion	159
8	Conclusions and Recommendations for Future Work	160
8.1	Concluding remarks	160
8.2	Contribution of this thesis	163
8.3	Recommendations for future work	163
	List of Publications Due to This Thesis	165
	Bibliography	167

List of Figures

1.1	Heart valves and ventricles during diastole and systole (Klabunde, 2011).	4
3.1	Siemens magnet field strength scanner(Remote sensing tutorial, 2009).	17
3.2	Short and long axis views of LV in CMRTools.	18
3.3	Oblique long axis MR image plane of LV.	19
3.4	A sample of unstructured grid of LV including proximal LA and ascending aorta for an abnormal case.	20
3.5	A sample of structured grid (Butterfly-Grid) for a normal LV. . .	21
3.6	A schematic view of one angular position for one layer.	24
3.7	Diagram of general simulation with re-meshing.	26
4.1	Results of grid dependency study.	30
4.2	Temporary variations of volume $vol(t^*)$ and velocity $V(t^*)$ (at mitral and aortic orifice) during diastole ($vol_D(t^*)$ and $V_D(t^*)$) and during systole($vol_O(t^*)$ and $V_O(t^*)$).	33
4.3	Comparison of streamlines at the onset of diastole for six cases; streamlines colored by velocity magnitude in ms^{-1}	35

LIST OF FIGURES

4.4	Comparison of streamlines at peak diastole for six cases; stream lines colored by velocity magnitude in ms^{-1}	35
4.5	Comparison of streamlines during slow filling phase for six cases; streamlines colored by velocity magnitude in ms^{-1}	36
4.6	Comparison of stream lines at end diastole for six cases; stream lines colored by velocity magnitude in ms^{-1}	37
4.7	Comparison of streamlines at peak systole for six cases; stream lines colored by velocity magnitude in ms^{-1}	38
4.8	Velocity at the mitral orifice during diastole and pressure difference between mitral orifice and apex ($\Delta P_D(t^*) = p_m(t^*) - p_a(t^*)$). . . .	40
4.9	Velocity at the aortic orifice during systole and pressure difference between apex and aortic orifice ($\Delta P_O(t^*) = p_a(t^*) - p_o(t^*)$). . . .	41
5.1	Temporary LV volume ($vol(t^*)$) versus dimensionless time(t^*). $vol_{max}(t^*)$ is the volume at the end of diastole.	50
5.2	Prescribed leaflets motions for some time steps (a) Mitral valve leaflets motions during rapid filling (from closed position to maximum opening). (b) Mitral valve leaflets motions during slow filling, late filling and closing (from maximum opening position to closed position). (c) Aortic valve leaflets motions during opening for some time steps (the reverse motion is for closing).	51
5.3	Temporary flow velocity at inlet ($V_D(t)$) and outlet ($V_O(t)$) versus time.	53
5.4	Flow patterns during rapid filling at (a) $t = 0.0185 \text{ s}$ (b) $t = 0.0369 \text{ s}$ (c) $t = 0.0738 \text{ s}$. Streamlines colored by velocity (cm s^{-1}). . . .	55

LIST OF FIGURES

5.5	Flow patterns during rapid filling at (a) $t = 0.1476$ s (b) $t = 0.1845$ s (c) peak of diastole; $t = 0.2214$ s. Streamlines colored by velocity (cm s^{-1}).	57
5.6	Flow patterns during decelerated diastole and slow filling at (a) $t = 0.2952$ s (b) $t = 0.3321$ s (c) $t = 0.369$ s. Streamlines colored by velocity (cm s^{-1}).	58
5.7	Flow patterns during slow filling and accelerated re-filling at (a) $t = 0.4428$ s (b) $t = 0.4797$ s (c) $t = 0.5166$ s. Streamlines colored by velocity (cm s^{-1}).	59
5.8	Flow patterns during decelerated re-filling and end-diastole at (a) $t = 0.5289$ s (b) $t = 0.5412$ s (c) $t = 0.5535$ s. Streamlines colored by velocity (cm s^{-1}).	60
5.9	Pressure contours during the mitral valve opening phase and net pressures (P_L) along the mitral leaflets for (a) rapid filling (b) peak of inlet velocity (c) during closing of mitral valve.	62
5.10	vorticity (ω) profiles on leaflets and LV for (a) rapid filling (b) peak of inlet velocity (c) during closing of mitral valve.	63
5.11	Flow patterns during IVC and initial systole at (a) $t = 0.572$ s (b) $t = 0.575$ s (c) $t = 0.578$ s. Streamlines colored by velocity (cm s^{-1}).	65
5.12	Flow patterns during initial time steps of systole at (a) $t = 0.581$ s (b) $t = 0.584$ s (c) $t = 0.588$ s. Streamlines colored by velocity (cm s^{-1}).	67
5.13	Flow patterns during accelerated ejection at (a) $t = 0.593$ s (b) $t = 0.603$ s (c) $t = 0.613$ s. Streamlines colored by velocity (cm s^{-1}).	68

LIST OF FIGURES

5.14	Flow patterns during accelerated systole at (a) $t = 0.627$ s (b) $t = 0.638$ s (c) $t = 0.648$ s. Streamlines colored by velocity (cm s^{-1}). . .	69
5.15	Flow patterns at (a) peak of systole; $t = 0.664$ s (b) $t = 0.775$ s (c) $t = 0.812$ s. Streamlines colored by velocity (cm s^{-1}).	70
5.16	Flow patterns during AV closure at (a) $t = 0.830$ s (b) $t = 0.849$ s (c) $t = 0.886$ s. Streamlines colored by velocity (cm s^{-1}).	71
5.17	Pressure contours and average of net pressure (P_L) on the AV leaflets during opening at Reynolds number (a) 1825 (b) 2834 and (c) 3270. The dimension of the numbers is Pa.	73
5.18	Pressure contours and average of net pressure (P_L) on the AV leaflets during closing at Reynolds number (a) 2031 (b) 1738 and (c) 1534. The dimension of the numbers is Pa.	74
5.19	Vorticity and shear stress distribution of the flow past the AV for Reynolds Numbers (a) 3270 (b) 3370 (c) 1534.	76
5.20	The pressure difference $\Delta P_D(t^*)$ during diastole and $\Delta P_O(t^*)$ and $\Delta P_S(t^*)$ during systole for 2D modeling with valve leaflets.	77
5.21	Rate of works during diastole in a 2D modeling of LV.	81
5.22	Rate of works during systole in a 2D modeling of LV.	83
5.23	Rate of works for 3D modeling of LV during systole.	84
5.24	Streamlines during rapid filling phase for LV without leaflets.	85
5.25	Streamline during slow filling, late filling and end-diastole for LV without leaflets.	86
5.26	Streamline during systole for LV without leaflets.	87
5.27	Pressure difference during diastole and systole for LV without valves.	88

LIST OF FIGURES

5.28	Pressure difference during one LV cycle for with and without presence of leaflets.	88
6.1	Temporary LV volume ($vol(t^*)$) versus dimensionless time(t^*). $vol_{max}(t^*)$ is the volume at the end of diastole. $t^* = 0$ shows the beginning of diastole and $t^* = 1$ shows the end of systole.	91
6.2	(a) Reconstruction of geometries of LV, left atrium and ascending aorta at the end-diastolic and end-systolic phases. (b) Grid topology for the LV, left atrium and ascending aorta. Top right shows the left atrium and ascending aorta view, and the bottom right shows apex view.	92
6.3	Time variations of the inlet velocity ($V_D(t)$) and the outlet velocity($V_O(t)$).	93
6.4	Four 2D longitudinal sections: P-P', Q-Q', R-R' and S-S'; and horizontal cross section H in two different views.	94
6.5	Streamlines during early phase of diastole at $t/T = 0.04$	96
6.6	Streamlines during diastolic acceleration at $t/T = 0.17$	97
6.7	Streamlines at the peak of inlet velocity during diastole; $t/T = 0.25$	98
6.8	Streamlines during decelerated early filling of diastole at $t/T = 0.33$	99
6.9	Streamlines during slow filling phase of diastole at $t/T = 0.46$	101
6.10	Streamlines during late filling phase of diastole at $t/T = 0.54$	103
6.11	Streamlines during late diastole at $t/T = 0.58$	104
6.12	Streamlines and velocity vectors at the end of diastole; $t/T = 0.63$	105
6.13	Streamlines during accelerated systole at $t/T = 0.66$	107
6.14	Streamlines at the peak of outlet velocity during systole; $t/T = 0.75$	108
6.15	Streamlines during systolic deceleration at $t/T = 0.83$	109

LIST OF FIGURES

6.16	Streamlines during decelerated ejection at $t/T = 0.91$	110
6.17	Streamlines during systolic deceleration at $t/T = 0.95$	111
6.18	Streamlines at the end of systole; $t/T = 1$	112
6.19	The time variation of the inflow velocity $V(t^*)$ and the pressure drop $\Delta P(t^*)$ in the 3D LV during diastole and systole.	113
6.20	Comparison of pressure differences between 3D modeling and 2D modeling with and without valves leaflets.	114
6.21	Rate of works during diastole for 3D modeling of LV.	118
6.22	Rate of works during systole for 3D modeling of LV.	119
7.1	Temporary volume versus dimensionless time for 25 before and after surgery LV geometries which derived from MRI; $t^* = 0$ and $t^* = 1$ show the first-diastole and end-systole, respectively.	125
7.2	Temporary velocity versus time for pre-surgery LV.	126
7.3	Pre-surgery end-diastole and end-systole geometries.	126
7.4	Flow patterns during initial time steps of diastole at $t = 0.048$ s for pre-surgery LV.	128
7.5	Flow patterns during accelerated diastole at $t = 0.192$ s for pre-surgery LV.	129
7.6	Flow patterns during accelerated diastole at $t = 0.337$ s for pre-surgery LV.	130
7.7	Flow patterns at the peak of diastole for pre-surgery LV.	131
7.8	Flow patterns during decelerated diastole at $t = 0.626$ s for pre-surgery LV.	132
7.9	Flow patterns during IVC at $t = 0.722$ s for pre-surgery LV.	133

LIST OF FIGURES

7.10 Flow patterns at the initial time steps of systole at $t = 0.730$ s for pre-surgery LV.	134
7.11 Flow patterns at the initial time steps of systole at $t = 0.771$ s for pre-surgery LV.	135
7.12 Flow patterns at the peak of systole at $t = 0.915$ s for pre-surgery LV.	136
7.13 Flow patterns at the end of systole at $t = 1.155$ s for pre-surgery LV.	137
7.14 The pressure difference $\Delta P_D(t^*)$ during diastole and $\Delta P_S(t^*)$ during systole for pre-surgery LV.	138
7.15 Rate of works during diastole for pre-surgery LV.	139
7.16 Rate of works during systole for pre-surgery LV.	139
7.17 Temporary velocity versus time for post-surgery LV.	141
7.18 Post-surgery end-diastole and end-systole geometries.	141
7.19 Flow patterns during initial time steps of diastole at $t = 0.033$ s for post-surgery LV.	142
7.20 Flow patterns during accelerated diastole at $t = 0.100$ s for post-surgery LV.	143
7.21 Flow patterns during accelerated filling at $t = 0.200$ s for post-surgery LV.	144
7.22 Flow patterns at the peak of diastole for post-surgery LV.	145
7.23 Flow patterns during decelerated diastole at $t = 0.401$ s for post-surgery LV.	146
7.24 Flow patterns during last time steps of diastole at $t = 0.435$ s for post-surgery LV.	147
7.25 Flow patterns during IVC at $t = 0.468$ s for post-surgery LV. . . .	148

LIST OF FIGURES

7.26 Flow patterns at the first time steps of systole at $t=0.476$ s for post-surgery LV.	149
7.27 Flow patterns at the first time steps of systole at $t = 0.487$ s for post-surgery LV.	150
7.28 Flow patterns at the first time steps of systole at $t = 0.495$ s for post-surgery LV.	151
7.29 Flow patterns at the first time steps of systole at $t = 0.502$ s for post-surgery LV.	151
7.30 Flow patterns at the peak of systole at $t = 0.636$ s for post-surgery LV.	152
7.31 Flow patterns at the end of systole at $t = 0.803$ s for post-surgery LV.	153
7.32 The pressure difference $\Delta P_D(t^*)$ during diastole and $\Delta P_S(t^*)$ dur- ing systole for after surgery.	154
7.33 Rate of works during diastole for after surgery LV.	155
7.34 Rate of works during systole for after surgery LV.	155
7.35 Comparison between pressure difference before and after surgery. .	157

List of Tables

4.1	Biodata for normal (N1, N2, N3) and abnormal (A1, A2, A3) cases.	29
4.2	Comparison between variables of the healthy subjects and the patients based on MRI data.	32
5.1	Comparison between KE and WP during diastole and systole. . .	84
7.1	Biodata before and after CABG+SVR (Male, 48 years old). . . .	124
7.2	Comparison between pre-surgery and post-surgery LV with normal LV.	158

Nomenclature

$2D$	Two-Dimensional
$3D$	Three-Dimensional
μ	dynamic viscosity
ω	vorticity
ρ	density
τ	viscous stress
V_D	inlet velocity during diastole
KE	total kinetic energy transfer
$\mathbf{n}$	normal vector
V_O	outlet velocity during systole
P	pressure
p_a	pressure at the apex
P_L	net pressure on the leaflet

NOMENCLATURE

p_m	pressure at the entrance of LA
p_o	pressure at the outlet of aortic root
Re	Reynolds number
Re_O	Reynolds number during diastole
Re_D	Reynolds number during systole
T	period of one LV cycle
t	dimensional time
t_D	Diastolic period
t_S	Systolic period
t^*	dimensionless time
$\mathbf{v}$	fluid velocity vector in a fixed coordinate
$\mathbf{v}_b$	moving boundary velocity vector
vol	volume
WP	total work done by pressure
ALE	Arbitrary Lagrangian-Eulerian
CCW	Counter Clockwise
CFD	Computational Fluid Dynamics
CT	Computerized Tomography

NOMENCLATURE

<i>CW</i>	Clockwise
<i>EDV</i>	End-Diastolic Volume
<i>EF</i>	Ejection Fraction
<i>ESV</i>	End-Systolic Volume
<i>FSI</i>	Fluid-Solid Interaction
<i>GCI</i>	Grid Convergence Index
<i>IVC</i>	Isovolumetric Contraction
<i>IVR</i>	Isovolumetric Relaxation
<i>LA</i>	Left Atrium
<i>LV</i>	Left Ventricle
<i>MI</i>	Myocardial Infarction
<i>MR</i>	Magnetic Resonance
<i>MRI</i>	Magnetic Resonance Imaging
<i>AV</i>	Aortic Valve
<i>MV</i>	Mitral Valve
<i>RA</i>	Right Atrium
<i>RV</i>	Right Ventricle
<i>SV</i>	Stroke Volume
<i>UDFs</i>	User Defined Functions

Chapter 1

General Introduction

1.1 Introduction

Heart failure is one of the greatest killers in the world. It is a condition that impairs the ability of the heart to pump or fill with a sufficient amount of blood through the body. Left ventricular (LV) dysfunction is a very common consequence of diverse heart disease. In developed countries, around 2 percent of adults suffer from heart failure, but in those over the age of 65, this increases to 6-10 percent (McMurray & Pfeffer, 2005; Authors/Task Force *et al.*, 2008). It is estimated that there are more than 15 million new cases of heart failure each year worldwide (Klabunde, 2011). Heart diseases such as myocardial infarction, ischemia, aortic stenosis and mitral prolapse could have serious effects on cardiac function. Yoganathan *et al.* (2005) reported that there were more than 50 different types of artificial heart valves used annually for over 170,000 valve implants worldwide. Blood flow patterns are very complicated in normal and abnormal cases. Analysis of intraventricular blood flow characteristics will aid diagnosis

1. Introduction

and treatment of LV dysfunction.

1.2 Motivation

Kinematic, dynamic and energy characteristics of blood flow in human LV are complicated and not yet fully understood. In addition, the relationship between complex flow patterns in LV and wall motion, shape of left ventricle in normal and abnormal cases, and other physiologic functions is still under debate. The combination of non-invasive magnetic resonance imaging (MRI) and computational fluid dynamics (CFD) has been well recognized as an effective method to study the complex dynamics of the cardiovascular system, such as diastolic and systolic blood flows in a left ventricle.

The non-invasive measures like energy transfer, quantification of vortices and pressure differences between basal and apex could be complementary to the ejection fraction for normal and abnormal LV pumping function. These quantitative measures might provide a sensitive method to detect early stages of LV dysfunction for example, heart failure with normal ejection fraction. In addition, these non-invasive data can be used for improvement of surgical treatments.

1.3 Heart physiology

The detailed anatomy of the heart can be found in anatomy textbooks (e.g. Klabunde, 2011). The following presents only a brief description of heart anatomy, cardiac cycle and heart failure so that the physiology of the cardiac cycle can be appreciated. The heart consists of two ventricles and two atriums. Ventricles

1. Introduction

pump the blood into different organs while atriums act as reservoirs for ventricles. Each ventricle has two valves for inlet and outlet of blood flow. Oxygenate blood enters to the left atrium (LA) from the lungs through four pulmonary veins. Blood flows across mitral valve (MV) and enter into the LV from the LA. By contraction of LV, blood flows eject into the ascending aorta, which then distributes blood through the body. Oxygen-depleted blood flow returns to the right atrium (RA) via the two major veins (superior vena cava and inferior vena cava). Then, Blood passes from the RA to the right ventricle (RV) through the tricuspid valve. Because of RV thin wall, the power of RV is much smaller than LV. Through pulmonic valve, blood ejects from RV and enters to the lungs where exchange of oxygen and carbon dioxide occur. This cycle again will be repeated to provide enough blood for the body (Klabunde, 2011).

The four valves of heart consist of two or three leaflets. The tricuspid and mitral valves is attached to ventricular walls by papillary muscles. The papillary muscles generate tension on the valve leaflets during contraction to prevent the back flow into atriums (Klabunde, 2011). By contrast, the outlets of ventricles (pulmonic and aortic valves) do not have the papillary muscles because they are passive structures. During opening fluid flow push the leaflets aside out and during closure reverse flow motion move back them into passage (McQueen & Peskin, 2000). A schematic view of heart and cardiac cycle during diastole and systole is presented in figure 1.1.

The main common causes of heart failure are myocardial infarction (MI) and coronary artery disease. Coronary artery disease decreases oxygenate blood delivery to myocardium and causes the impaired function. In MI cases, infarcted tissues behave same as a rigid wall and do not expand and contract. Thus, non-

1. Introduction

Figure 1.1: Heart valves and ventricles during diastole and systole (Klabunde, 2011).

infarcted tissues must work harder than usual and this extra work can finally reduce the ability of them (overloading) and cause the heart failure (Klabunde, 2011).

1.4 Cardiac cycle

One cardiac cycle can be divided into four major phases: diastole, isovolumetric contraction (IVC), systole and isovolumetric relaxation (IVR). During diastole blood enters to the LV through the mitral valve by ventricular dilation while aortic valve is closed. Diastole can be divided into three sub-phases: early filling (rapid filling), slow filling (diastasis) and late filling (re-filling). By starting of LV expansion the mitral valve opens and because of high pressure difference between the LA and LV, accelerate blood enters into LV. As the LV continues to fills with blood and expands, the intraventricular pressure rises. Thus the pressure

1. Introduction

gradient across the mitral valve reduces so that the rate of filling falls. During the late filling the blood again is accelerated by atrial contraction. By end of late filling mitral valve closes and this phase is called IVC. During IVC both valves are closed and the volume is constant. During this phase intraventricular pressure rises and the LV will be ready for ejection.

By end of IVC, the systole is begun. During systole, the LV contracts and pushes the blood flow into ascending aorta through the aortic valve. In this phase, the mitral valve is closed and aortic valve is opened. Systolic phase also can be divided into two phases: rapid ejection (accelerated systole) and reduced ejection (decelerated systole). Generally during systole LV pumps the blood with high pressure into vessels but during rapid ejection, because of high pressure gradients, the blood velocity is higher than during reduced ejection. By closing aortic valve, IVR is begun. During this phase both valve are closed and the volume remains constant. LV begins to be relaxed and pressure decreases. By end of IVR one cardiac cycle will be completed.

1.5 Objective and outline of this study

The primary objective of this project is to investigate 2D and then 3D intra-ventricular flow patterns in both heart failure patients and normal hearts by combination of CFD and MRI techniques. This can provide practical and useful insight into how the LV is functioning in terms of blood flow velocity, pressure gradients, kinematic, dynamic and energy characteristics within it. It will shed light on how the LV copes with increased stiffness in disease states, for instance, myocardial infarct. Such analysis can help to facilitate the diagnosis and treat-

1. Introduction

ment of LV heart failure.

For simulation of LV, the research approach comprised of the following steps: (1) MRI images of the LV for 25 frames, (2) the construction of a dynamic model LV from the resulting images, (3) generation of computational mesh, and finally, (4) CFD simulation, post processing, analysis and induction of the results. This thesis is organized as follows:

Chapter 2 describes the background and related previous works.

In Chapter 3, the methodology of research is described and the details of computational modeling are discussed. In addition, a brief description of acquisition of MRI data and reconstruction of geometry are presented.

Chapter 4 presents the results of 2D simulation for three normal and three abnormal cases. The time variation of pressure drop is well correlated with the acceleration and deceleration of diastolic and systolic blood flows in the LV. In this chapter, we have demonstrated a quantitative assessment of LV conditions in normal and patients with myocardial infarction.

In Chapter 5, the results of 2D modeling for a normal subject with and without valve leaflets including the proximal LA and ascending aorta are presented. The vortices associated with the movements of valve leaflets, and flow acceleration and deceleration are discussed along with pressure drops, pressures on the leaflets and shear stresses. The cardiac pumping characteristics are further studied by the rate of energy transfer from the ventricle to the blood flow, the rate of work done by pressure and shearing stresses, the rate change of kinetic energy and dissipation of energy.

In Chapter 6, 3D modeling of LV including proximal left atrium and ascending aorta (without the valve leaflets) are modeled. The 3D flow processes are further

1. Introduction

analyzed by calculating the Lagrange stream function on a sequence of longitudinal planes. These 2D plots of flow patterns of longitudinal planes demonstrate not only the flow produced by the dilation and contraction but also the formation and decay of 3D vortices in the ventricle. The work energy equation is well used to quantify the energy transfer from the contraction and dilation of the ventricle to pulsating flow processes. The net rate of work done by pressure is primarily contributed by the rate of kinetic energy. For the normal subject, the work done by shear stresses and the dissipation of energy are rather small. The results of energy characteristics of diastolic flow are compared with those of a 2D model with opening and closing of the mitral leaflets as obtained from MRI data.

In Chapter 7, 3D modeling of LV for a case before and after surgery is described. The effects of surgery to kinematic, dynamic and energy characteristics of blood flow in LV are discussed.

Chapter 8 concludes and summarizes the work, contributions, and provides an insight into further work to be performed.

The contents of Chapters 2 to 7 are based on separate articles, *i.e.* Khalafvand *et al.* (2011), Khalafvand *et al.* (2012a) and Khalafvand *et al.* (2012b) which are published or have been submitted for publication (see page 165 for the list of publications due to this thesis). Hence, recurrence and overlap of methods and modeling descriptions occurs between these chapters.

Chapter 2

Literature Review

2.1 Introduction

In recent years, researchers have reported various models for studying flow patterns in the left ventricle. Since the heart is a multiphysics organ, Hunter *et al.* (2003) proposed to establish a computational framework for all heart functions including structure and cardiac cells behavior. However, the lack of knowledge about linking and interfacing the different functions in heart made it difficult to achieve this final goal of heart modeling. A particular problem of interest is the simplified models of blood flow in the LV. Researchers have tried to simplify the geometry and flow in LV with different assumptions and numerical approaches. Thus, in this chapter, we introduce important works related to the CFD simulation of flow through LV. Initially effective experimental studies are explained followed by the most active numerical approaches to simulate heart flow are described. It should be mentioned that other related studies will also be introduced in related chapters.

2. Literature Review

2.2 Experimental studies

Generally many experimental studies are based on Echo-Doppler or Phase-Mapping MRI measurements. Kim *et al.* (1995) investigated the vortex formation in LV using magnetic resonance velocity mapping. They found a uniform velocity distribution across the anterior to posterior diameter at the mitral valve. A large counter clockwise (CCW) vortex at the onset of slow filling phase in LV and a small one in the tip of mitral leaflets during late filling phase were observed. They did not observe any vortices during systole and blood flow was towards the outflow tract.

The important non-invasive measurements for a normal heart were done by Kilner *et al.* (2000). They used MR phase-velocity mapping to capture changes in the direction of blood. They focused on asymmetries and changes in direction of flow in heart chambers. During early diastole, they reported asymmetry recirculation of blood flow beneath of mitral valve leaflets especially round anterior leaflet of mitral valve. A part of blood flow also redirected towards the outflow tract. They proposed that the asymmetry recirculating flow might help to minimize energy dissipation in the heart.

Three dimensional *in vivo* velocity measurements were done by Ebberts *et al.* (2002). After velocity measurements, they solved Poisson equation to derive relative pressure in LV. The pressure differences between basal and apical during both diastole and systole were obtained. They found similar pressure differences patterns during diastole and systole. The pressure differences due to convective inertia were discussed during filling and ejection. They concluded that these pressure differences were small at the peak of inlet and outlet flow velocities,

2. Literature Review

while they dominated at the onset and termination of both ventricular filling and ejection.

2.3 Numerical methods

In recent years, the numerical simulation of the blood flow in LV has made great progress. Cheng *et al.* (2005) classified all numerical approaches into three types: geometry-prescribed CFD methods, fluid-structure interaction (FSI) methods and immersed boundary methods. Geometry prescribed CFD methods employed the boundary movement as a boundary condition. While both FSI and immersed boundary methods were aimed at modeling the interaction of fluid and structure. In 1972, Peskin introduced immersed boundary method for modeling the flow around heart valves (Peskin, 1972). The immersed boundary method simplified the structure as an elastic boundary which immersed in fluid, and interacting with it. This method developed in their latter studies for modeling of the blood flow in heart (McQueen & Peskin, 1985, 1989, 1997, 2000, 2002). The FSI methods used the finite element method for modeling the structure and CFD for fluid domain. The FSI methods were further developed in recent years and using more realistic coupling algorithms between fluid and structure (Watanabe *et al.*, 2004; Cheng *et al.*, 2005; Krittian *et al.*, 2010a).

In the present study geometry prescribed CFD method was used for modeling the blood flow in LV. Thus in this chapter the important studies by using this method are described. The studies which used geometry prescribed CFD method could be roughly divided into two groups: Geometry prescribed methods based on generic (or mathematical) models, and based on Computerized Tomography

2. Literature Review

(CT) or MRI data. Generally, geometry prescribed CFD methods use arbitrary Lagrangian-Eulerian (ALE) formulation of the Navier-Stokes equation. The ALE formulation allows the computation of the flow domain on a moving grid.

2.3.1 Generic models

In recent years, 2D and 3D generic models (Vierendeels *et al.*, 2000; Baccani *et al.*, 2002*a,b*, 2003; Bolzon *et al.*, 2003; Domenichini *et al.*, 2005; Domenichini & Pedrizzetti, 2011; Pedrizzetti & Domenichini, 2005) were developed and helped to a preliminary understanding of fluid dynamic in LV. Generic models mimic the LV deformation under axisymmetric assumptions as a portion of prolate spheroid (or truncated ellipsoid) with moving walls and usually use a symmetrical (centralized or non-centralized) circular orifice.

Vierendeels *et al.* (2000) modeled filling flow phase (diastole) in the LV by means of 2D axisymmetric model. They calculated the pressure and velocity distribution in the LV model and showed that during flow acceleration the vortices generated below of inlet and during deceleration moved towards apex.

Baccani *et al.* (2002*a,b*, 2003) studied the fluid dynamics during LV filling. A model of vortex dynamics in a truncated prolate spheroid was modelled by Baccani *et al.* (2002*a*). They focused on the birth, development and separation of vortex sheet which initiated from inlet edge. They described the results in terms of vorticity and observed that during deceleration the vorticity detaches from inlet edge and forms a vortex ring which moves towards the apex. They further studied the effects of mitral valve to LV fluid dynamics in a simple model of mitral valve (Baccani *et al.*, 2002*b*). They used different boundary conditions

2. Literature Review

instead of mitral valve to reproduce some major effects given by the presence of an orifice with opening leaflets (Baccani *et al.*, 2003). They showed that the vortex wake formed from the transmitral jet during rapid filling is more complex in the presence of new boundary conditions. The models were under axisymmetric assumptions but neglect 3D effects fluid flow.

Bolzon *et al.* (2003) investigated a pulse flow inside a cylindrical duct. The flow entered into the duct through a non centralized circular orifice. They analyzed the early stages of development of the 3D wake. They demonstrated how a small eccentricity produced a 3D vortex wake. They further suggested that the movement of vortex ring was due to viscous forces and boundary layer near the wall. Domenichini *et al.* (2005) extended the model of Baccani *et al.* (2002a) to analyze 3D effects of flow movement in a ventricular shape cavity. They analyzed the fluid flow for different magnitudes of eccentricity, flow and geometry parameters. They showed that the characteristics of entering jet directly affected the solution. Pedrizzetti & Domenichini (2005) with the similar method of their previous studies focused on the asymmetry in LV. They used a different jet model as an inlet for LV shape cavity to provide asymmetry conditions. They demonstrated that flow patterns in simplified normal heart were optimal in term of minimization of the energy dissipation and vortex was formed from mitral jet shear layer. Moreover, Domenichini *et al.* (2007) validated their results in an experimental model using a 3D axisymmetric model of LV.

Domenichini & Pedrizzetti (2011) investigated the changes of fluid dynamics in an infarcted ventricle with the same method of their previous works. They concluded that in the presence of apical akinesia and even more for dyskinesia, the flow could not travel toward apical area.

2. Literature Review

Generic models suffer from the unrealistic geometry for LV because they model it as a portion of prolate spheroid under axisymmetric assumptions. In addition, in all aforementioned works, they studied only the LV modeled during filling phase. These models provide a theoretical perspective for complex flow patterns in realistic intraventricular flow patterns, but they are far from a realistic modeling of LV.

2.3.2 Geometry prescribed CFD/MRI method

In this method the LV boundary movement is derived from MRI data and thus provided more realistic boundary conditions than generic models. This method which is similar to generic models used ALE formulation of Navier-Stokes equation allows the movement of boundaries.

Saber *et al.* (2001) used finite volume method in STAR-CD (Computational Dynamics Ltd., London, UK) to model the fluid flow in LV. They used orifices instead of valves and uniform velocity applied on orifices during diastole and systole. They observed circulation of blood flow beneath the mitral valve. Saber *et al.* (2003) completed and developed their previous study. They added proximal left atrium and ascending aorta to the model of LV. A large anterior CCW vortex and a small posterior CCW vortex were observed during diastole.

The effects of inlet boundary conditions on fluid flow patterns were assessed by Long *et al.* (2003). They used CFX4 (CFX international, AEA Technology, Harwell) solver for CFD modeling of intraventricular fluid flow. Hybrid and pressure boundary conditions were applied on inflow and sensitivity analysis were done. They concluded that the flow patterns were highly sensitive to inflow boundary

2. Literature Review

conditions. Long *et al.* (2008) investigated the flow patterns of LV using MRI data of six healthy cases. The methodology was the same as their previous study (Long *et al.*, 2003). They observed a main CCW vortex for three of the cases and CW vortex for one case during diastole.

Nakamura *et al.* (2006) examined four different type of mitral valve to assess their effects on intraventricular blood flow patterns. They used 3D symmetric geometry of LV. The mitral valve was treated as a 2D circular orifice without leaflets which the open area changed with time. They used four different modes of direction for opening. The results showed that the flow patterns in LV are different for each type of opening and might be important for efficient design of artificial heart valves.

Schenkel *et al.* (2009) simulated the LV blood flow in a model of LV including 2D presentation of valves. They projected the valves area on a circular surface in which the opening area changed with time. Generic model was used for upper areas of both mitral and aortic valves to provide more realistic boundary conditions. They used more accurate interpolation method (third order Bezier interpolation) than previous studies. They concluded that the initial vortex ring during diastole developed asymmetrically because of asymmetric jet in mitral valve.

2.4 Summary

There are three ways for numerical modeling of LV: Geometry prescribed methods, Immersed boundary method and FSI methods. It should be mentioned that each of different ways for LV modeling should be used in their right and could not be considered as an alternative to each other (Schenkel *et al.*, 2009). Two

2. Literature Review

different methods of Geometry prescribed CFD method were discussed. Generic models provided some basic concepts of LV flow physics which could help for better understanding of intraventricular blood flow patterns. Combined CFD/MRI method provided realistic boundary movement of LV and the results are validated in some studies (Schenkel *et al.*, 2009; Long *et al.*, 2008). In present study combined CFD/MRI method was used based on dynamic mesh and realistic MRI data. This type of method has been well recognized to provide useful data for clinicians. The methodology of present study is similar to Schenkel *et al.* (2009) but it is independent and provides more details of blood flow behavior (*i.e.* energy characteristics). The details will be discussed later in related chapters.

Chapter 3

Methodology

3.1 Introduction

In this chapter the methodology of simulation is prescribed. First, MRI data acquisition and processing are explained. Second, numerical methodology consists of grid generation, governing equations, boundary conditions and dynamic mesh method are explained.

3.2 MRI data acquisitions and image processing

MRI is a noninvasive and safe repeated technique for assessing heart functions and dysfunctions. MRI is safer than other methods such as X-ray and computed tomography (CT) (Schenck, 2000; De Gonzalez & Darby, 2004).

MRI scanning was performed using steady-state free precession cine gradient echo sequences. All subjects in this study, was imaged on a 1.5T Siemens scanner (Avanto, Siemens Medical Solutions, Erlangen) at department of cardiology of

3. Methodology

National Heart Center of Singapore (see figure 3.1). Some preliminary short-axis acquisitions were used to locate the plane passing through the mitral and aortic valves. The following planes (ventricular 2-chamber, 4-chamber and short-axis planes with 12-14 equidistant slices covering both ventricle and atrium) were acquired. The field of view was typically 320 mm with in-plane spatial resolution of less than 1.5 mm. Each slice was acquired in a single breath hold, with 25 temporal phases per heart cycle. The averaged duration needed for the entire image acquisition was 30 minutes. The short- and long-axes views (or planes) derived from the MRI were utilized to carry out 3D LV reconstruction in conjunction with dynamics LA and ascending aorta using a customized algorithm.

Figure 3.1: Siemens magnet field strength scanner(Remote sensing tutorial, 2009).

The processing methods of MRI data for LV were developed and used in National Heart Center previous studies (Zhong *et al.*, 2009, 2011; Yeo *et al.*,

3. Methodology

2009). Briefly, the MRI data were processed using a semi-automatic technique provided in the CMRTools suite (Cardiovascular Solution, UK). Short- and long-axis images were displayed simultaneously such that segmentation in the two planes proceeded interactively to reduce registration errors (see figure 3.2).

Figure 3.2: Short and long axis views of LV in CMRTools.

For each phase, every control point on the endocardium was constrained to lie on the intersection of the short- and long-axis views. The construction of long-axis planes (orthogonal to the short-axis planes oriented at regular angular intervals) enabled the fitting of a series of B-spline curves to represent the contours of the endocardial surface. This allowed the addition or manipulation of control points to obtain the desired boundary locations. The oblique long axis MR image was shown in figure 3.3.

The papillary muscles and trabeculae were included in the chamber volume

3. Methodology

Figure 3.3: Oblique long axis MR image plane of LV.

to obtain smooth endocardial contours. From the reconstructed LV, the chamber volume, stroke volume and ejection fraction were determined.

3.3 CFD modeling

3.3.1 Geometry reconstruction and grid generation

In order to solve the fluid domain, 25 frames of LV endocardial walls were derived from the MRI during one cycle. The reconstruction of LV geometry for CFD simulation was performed by using a semi-automatic method in Gambit 2.4.6 software. For each of segmented geometry, separate unstructured grids consisting of tetrahedral cells for 3D modeling and triangular cells for 2D modeling were

3. Methodology

generated in Gambit 2.4.6 (ANSYS Inc., 2009). It should be mentioned that, in the initial stages of the current thesis, both structured and unstructured grids were generated to model the LV. However, for all cases unstructured grids were used because most of modern CFD solvers were based on unstructured grids. One sample of unstructured grid for 3D modeling of LV including proximal LA and ascending aorta for an abnormal subject was depicted in figure 3.4. Moreover, one structured grid with quadrilateral cells for a normal 3D LV was shown in figure 3.5. The type of structured mesh in figure 3.5 was so called Butterfly-Grid which contained two O grids.

Figure 3.4: A sample of unstructured grid of LV including proximal LA and ascending aorta for an abnormal case.

The resolution of MRI time intervals was not enough fine for CFD simulation. To have refined resolution for CFD simulation, intermediate geometries were re-

3. Methodology

Figure 3.5: A sample of structured grid (Butterfly-Grid) for a normal LV.

quired to modeling the volume changes and to have the Courant number less than unity (product of local fluid velocity with ratio of time step to mesh spacing $\frac{u\Delta t}{\Delta x} < 1$) (Ferziger & Peric, 1997). For this purpose, after time step independency study, intermediate grids were generated for each initial measurement interval between 25 reconstructed grids from the original MRI data. That gave number of time steps for one cardiac cycle ($= 24 \times$ intermediate grids). The intermediate grids were generated by cubic spline interpolation.

3.3.2 Governing equations

For solving the fluid flow domain by finite volume method, the arbitrary Lagrangian-Eulerian (ALE) formulation of Navier-Stokes (Hirt *et al.*, 1974) is used for modeling the fluid flow in the LV. For an arbitrary volume V and surface S moving with local surface velocity of $\mathbf{v}_b$, the 3D integral form of continuity equation

3. Methodology

(Hirt *et al.*, 1974; Ferziger & Peric, 1997) is given as

$$\frac{\partial}{\partial t} \int_V \rho dV + \int_S \rho(\mathbf{v} - \mathbf{v}_b) \cdot \mathbf{n} dS = 0 \quad (3.1)$$

where $\mathbf{n}$ is the outwardly directed vector normal to dS . ρ is the density of the fluid, $\mathbf{v} = (u, v, w)$ is the velocity vector of the fluid in the fixed coordinate system, $\mathbf{v}_b = (u_b, v_b, w_b)$ is the velocity vector of the moving boundary (the velocity of boundary S of control volume V).

The momentum equation in 3D integral form (Hirt *et al.*, 1974; Ferziger & Peric, 1997) is given by

$$\frac{\partial}{\partial t} \int_V \rho \mathbf{v} dV + \int_S \rho \mathbf{v}(\mathbf{v} - \mathbf{v}_b) \cdot \mathbf{n} dS = - \int_S p \mathbf{n} dS + \int_S \boldsymbol{\tau} \cdot \mathbf{n} dS \quad (3.2)$$

where p is the pressure and $\boldsymbol{\tau}$ is the viscous stress tensor. In the equation (3.2), the gravity force is included in p for homogeneous fluid in a closed system. For an incompressible Newtonian fluid, $\boldsymbol{\tau}$ is expressed as

$$\tau_{ij} = \mu \left(\frac{\partial u_i}{\partial x_j} + \frac{\partial u_j}{\partial x_i} \right) \quad (3.3)$$

We have assumed μ is constant because blood flows in large arteries can be treated as homogeneous Newtonian fluid. The density and dynamic viscosity (μ) used in this study are 1050 kg m^{-3} , and $0.00316 \text{ Pa} \cdot \text{sec}$, respectively.

3. Methodology

3.3.3 CFD simulation and boundary conditions

In the present study, the mean velocities at inlet ($V_D(t)$) and outlet ($V_O(t)$) of the computational model were derived from the change of LV volume ($vol(t)$) as below:

$$V_D(t) = \frac{1}{A_{inlet}} \frac{\partial(vol(t))}{\partial t} \quad (3.4)$$

$$V_O(t) = \frac{1}{A_{outlet}} \frac{\partial(vol(t))}{\partial t} \quad (3.5)$$

They were uniformly applied at the inlet during diastolic flow and at the outlet for systolic flow. For exact inlet and outlet velocity profiles, MRI measurement is needed; however, the non-invasive measurements of Kilner *et al.* (2000) show that the velocity profiles at the inlet of LV approximately are uniform. In addition to the prescribed wall motion derived from MRI data, the velocity during diastole was applied at the inlet of left atrium along with no flow at the outlet of the aortic valve. During systole, the velocity at the outlet was applied along with no flow at the inlet.

The Navier-Stokes equations for the 3D flow with dynamic mesh were solved using a finite-volume based CFD solver ANSYS-Fluent 12 (ANSYS Inc., 2009). The dynamic mesh in ANSYS-Fluent uses the ALE formulation with re-meshing techniques. ANSYS-Fluent allows users to define the movement of boundaries by using user defined functions (UDFs). The motion of the LV boundaries is very complicated because of its movement in different directions. For describing the motion of LV boundaries, the UDFs were written by using C programming. As shown in the figures 3.4 and 3.5, the geometry of LV was constructed by differ-

3. Methodology

ent surface layers (horizontal slices) from the basal to apical. For describing the motion of the boundaries, each layer was divided into several surfaces by fixed angular positions in which all of them initiated from the center of the layer containing it. The points on the boundary could move inwards and outwards along a ray of fixed angular positions (Saber *et al.*, 2001). A schematic view of one angular position for one layer was shown in figure 3.6. The 3D LV models in present study contained around 120 layers and the motion of all of them were defined for CFD solver by writing UDFs. In every time step the UDFs were implemented

Figure 3.6: A schematic view of one angular position for one layer.

to update the motion of LV walls. The surface velocities for body fitted grid were calculated from the differences between the current and former grids. Mesh motion in a CFD solver such as ANSYS-Fluent does not change the topology of the mesh, and therefore if the deformation is excessive, the solver will fail. Under this situation, it is better to replace the distorted mesh with a new one.

The mesh motion is a result of boundary motion. The control of mesh quality is a technique to make sure the mesh is still usable when the domain deforms. The grid quality is monitored for every time step and solution is stopped when the

3. Methodology

grid quality is fallen under the criteria. The criterion for grid quality in present work is that the maximum value of face skew angle must be lower than 60 degree. In current study, the spring-based smoothing method is normally used for zones with a triangular or tetrahedral mesh. When the boundary displacement is large compared to the local cell sizes, the cell quality can deteriorate or the cells can become degenerate. This will invalidate the mesh (e.g., result in negative cell volumes) and consequently, will lead to convergence problems when the solution is updated to the next time step. Under these conditions re-meshing methods are used for degeneration of mesh in affected zones (ANSYS-Fluent 12 documentation, 2009).

There are different re-meshing methods in which they can use depend on the mesh problem. In this study local re-meshing method is used. Using the local re-meshing method, ANSYS-Fluent marks cells based on cell skewness and minimum and maximum length scales as well as an optional sizing function. ANSYS-Fluent evaluates each cell and marks it for re-meshing if it meets mesh criteria. The re-meshing techniques interpolate previous calculation results to the new mesh, and continue the iteration. More information about the different re-meshing methods are available in ANSYS-Fluent documentation (2009). Figure 3.7 shows a diagram of CFD solution including re-meshing loop.

Simulation was performed in a discontinuous time steps fashion during one cardiac cycle because for each time step there is a new grid (Schenkel *et al.*, 2009). To reach a periodic solution, simulation was repeated for four cycles. To achieve the best convergence and accurate solution, a second order upwind scheme was employed. The resulting algebraic equation system was solved using the implicit PISO (Pressure Implicit with Splitting of Operators) algorithm (Issa, 1986).

3. Methodology

Figure 3.7: Diagram of general simulation with re-meshing.

3. Methodology

3.4 Summary

In this chapter, the acquisition and processing MRI data and CFD modeling of fluid flow in LV was described. MRI data were segmented and reconstructed for 25 frames of LV during one cycle. Then, intermediate grids were generated between those initial frames which derived from MRI. Next, dynamic mesh in ANSYS-Fluent software based on UDFs was employed for modeling of LV motion boundaries. Smoothing and re-meshing methods were used to provide good quality for the new grids.

Chapter 4

Two-Dimensional Modeling of LV for Normal and Abnormal Cases Without Valve Leaflets

4.1 Introduction

In this chapter, patient specific 2D geometries were used to compute velocity and pressure fields. The aim was that to quantify the vortices and pressure difference between basal and apical for 2D modeling of LV. The modeling was performed by using normal and abnormal cases. Three healthy volunteers and three patients after myocardial infarction (MI) are included in this study. The general methodology for modeling of LV was discussed in the Chapter 3 and the relevant information for grid selection and time steps are presented in next sections.

Although 3D modeling is more realistic than 2D analysis, the input data are enormous if the twisting components of cardiac motion are included in the analy-

4. 2D Modeling of LV for Normal and Abnormal Cases

sis. As 3D pulsating blood flow processes are complicated to quantify and present graphically, 2D models are useful and practical to capture the main characteristics of the flow phenomena.

4.2 Biodata for cases

This study involved three normal subjects and three patients after MI. All subjects underwent diagnostic MRI scan. None of the normal subjects had (i) significant valvular or congenital cardiac disease, (ii) history of MI, (iii) coronary artery lesions, or (iv) abnormal blood pressure, end-diastolic volume (EDV) or ejection fraction (EF). The Biodata for normal and abnormal cases are shown in table 4.1.

Table 4.1: Biodata for normal (N1, N2, N3) and abnormal (A1, A2, A3) cases.

Case	Gender	Age	Blood pressure	Heart rates (beats/min)
N1	M	40	110/65	68
N2	M	50	128/81	80
N3	M	45	122/73	60
A1	M	55	107/71	90
A2	M	52	96/66	71
A3	F	55	172/112	108

4.3 Grid generation

In order to solve the fluid domain for one cycle, unstructured grids were generated first for each of MRI 25 frames of LV endocardial walls. The domain was covered

4. 2D Modeling of LV for Normal and Abnormal Cases

Figure 4.1: Results of grid dependency study.

by triangular elements. To obtain a continuous solution, the intermediate meshes were generated between every two frames. Since the duration of one LV cycle and geometry were different for all of cases, the intermediate grids and time steps were different. For example, for N1 case, to keep the Courant number closed to one, 70 intermediate meshes were needed when the time step is $0.53 \times 10^{-3}s$. The cubic spline interpolation was applied between individual grids to produce the 75 intermediate grids. Thus the position of each node passed continuously into each other. The surface velocities for ALE formulation were calculated from the displacement from the former grids to the current grids.

To find an optimum number of elements (cells) for modeling of the LV chamber, the grid convergence index (GCI) was used for assessing grid invariant solution (Roache, 1998). Four sets with different cells number (5000, 10000, 15000 and 20000) were tested for computing the GCI. As shown in figure 4.1, the results indicated that the global flow features did not change much when cell numbers were increased from 15000 to 20000.

4. 2D Modeling of LV for Normal and Abnormal Cases

4.4 Initial and boundary conditions

The mitral and aortic orifices were the inlet and outlet of the computational model, respectively. From MRI data, the time-varied LV volume was obtained. Its time derivative provided the input data of velocities at the mitral and aortic orifices. The diastolic flow simulation was simplified by using the prescribed diastolic velocity $V_D(t)$ at the inlet while in the systolic phase, the boundary condition was the outward velocity $V_O(t)$ at the aortic orifice. The other boundary conditions are the time-dependent velocities on the LV chambers, and on the closed valve and valve annulus. The input boundary condition for the computational flow simulation was based on time dependent uniform velocity profiles at the inlet and outlet and the motion of the LV chamber. The blood flow was characterized by rapidly accelerated and decelerated laminar flow due to LV dilatation and contraction.

4.5 Results

The study was aimed at investigating three patients with MI and three healthy volunteers. The MRI data after post processing were presented in table 4.2 for healthy volunteers and patient cases. For patient cases, the EF(mean value: 20.3% versus 69% for the normal) was significantly lower than the normal value, and their EDV(mean value: 276.7 ml versus 122.3 ml) and end-systolic volume(ESV)(mean value: 220.3 ml versus 37.3 ml) were much greater than those for normal subjects. Notice that in table 4.2, T is one cardiac cycle period which is divided into diastolic period (t_D) and systolic period (t_D).

4. 2D Modeling of LV for Normal and Abnormal Cases

Table 4.2: Comparison between variables of the healthy subjects and the patients based on MRI data.

variables	Normal Cases			Abnormal Cases		
	N1	N2	N3	A1	A2	A3
t_D (s)	0.59	0.49	0.77	0.36	0.47	0.28
t_S (s)	0.3	0.25	0.38	0.24	0.34	0.24
T (s)	0.89	0.74	1.15	0.6	0.81	0.52
EDV (cc)	139	107	121	329	244	257
ESV (cc)	34	33	45	259	170	232
SV (cc)	105	74	76	70	74	25
EF (%)	75	69	63	21	30	10

4.5.1 Temporary LV volume and velocities for inlet and outlet

The temporary volume and the velocity across mitral and aortic orifices used in this study are presented in figure 4.2. As described in Chapter 1, cardiac cycle consists of four phases: diastole, IVC, systole and IVR. During diastolic filling phase, the velocity-time curve at the mitral orifice usually had two peaks. The first peak was due to ventricular relaxation and called the early filling phase. The second peak was due to atrial contraction known as late filling phase. For two MI cases (A1 and A3), only one velocity peak appeared during diastolic filling phase. The curves $vol_D(t^*)$ and $V_D(t^*)$ show, respectively, the time variation of volume and velocity during diastole, and $vol_O(t^*)$ and $V_O(t^*)$ for systole. In figure 4.2, the dimensionless time is defined as $t^* = t/T$.

4. 2D Modeling of LV for Normal and Abnormal Cases

Figure 4.2: Temporary variations of volume $vol(t^*)$ and velocity $V(t^*)$ (at mitral and aortic orifice) during diastole ($vol_D(t^*)$ and $V_D(t^*)$) and during systole ($vol_O(t^*)$ and $V_O(t^*)$).

4. 2D Modeling of LV for Normal and Abnormal Cases

4.5.2 Velocity distribution in normal and MI subjects

To compare the flow patterns in the LV chamber, the results are discussed at the beginning of diastole, at the peak of diastole, during slow filling, at the end-diastole, and at the peak systole. Figure 4.3 presents the streamlines at the onset of diastole with colors for velocity magnitude. Since the presence and movement of the mitral valve were not modeled in the present study, the flow at the mitral valve was modeled as a bell mouth inlet with time-dependent uniform velocity profiles prescribed as input boundary condition. The curvature of the junction between the mitral valve and the aortic root resulted in high convective accelerations there. The flow patterns showed a typical interaction of accelerated flow and boundary layer development (Hung & Tsai, 1997). The blood from the mitral orifice entered into the LV and rapid filling phase began.

The streamlines at the peak diastole are depicted in figure 4.4. The results indicated that a strong clockwise (CW) vortex was developed near the junction of aortic and mitral valves. The flow patterns would be different if mitral leaflet motion were modeled. The accelerated blood flows with vortices were dominated by the size and dilation of the left ventricle. At a later time, the vortices became bigger. The results were similar to those reported by Kilner *et al.* (2000).

4. 2D Modeling of LV for Normal and Abnormal Cases

Figure 4.3: Comparison of streamlines at the onset of diastole for six cases; streamlines colored by velocity magnitude in ms^{-1} .

Figure 4.4: Comparison of streamlines at peak diastole for six cases; stream lines colored by velocity magnitude in ms^{-1} .

4. 2D Modeling of LV for Normal and Abnormal Cases

Figure 4.5 compares the flow patterns during slow filling phase for normal and abnormal LV dilatations. For the three normal cases, the generation of new vortices with the atrial contraction are evident. Because of the extended geometry of the aortic root, four vortices appeared for the N1 case. Only three vortices were developed for the N2 case; they were related to the smaller size of the LV chamber. The ventricle was almost occupied by the main vortex for the N3 case. The effect of the ventricle size, shape and velocities resulted in the turning of

Figure 4.5: Comparison of streamlines during slow filling phase for six cases; streamlines colored by velocity magnitude in ms^{-1} .

streamlines from the main flow to the dilating endocardium. The flow pattern for the N3 case was dominated by a strong CW vortex and smaller boundary vortices; they were produced by slow inlet momentum. Also shown in figure 4.5 are the

4. 2D Modeling of LV for Normal and Abnormal Cases

diastolic flow patterns for the MI patients with weak inflow and momentum; no new vortices were developed during late diastole.

At the end of diastole, the flow characteristics were changed rapidly by flow deceleration with vanishing inflow. The computational results at the end diastole are shown in figure 4.6. The flow patterns were characterized by the growth of vortices in size associated with rapid flow deceleration (Hung & Tsai, 1997).

In normal cases, the counter clockwise (CCW) and CW vortices appeared strong;

Figure 4.6: Comparison of stream lines at end diastole for six cases; stream lines colored by velocity magnitude in ms^{-1} .

there were also some small vortices existing near the mitral and aortic valves. In MI cases there were only two main vortices, one CW behind aortic valve and one CCW under the mitral valve. This is because of lower momentum and kinetic

4. 2D Modeling of LV for Normal and Abnormal Cases

energy of diastolic flow in the enlarged ventricles.

The velocity and pressure fields at the end diastole were used to model the systolic blood flow in the LV chamber. The model was based on mitral valve closed and aortic valve opened. The period of this phase was shorter than diastolic phase, characterized by higher flow acceleration from the LV wall to the aortic orifice. Due to the associated large pressure drop between the apex and the aortic valve, the streamlines were quickly straightening (from apex) all the way to aortic root. The vortices at the end diastole vanished quickly. The pressure drop increased for the rapidly accelerated systolic flow toward the aortic valve as illustrated in figure 4.7.

Figure 4.7: Comparison of streamlines at peak systole for six cases; stream lines colored by velocity magnitude in ms^{-1} .

4. 2D Modeling of LV for Normal and Abnormal Cases

4.5.3 Pressure drop in normal and MI subjects

The pressure difference ($\Delta P_D(t^*) = p_m(t^*) - p_a(t^*)$) between mitral orifice and the LV apex and that ($\Delta P_O(t^*) = p_a(t^*) - p_o(t^*)$) between the apex and the center of the aortic orifice are compared for all the cases of diastole (see figure 4.8) and systole (figure 4.9) flows.

During the filling phase, the maximum pressure difference occurred slightly ahead of the peak flow. For case N1, the pressure difference was much higher than the rest of five cases because of higher flow rate. As shown in figure 4.9, the maximum pressure differences in all cases appeared slightly ahead of the peak ejection. The increase and decrease in pressure difference is related to flow acceleration and deceleration. At the onset of systole, a pressure gradient increased as increase in the LV contraction. This pressure field redirected and accelerated the blood towards the LV outflow tract. Because of higher flow rates, the pressure difference, $\Delta P_O(t^*)$, of the normal cases was greater than that of the MI cases. In the normal subjects, the maximum pressure difference was 5.3 mmHg for case N2 during rapid systolic ejection; 4.2 mmHg for N1; and 3.53 mmHg for N3. In MI cases, the maximum pressure difference was 2.39 mmHg for case A2; 1.69 mmHg for A1; and 1.24 mmHg for A3.

4. 2D Modeling of LV for Normal and Abnormal Cases

Figure 4.8: Velocity at the mitral orifice during diastole and pressure difference between mitral orifice and apex ($\Delta P_D(t^*) = p_m(t^*) - p_a(t^*)$).

4. 2D Modeling of LV for Normal and Abnormal Cases

Figure 4.9: Velocity at the aortic orifice during systole and pressure difference between apex and aortic orifice ($\Delta P_o(t^*) = p_a(t^*) - p_o(t^*)$).

4. 2D Modeling of LV for Normal and Abnormal Cases

4.6 Discussion

4.6.1 Analysis of LV vortex, velocity and pressure distributions

In many previous studies, the left ventricle was modeled by using different assumptions and the pressure drop in LV for different cases were not reported. In this study, the pressure differences between the mitral valve and apex are compared for six cases in figure 4.8. The time scale is normalized by its duration for comparison. Clearly, in vivo measurements of pressure in the vortices are neither possible nor necessary; they can be computed. Figure 4.9 shows the calculated pressure drops for the ejection phase; they are normalized by its duration for comparison. The present results also showed the adverse drop occurred during flow deceleration (Hung & Tsai, 1997). A non-invasive measurement was reported by Ebbers *et al.* (2002) for a normal case. They concluded that, the pressure difference demonstrated the reversal of the transmittal pressure gradient during the LV filling and ejection. During these phases, intra-ventricular pressure gradients were observed. Our CFD results show similar results in figure 4.8 and figure 4.9. The maximum difference of apical and basal pressures during diastole occurred near the maximum acceleration and deceleration phases of the rapid filling and was in agreement with the values reported by Vierendeels *et al.* (2000), Ebbers *et al.* (2002) and Cheng *et al.* (2005). The flow patterns were very sensitive to the boundary conditions (Long *et al.*, 2003; Schenkel *et al.*, 2009) and the shape of LV, thus comparison between different cases could not be quantitative but qualitative. The flow patterns for different normal cases (figures 4.3 - 4.7) were

4. 2D Modeling of LV for Normal and Abnormal Cases

different because of different shape, contraction and dilation of LV and its associated inlet and outlet velocities. The results showed a large range of normal or abnormal cardiac contraction and dilation.

4.6.2 Limitations

The results of the two-dimensional model are limited. The streamlines are different from transient spiral flows in the ventricle. The 3D geometry and motion of the LV chamber result in secondary flows which are not included in the 2D analysis. Since diastolic flow in the LV chamber is primarily produced by the wall dilation and systolic flow by LV contraction, the magnitude of secondary flow is relatively small. The streamlines obtained in the 2D model indicate the main characteristics of flows produced in the left ventricle. Also, the study did not include the mitral valve motion in the LV flow processes. It was limited to six cases, and for a comprehensive conclusion more cases would be required. The complexity of 3D pulsating flows with 3D wall motion is very difficult to present graphically. It is believed that the pressure-flow relationship of the 2D model is a good indication for the 3D model which will be a follow-up study.

4.6.3 Clinical implications

There has been emerging interest in evaluation of intra-ventricular flow pattern and the underlying relationship between the intra-ventricular blood flow pattern and the ventricular function (Domenichini *et al.*, 2005; Kilner *et al.*, 2000). Several studies using MRI demonstrated that in the LV, inflow through the mitral orifice could give rise to recirculating flows beneath the valve leaflets and eventu-

4. 2D Modeling of LV for Normal and Abnormal Cases

ally becomes the intra-ventricular vortices (Domenichini *et al.*, 2005; Kilner *et al.*, 2000). The vortices dominated in the LV and redirected the blood flow toward the outflow track. Further, LV flow dynamics using MRI suggested that vortices within the LV reflected the function and geometry of the ventricle, and abnormal vortices might indicate the underlying suboptimal fluid dynamics of dysfunctional LV (Domenichini *et al.*, 2005; Baccani *et al.*, 2002a; Gharib *et al.*, 2006). But to our knowledge, no study was reported on quantification of vortex flow parameters related to the LV function. In this study, we presented information about the location and shape of vortices and pressure difference in relationship with the flow acceleration and deceleration. Because of lower stroke volume, the pressure differences were significantly lower in patients after MI compared to normal subjects. The study illustrated that these non-invasive measures like vortices and pressure differences could be complementary to the ejection fraction for normal and abnormal LV pumping function. These quantitative measures might provide a sensitive method to detect early stages of LV dysfunction for example, heart failure with normal ejection fraction (Zhong *et al.*, 2011).

4.6.4 Conclusion

The pressure differences were significantly lower in patients after MI compared to normal subjects. In the normal subjects, the mean EDV and ESV were 122.3 ± 16.04 ml (mean $\pm$ SD) and 37.3 ± 6.6 ml, respectively. The EF was $69 \pm 6\%$. While for the patients with MI, the mean EDV and ESV were increased to 276.7 ± 45.7 ml and 220.3 ± 45.6 ml and consequently EF was decreased to $20.3 \pm 10\%$. Ventricular flow patterns in the MI patients were significantly altered. At end-

4. 2D Modeling of LV for Normal and Abnormal Cases

diastole, three main vortices and several small vortices were observed in normal subjects. Because of lower momentum in an enlarged ventricle, only two weak vortices were seen in the MI patients. The maximum pressure differences between basal and apex were substantially larger in normal subjects than the MI patients (4.34 ± 0.89 mmHg vs. 1.77 ± 0.58 mmHg). The time variation of pressure drop was well correlated with the acceleration and deceleration of diastolic and systolic blood flows in the left ventricle. In this study, we demonstrated a quantitative assessment of LV conditions in normal and patients with MI. The results of this investigation appeared promising to provide useful information on intra-LV flow velocity and pressure drops with diverse heart diseases.

Chapter 5

Two-Dimensional Modeling of LV with Valve Leaflets for a Normal Case

5.1 Introduction

Left ventricle (LV) and the aortic and mitral valves have a crucial role to pump the blood through the body. Analysis of blood flow pattern in LV, mitral valve (MV) and aortic valve (AV) can provide useful information for diagnosis and treatment of LV and valves dysfunctions. MV and AV diseases could have serious effects on the efficiency of blood pumping by LV (i.e. mitral regurgitation and mitral stenosis). Thus for treatment of these diseases typical solutions are surgical repair or replacement of the valve. Annually, there are over 170,000 valve implants worldwide (Yoganathan *et al.*, 2005). Study of flow patterns over the valve leaflets is very important for diagnosis of the normal and abnormal func-

5. 2D Modeling of LV with Valve Leaflets

tions of AV and MV valves. Because of the above reasons, there are many studies about the modeling of AV and MV with different methods to reach an efficient design and to detect the abnormal function of the valves. All studies in this area can be divided into four types: AV modeling; MV modeling, LV modeling without valves and finally modeling of LV including the valves. The numerical methods for modeling the valves are the same as LV and described in Chapter 2. CFD analysis of fluid in AV is begun in the early 1970s. Bellhouse & Talbot (1969) and Bellhouse (1969) investigated experimentally and theoretically the fluid mechanics of the AV with a rigid-walled model of leaflets. They reported that flow deceleration produces adverse pressure gradient for the aortic valve closure without regurgitation. Gillani & Swanson (1976) numerically and experimentally investigated the formation and development of annular vortex in a spherical-cavity model of AV under axisymmetric assumptions. They concluded that because of an early separation, the cavity was occupied by vortices. Lee & Talbot (1979) further studied strong flow deceleration for optimal valve closure. The effect of Strouhal numbers on valve motion was reported by Van Steenhoven & Van Dongen (1979). Hemodynamic characteristics of the AV during opening, mathematically modeled by Hung & Schuessler (1977). In recent years 2D and 3D modeling of AV is developed by using FSI and dynamic mesh models. De Hart *et al.* (2000) and Stijnen *et al.* (2004) used FSI for 2D modeling of AV. In addition, a 2D FSI model of the AV developed based on the dynamic mesh method of the Fluent software by Dumont *et al.* (2004). The 3D symmetrical FSI model of AV is studied by De Hart *et al.* (2003) and they obtained velocity vectors around the aortic valve.

The modeling of MV is very complicated because of complex geometry, move-

5. 2D Modeling of LV with Valve Leaflets

ments of leaflets and relative movement of MV annulus to LV. Two-dimensional (2D) CFD models have been used for parametric design studies of mechanical heart valves (King *et al.*, 1994; McQueen & Peskin, 1985). Aluri & Chandran (2001) and Lai *et al.* (2002) simulated 2D flow of mechanical heart valves from a fully open position to closure and reported large negative pressure gradients for valve closure. Aortic flow through a bi-leaflet mechanical valve during the first half of systole was studied by King *et al.* (1996) using both CFD and experiment. Large vortices were observed on either side of the central orifice jet in the sinus area. Quaini *et al.* (2011) simulated hemodynamic conditions in regurgitant MV Flow. The results were compared with *in vitro* measurements. The finite element models also were used for further understanding of normal and abnormal mitral functions. Generally, they were employed to study stress and strain distribution for different shapes and material properties of the annulus, mitral valve and papillary muscles (Cochran & Kunzelman, 1998; Einstein *et al.*, 2004, 2005; Kunzelman *et al.*, 1993, 1997, 1998; Lim *et al.*, 2005; Maisano *et al.*, 2005; Prot *et al.*, 2009; Prot & Skallerud, 2009; Votta *et al.*, 2007, 2008). In addition, in recent years FSI models (Einstein *et al.*, 2010; Kunzelman *et al.*, 2007; Lau *et al.*, 2010) and IB models (Watton *et al.*, 2007, 2008) are developed for MV. Einstein *et al.* (2010) reviewed and discussed different FSI strategies and methods for modeling mitral valve motion.

All studies about the modeling of LV without valves leaflets (third type) were described in chapter 2 and divided into: 2D and 3D generic models (Vierendeels *et al.*, 2000; Baccani *et al.*, 2002*a,b*, 2003; Bolzon *et al.*, 2003; Domenichini *et al.*, 2005; Pedrizzetti & Domenichini, 2005), geometry-prescribed CFD methods (Saber *et al.*, 2001, 2003; Long *et al.*, 2003, 2008; Schenkel *et al.*, 2009), fic-

5. 2D Modeling of LV with Valve Leaflets

titious and realistic FSI methods (Peskin, 1972, 1977; McQueen & Peskin, 1985, 1989, 1997, 2000, 2002; Watanabe *et al.*, 2004; Cheng *et al.*, 2005; Krittian *et al.*, 2010b). In most of the previous studies, the mitral and aortic valve motions were not considered (Khalafvand *et al.*, 2011). In our knowledge there are not any attempt to model the LV associated with motion of valve leaflets. There are few studies (fourth type) which modeled the flow patterns in LV using 2D planar orifices instead of valves leaflet motion (Nakamura *et al.*, 2006; Schenkel *et al.*, 2009).

In this chapter we have tried to present a general study about 2D LV modeling for a normal subject. In several ways we have demonstrated new insights into kinematic, dynamic and energy characteristics of blood flow in LV. A 2D model of LV including both AV and MV is performed by using dynamic mesh. The flow patterns in LV associated with valve leaflets are derived from the Navier-Stokes equations. The pressure and shear stress on valve leaflets are derived during diastole and systole. In addition, the pressure drop and vorticity contours are studied for the flow past the MV and AV leaflets. Energy characteristics of LV during filling and ejection are analyzed by using work-energy equation. The work done by Kinetic energy, pressure, dissipation, shear stress and the rate change of kinetic energy are calculated. In order to assess the effects of valve leaflets on kinematic, dynamic and energy characteristics of blood flow in LV, the modeling is repeated for LV without presence of mitral and aortic leaflets during filling and ejection.

5. 2D Modeling of LV with Valve Leaflets

Figure 5.1: Temporary LV volume ($vol(t^*)$) versus dimensionless time(t^*). $vol_{max}(t^*)$ is the volume at the end of diastole.

5.2 Methodology

In order to solve the fluid domain, 25 frames of LV endocardial walls were derived from the MRI during one cycle. A typical temporary volume versus time for 25 LV geometries which derived from MRI can be seen in the figure 5.1. Notice that in figure 5.1, the dimensionless time is defined as $t^* = t/T$ (T = period of one cycle), thus $t^* = 0$ shows the beginning of diastole and $t^* = 1$ shows the end of systole.

For each of segmented geometry, separate unstructured grids consisting of triangular cells were generated, also by a semi-automatic method. The mitral and aortic valve motions during diastole and systole were derived from MRI data. The prescribed motions of leaflets for some time steps were shown in the figure 5.2.

To have a refined mesh, 75 intermediate grids were generated between initial 25 grids. In addition, in the first time steps of systole more intermediate grids were

5. 2D Modeling of LV with Valve Leaflets

generated to capture the changes of vortices.

Grid dependency study was done for five different grids and the number of grid

Figure 5.2: Prescribed leaflets motions for some time steps (a) Mitral valve leaflets motions during rapid filling (from closed position to maximum opening). (b) Mitral valve leaflets motions during slow filling, late filling and closing (from maximum opening position to closed position). (c) Aortic valve leaflets motions during opening for some time steps (the reverse motion is for closing).

cells were increased by the factor of 1.5 (6000, 9000, 13500, 20250 and 30375). The results showed that the global flow features (velocity component) did not change for the cell numbers above 20250. The changes of global flow features in the grid range of 13500 to 20250 were negligible, however, for more accurate

5. 2D Modeling of LV with Valve Leaflets

solution 20250 cell numbers was chosen for 2D modeling.

The boundary conditions were relocated to the inlet of LA and outlet of ascending aorta. The inlet velocity during diastole ($V_D(t)$) and outlet velocity during systole ($V_O(t)$) were applied in orifices. Figure 5.3 was shown for the change of inlet and outlet velocities versus time.

5.3 Results and discussions

For present normal subject, the end systolic volume (ESV) and end diastolic volume (EDV) are, respectively, 48.8 ml and 162.5 ml, resulting in the stroke volume (SV) of 113.7 ml and EF of 70%. The cardiac period (T) is 0.88 s, wherein diastolic time is 0.55 s and systole 0.33 s. Figure 5.3 shows temporal variations of the inlet velocity $V_D(t)$ at the left atrium for diastolic flow and $V_O(t)$ at the outlet for systole. Also shown in the figure 5.3 is the time variation of the Reynolds numbers:

$$\text{Re}_D(t) = \rho V_D(t) D_i(t) / \mu \quad (5.1)$$

$$\text{Re}_O(t) = \rho V_O(t) D_o(t) / \mu. \quad (5.2)$$

in which $D_i(t)$ and $D_o(t)$ are the dimension of the inlet and outlet of the computational model.

5.3.1 Diastolic flow development

Figure 5.4 shows the streamlines during rapid filling phase. The red curve on the left side of each contour shows the change of volume versus time as presented in

5. 2D Modeling of LV with Valve Leaflets

Figure 5.3: Temporary flow velocity at inlet ($V_D(t)$) and outlet ($V_O(t)$) versus time.

figure 5.1. The blue marker on the red line shows the volume of LV for selected time step. The mitral leaflets pushed open by blood flow due to contraction of LA. In a time interval of 0.0185 s, the Reynolds number (Re_D) increases from zero to 650. At $t = 0.0185$ s because of high inflow jet velocity, two vortices with different directions are formed at the tip of leaflets. A counter clockwise (CCW) vortex is formed at the tip of the anterior leaflet and a clockwise (CW) vortex at the tip of the posterior leaflet. However, the CCW vortex is stronger than CW vortex because of curvature effects. During the opening of valve leaflets, the direction of the motion of leaflets is toward downstream (LV side). Therefore, on the upstream surface of the moving leaflets, the streamlines end on the leaflet. On the downstream side, the vortex is dominated at the tip of leaflets and the momentum produced by the leaflet is directed to the vortex by streamlines. Generally in the lack of vortices the streamlines must initiate from the LV side of leaflets. However,

5. 2D Modeling of LV with Valve Leaflets

vortices push back the other streamlines on the LV side of leaflets and cause small vortices at each leaflet root. The current modeling includes the upward motion of the closed aortic valve. Thus, because of this upward motion the streamlines underneath of aortic root end on AV section. Because of motion of leaflets no boundary layer is developed on the surface of leaflets.

As the wall dilation is continued the diameter of inlet jet increases. The Re_D rises from 650 in figure 5.4a to 824 in figure 5.4b. The maximum velocity in inflow jet decreases from 66 cm s^{-1} to 40 cm s^{-1} because of increasing jet diameter. In figure 5.4c, the Re_D increases to 1355 and the curvature of anterior leaflet changes from convex to concave.

As shown in figure 5.5, the rapid filling continues and Re_D reaches to maximum magnitude of 2842. During $t = 0.084 \text{ s}$ to 0.192 s , the leaflets are fully open and most of blood flow enters to LV during this time interval. The vortices increases in size and move toward LV wall by motion of leaflets and accelerated blood flow from LA. The maximum velocity of blood flow in LV during diastole is 84 cm s^{-1} and occurs at $t = 0.2214 \text{ s}$ (see figure 5.5c).

With the start of flow deceleration, the diameter of inlet jet reduces and the Re_D is decreased from 2842 to 1762 at $t = 0.2952 \text{ s}$. During this time interval (0.0738 s) a rapid energy transfer occurs from the main flow to vortices during flow deceleration (Hung & Tsai, 1997). The evidence of this phenomenon is growth of vortices in figure 5.6a. During slow filling phase the Reynolds number reduces significantly and drops to 1025 at $t = 0.3321 \text{ s}$, 320 at $t = 0.369 \text{ s}$ and finally to the minimum magnitude of 213 at $t = 0.4428 \text{ s}$ (see figures 5.6c and 5.7a). The main feature of slow filling phase is the growth of vortices in the LV. In the lack of powerful inlet momentum into LV, the maximum velocity still is large. This

5. 2D Modeling of LV with Valve Leaflets

Figure 5.4: Flow patterns during rapid filling at (a) $t = 0.0185$ s (b) $t = 0.0369$ s (c) $t = 0.0738$ s. Streamlines colored by velocity (cm s^{-1}).

5. 2D Modeling of LV with Valve Leaflets

phenomenon shows the minimum energy dissipation in LV.

Figure 5.7*b* and *c* show the accelerated re-filling phase. The re-filling phase can be divided into accelerated and decelerated re-filling. During accelerated filling, the inflow velocity and diameter of inflow jet increase again, and the Re_D rises from 213 to 1094 and then 1992. As can be seen in 5.7*c*, the momentum produced by inflow and moving leaflets pushes the vortices toward apex and LV walls and vanish the small vortices. In fact, the small vortices are merged to other vortices. Figure 5.8 shows the decelerated re-filling and finally valve closure. The Re_D drops to 997 at $t=0.5289$ s and then to 398 at $t=0.5412$ s (see figure 5.8*a* and 5.8*b*). Same as decelerated rapid filling, the vortices increase in size and numbers. On the ventricular side, the momentum of vortices is directed towards valve closure while the leaflet motion pushes flow from the left atrium to the ventricle; they are visible from the streamlines moving with the leaflets for $Re_D = 997$ and 398. The reverse pressure gradient during these time steps helps to closure of MV leaflets (will be discussed in the next sections). Finally at $t = 0.5535$ s, the leaflets are fully closed along with higher momentum transferred to the two main vortices (see figure 5.8*c*). As discussed in previous chapter, the strength, number and size of vortices at the end of diastole is different for patients and normal cases. The powerful vortices, during closure of both valves, recirculate the blood flow and minimize the dissipation of energy. The energy characteristics will be discussed using work-energy equation in section 5.3.4.

5. 2D Modeling of LV with Valve Leaflets

Figure 5.5: Flow patterns during rapid filling at (a) $t = 0.1476 \text{ s}$ (b) $t = 0.1845 \text{ s}$ (c) peak of diastole; $t = 0.2214 \text{ s}$. Streamlines colored by velocity (cm s^{-1}).

5. 2D Modeling of LV with Valve Leaflets

Figure 5.6: Flow patterns during decelerated diastole and slow filling at (a) $t = 0.2952$ s (b) $t = 0.3321$ s (c) $t = 0.369$ s. Streamlines colored by velocity (cm s^{-1}).

5. 2D Modeling of LV with Valve Leaflets

Figure 5.7: Flow patterns during slow filling and accelerated re-filling at (a) $t = 0.4428$ s (b) $t = 0.4797$ s (c) $t = 0.5166$ s. Streamlines colored by velocity (cm s^{-1}).

5. 2D Modeling of LV with Valve Leaflets

Figure 5.8: Flow patterns during decelerated re-filling and end-diastole at (a) $t = 0.5289$ s (b) $t = 0.5412$ s (c) $t = 0.5535$ s. Streamlines colored by velocity (cm s^{-1}).

5. 2D Modeling of LV with Valve Leaflets

Figure 5.9 shows the average of net pressure (P_L) on mitral leaflets and pressure contours during diastole for Reynolds numbers 1355, 2842 and 398. The relative pressure is derived from the solution of Navier-Stokes equations. However, the magnitudes of actual pressure in LV are unknown and only by *in vivo* experiments can be obtained. The reference pressure to compute the absolute pressure is set 10 mmHg (1333 Pa) at the apex, to prevent negative magnitudes in the center of vortices. However, the reference pressure can be set to other magnitudes. The pressure gradients could be derived by solution of Navier-Stokes equation. Figure 5.9a and 5.9b shows the pressure contours in LV and net pressures along the mitral leaflets for the valve opening phase. While figure 5.9c is for the closure of mitral valve leaflets. The pressure drop ΔP_D in the figure 5.9 is defined as the difference between inlet section and the apex. The pressure loops are in an excellent consistency with vortices during acceleration and deceleration. Figure 5.10 shows vorticity(ω) distributions near the mitral valve and the shear distribution on both sides of the leaflets for the Reynolds numbers 1355, 2842 and 398. Further, the maximum vorticity (ω_{max}) for different Reynolds numbers is listed in the figure 5.10. The results show that the shear is very small on the both sides of leaflets.

5. 2D Modeling of LV with Valve Leaflets

Figure 5.9: Pressure contours during the mitral valve opening phase and net pressures (P_L) along the mitral leaflets for (a) rapid filling (b) peak of inlet velocity (c) during closing of mitral valve.

5. 2D Modeling of LV with Valve Leaflets

Figure 5.10: vorticity (ω) profiles on leaflets and LV for (a) rapid filling (b) peak of inlet velocity (c) during closing of mitral valve.

5. 2D Modeling of LV with Valve Leaflets

5.3.2 Systolic blood flow

After the closure of MV, the IVC phase is begun. In this study, the duration of IVC is 0.019 s, which is the time from the end diastole to the onset of systole. During IVC, the pressure in LV rises while the volume is constant. The vortices in figure 5.8c are merged and present in figure 5.11a which shows the end of IVC. At the first time steps of systole, when the AV leaflets begin to open, the vortices minimize the energy dissipation in LV. However the vortices sustain for a short time interval of 0.06 s. To capture the change of vortices during this time interval, the fine time steps are used. The results are presented in figures 5.11, 5.12 and 5.13.

In a time interval of 0.006 s the Re_O increases from zero to 100 (figure 5.11b) and 200 (figure 5.11c). During this time steps, the AV leaflets push open by high pressure due to LV contraction. In sinus of Valsalva side, because of AV outflow jet and upstream boundary condition some vortices start to form. In this study, prescribed time-dependent velocity $V_O(t)$ shown in figure 5.3 are used as boundary condition. Since the downstream condition would not have much effect on flow past the aortic valve, it was employed in this study.

Figure 5.12 shows the flow patterns for $Re_O = 400, 800$ and 1200 . At this stage, the wall contraction pushes the blood flow toward aortic root. The flow patterns show that the wall contraction does not affect the vortices and this phenomenon reflects an optimal transport of blood to the aorta during opening of AV. Figure 5.13a shows the the streamlines for $Re_O = 1750$. At this time step the AV leaflets push the blood flow toward sinus of Valsalva and the streamlines initiate on surface of leaflets. On the ventricle side, the streamlines out of jet center are

5. 2D Modeling of LV with Valve Leaflets

Figure 5.11: Flow patterns during IVC and initial systole at (a) $t = 0.572$ s (b) $t = 0.575$ s (c) $t = 0.578$ s. Streamlines colored by velocity (cm s^{-1}).

5. 2D Modeling of LV with Valve Leaflets

end on leaflets. Figure 5.13*b, c* show the flow patterns for $Re_O = 1825$ and 1890. The vortices in LV become smaller and travel toward aortic root and left side of LV. Figure 5.14 shows the flow patterns during high accelerated AV leaflets opening. The Re_O increases from 1890 to 3270 and finally in figure 5.14*c* the vortices in LV disappear by powerful wall contraction. The momentum produced by leaflets transfers to vortices in sinus of Valsalva.

In this study, the duration of fully opening AV leaflets is 0.110 s. The leaflets are considered fully open in figures 5.15*a* and 5.15*b*. At $t = 0.664$ s, the Re_O reaches the maximum magnitude of 3370. In figures 5.15*a* and *b*, all streamlines are straightened toward aortic root. The flow patterns during decelerated ejection and finally valve closure are depicted in figure 5.15*c* and figure 5.16. In this study, the duration of valve closure is 0.074 s. During this period, vortices are dominated in aortic root and streamlines end on leaflets in sinus of Valsalva side. Because of adverse pressure gradients associated with rapid flow deceleration, the valve is closed without aortic regurgitation (Bellhouse & Talbot, 1969; Hung *et al.*, 1978). Figure 5.16*c* shows the flow patterns at the end of systole. The velocity in LV is too low ($\leq 13 \text{ cm s}^{-1}$) and weak vortices are dominated in LV.

5. 2D Modeling of LV with Valve Leaflets

Figure 5.12: Flow patterns during initial time steps of systole at (a) $t = 0.581$ s (b) $t = 0.584$ s (c) $t = 0.588$ s. Streamlines colored by velocity (cm s^{-1}).

5. 2D Modeling of LV with Valve Leaflets

Figure 5.13: Flow patterns during accelerated ejection at (a) $t = 0.593$ s (b) $t = 0.603$ s (c) $t = 0.613$ s. Streamlines colored by velocity (cm s^{-1}).

5. 2D Modeling of LV with Valve Leaflets

Figure 5.14: Flow patterns during accelerated systole at (a) $t = 0.627$ s (b) $t = 0.638$ s (c) $t = 0.648$ s. Streamlines colored by velocity (cm s^{-1}).

5. 2D Modeling of LV with Valve Leaflets

Figure 5.15: Flow patterns at (a) peak of systole; $t = 0.664$ s (b) $t = 0.775$ s (c) $t = 0.812$ s. Streamlines colored by velocity ($cm s^{-1}$).

5. 2D Modeling of LV with Valve Leaflets

Figure 5.16: Flow patterns during AV closure at (a) $t = 0.830$ s (b) $t = 0.849$ s (c) $t = 0.886$ s. Streamlines colored by velocity (cm s^{-1}).

5. 2D Modeling of LV with Valve Leaflets

Figure 5.17 shows the pressure contours and average of net pressure (P_L) on the leaflets during opening of AV. Figure 5.17*a* shows the pressure contours at the onset of systole for $Re_O = 1825$. The pressure contours are dominated by vortices and the maximum pressure is close to the apex. Due to centrifugal effects, pressure contours are circular close to vortices. During the other opening time steps (figures 5.17*b* and 5.17*c*), the pressure increases in LV due to wall contraction. The pressure difference between apex and outlet of LV is indicated by ΔP_O and during accelerated ejection rises. The pressure differences will be discussed further in section 5.3.3 (page 77).

The pressure contours and net pressure on the leaflets during valve closure are depicted in figure 5.18. The pressure contours has a tongue shape and pressure decreases in the LV. The pressure on the aortic side is higher than LV side of leaflets and produce a net pressure toward LV. The ΔP_O during closure is negative due to low pressure in LV.

5. 2D Modeling of LV with Valve Leaflets

Figure 5.17: Pressure contours and average of net pressure (P_L) on the AV leaflets during opening at Reynolds number (a) 1825 (b) 2834 and (c) 3270. The dimension of the numbers is Pa.

5. 2D Modeling of LV with Valve Leaflets

Figure 5.18: Pressure contours and average of net pressure (P_L) on the AV leaflets during closing at Reynolds number (a) 2031 (b) 1738 and (c) 1534. The dimension of the numbers is Pa.

5. 2D Modeling of LV with Valve Leaflets

The shear stress and vorticity distribution are also studied and shown in figure 5.19. The results are presented for $Re_O = 3270, 3370$ and 1534 . The shear stress is plotted for left side of aortic root. It is divided into four parts and shown in the left side of figure 5.19*a, b* and *c*. The A and B indicate, respectively, the LV side and the aortic root side surfaces of leaflet. The C and D show the shear stress on the left side of sinus of Valsalva and aortic root, respectively. The study of shear stress is very important to assess the probability of damages due to jet flow during rapid ejection. However, the results show that the duration of jet flow is too short to cause any damage.

5. 2D Modeling of LV with Valve Leaflets

Figure 5.19: Vorticity and shear stress distribution of the flow past the AV for Reynolds Numbers (a) 3270 (b) 3370 (c) 1534.

5. 2D Modeling of LV with Valve Leaflets

5.3.3 Pressure difference

Figure 5.20 shows the pressure difference ($\Delta P(t^*)$) between basal and apical of LV. The $\Delta P(t^*)$ is derived from Navier-Stokes equations which is well correlated with the acceleration and deceleration of the diastolic flow. The difference between pressure (p_a) at the apex of LV and the instantaneous mean pressure (p_m) at the entrance of the left atrium and is indicated by $\Delta P_D(t^*) = \Delta p_a(t^*) - \Delta p_m(t^*)$. The difference between pressure at the apex and p_o at the outlet of the aortic flow is indicated by $\Delta P_O(t^*) = \Delta p_a(t^*) - \Delta p_o(t^*)$. The pressure drop is compared with $V_D(t^*)$ and $V_O(t^*)$ in the figure 5.20. Also shown in the figure is the pressure drop $\Delta P_S(t^*)$ between the apex and the aortic root. The difference between $\Delta P_O(t^*)$ and $\Delta P_S(t^*)$ is the pressure drop for blood flow past the aortic valve.

Figure 5.20: The pressure difference $\Delta P_D(t^*)$ during diastole and $\Delta P_O(t^*)$ and $\Delta P_S(t^*)$ during systole for 2D modeling with valve leaflets.

5. 2D Modeling of LV with Valve Leaflets

5.3.4 Energy characteristics of diastolic and systolic flow

The energy characteristics of blood flow in LV during diastole and systole can be further studied by using work-energy equation. An integral form of the work-energy equation can be expressed as (Hung & Schuessler, 1977; Brown & Hung, 1977; Macagno & Hung, 1967; Hung *et al.*, 2008):

$$\begin{aligned}
& \int \int \frac{\rho V^2}{2} \left(u \frac{\partial x}{\partial n} + v \frac{\partial y}{\partial n} + w \frac{\partial z}{\partial n} \right) dS + \int \int \int \left(\frac{\rho}{2} \frac{\partial V^2}{\partial t} \right) dx dy dz = \\
& - \int \int (p + \rho g y) \left(u \frac{\partial x}{\partial n} + v \frac{\partial y}{\partial n} + w \frac{\partial z}{\partial n} \right) dS \\
& + \int \int \mu \left(\frac{\partial u^2}{\partial x} \frac{\partial x}{\partial n} + \frac{\partial v^2}{\partial y} \frac{\partial y}{\partial n} + \frac{\partial w^2}{\partial z} \frac{\partial z}{\partial n} \right) dS \\
& + \int \int \mu \left(v \left(\frac{\partial v}{\partial x} + \frac{\partial u}{\partial y} \right) + w \left(\frac{\partial w}{\partial x} + \frac{\partial u}{\partial z} \right) \right) \frac{\partial x}{\partial n} dS \\
& + \int \int \mu \left(u \left(\frac{\partial v}{\partial x} + \frac{\partial u}{\partial y} \right) + w \left(\frac{\partial v}{\partial z} + \frac{\partial w}{\partial y} \right) \right) \frac{\partial y}{\partial n} dS \\
& + \int \int \mu \left(u \left(\frac{\partial w}{\partial x} + \frac{\partial u}{\partial z} \right) + v \left(\frac{\partial w}{\partial y} + \frac{\partial v}{\partial z} \right) \right) \frac{\partial z}{\partial n} dS \\
& - \int \int \int \mu \left(2 \left(\frac{\partial u}{\partial x} \right)^2 + 2 \left(\frac{\partial v}{\partial y} \right)^2 + 2 \left(\frac{\partial w}{\partial z} \right)^2 \right. \\
& \left. + \left(\frac{\partial u}{\partial y} + \frac{\partial v}{\partial x} \right)^2 + \left(\frac{\partial u}{\partial z} + \frac{\partial w}{\partial x} \right)^2 + \left(\frac{\partial w}{\partial y} + \frac{\partial v}{\partial z} \right)^2 \right) dx dy dz \quad (5.3)
\end{aligned}$$

In which ρ = the blood density, V = the velocity, (u, v, w) = the velocity components in the Cartesian coordinates, the gravitational acceleration, p = the pressure, μ = the dynamic viscosity of blood, and dS = the increment for surface integral along the wall, inlet and outlet sections. The first integral on the left hand side demonstrates the time rate of KE flux on the boundaries and the second term is the time rate of change of KE in the LV. The integrals on the right hand side represent, respectively, the rate of work done by pressure, the normal

5. 2D Modeling of LV with Valve Leaflets

viscous stresses, the rate of work done by shear stresses on x direction, the rate of work done by shear stresses on y direction, the rate of work done by shear stresses on z direction, and the rate of dissipation of energy (Hung *et al.*, 2008).

The equation can be also expressed as:

$$\begin{aligned}
 \int \int \left(p + \rho g y + \frac{\rho V^2}{2} \right) \left(u \frac{\partial x}{\partial n} + v \frac{\partial y}{\partial n} + w \frac{\partial z}{\partial n} \right) dS = \\
 \int \int \int \left(\frac{\rho}{2} \frac{\partial V^2}{\partial t} \right) dx dy dz + \int \int V \cdot \tau dS \\
 - \int \int \int \mu \left(2 \left(\frac{\partial u}{\partial x} \right)^2 + 2 \left(\frac{\partial v}{\partial y} \right)^2 + 2 \left(\frac{\partial w}{\partial z} \right)^2 \right. \\
 \left. + \left(\frac{\partial u}{\partial y} + \frac{\partial v}{\partial x} \right)^2 + \left(\frac{\partial u}{\partial z} + \frac{\partial w}{\partial x} \right)^2 + \left(\frac{\partial w}{\partial y} + \frac{\partial v}{\partial z} \right)^2 \right) dx dy dz \quad (5.4)
 \end{aligned}$$

The integral on the left hand side can be divided into three parts: the inlet and outlet sections and the wall of LV chamber. The results of each term can be checked by the balance of this equation for a few time steps. When the velocity is normal to the wall, the work done by viscous stresses will be zero. Thus the equation (5.4) in 2D form is expresses as:

$$\begin{aligned}
 \int_w \left(\frac{\rho V^2}{2} + p \right) V_N d\ell + \int \int \left(\frac{\rho}{2} \frac{\partial V^2}{\partial t} \right) dx dy \\
 - \int_m \left(\frac{\rho V_m^2}{2} + p_m \right) V_N d\ell - \int V \cdot \tau d\ell \\
 + \int \int \mu \left(2 \left(\frac{\partial u}{\partial x} \right)^2 + 2 \left(\frac{\partial v}{\partial y} \right)^2 \right. \\
 \left. + \left(\frac{\partial u}{\partial y} + \frac{\partial v}{\partial x} \right)^2 \right) dx dy = 0 \quad (5.5)
 \end{aligned}$$

5. 2D Modeling of LV with Valve Leaflets

For assessment the power of each term in equation (5.5), curves A to G are computed and plotted in figure 5.21. The data for curves are computed based on equations (5.6) to (5.11). Curve A is for kinetic energy flux of the wall dilation:

$$A = \int_W \left(\frac{\rho V^2}{2} \right) V_N d\ell \quad (5.6)$$

Since the actual pressure in the ventricle cannot be obtained from the computational flow simulation, the magnitude of pressure is based on a reference pressure of 10 mmHg at the apex of the ventricle. Curve B is the sum of Curve A and the work done by pressure with the wall dilation:

$$B = A + \int_W (p) V_N d\ell = \int_W \left(\frac{\rho V^2}{2} + p \right) V_N d\ell \quad (5.7)$$

Curve C is the sum of Curve B and the integral of time rate change of kinetic energy:

$$C = B + \int \int \left(\frac{\rho}{2} \frac{\partial V^2}{\partial t} \right) dx dy = \int_W \left(\frac{\rho V^2}{2} + p \right) V_N d\ell + \int \int \left(\frac{\rho}{2} \frac{\partial V^2}{\partial t} \right) dx dy \quad (5.8)$$

Curve D = Curve C minus the rate of work done by pressure at the mitral valve inlet:

$$D = C - \int_m (p_m) V_N d\ell = \int_W \left(\frac{\rho V^2}{2} + p \right) V_N d\ell + \int \int \left(\frac{\rho}{2} \frac{\partial V^2}{\partial t} \right) dx dy - \int_m (p_m) V_N d\ell \quad (5.9)$$

5. 2D Modeling of LV with Valve Leaflets

Figure 5.21: Rate of works during diastole in a 2D modeling of LV.

Curve E = Curve D minus the kinetic energy flux at the mitral valve inlet:

$$E = D - \int_m \left(\frac{\rho V_m^2}{2} \right) V_N d\ell = \int_W \left(\frac{\rho V^2}{2} + p \right) V_N d\ell + \int \int \left(\frac{\rho}{2} \frac{\partial V^2}{\partial t} \right) dx dy - \int_m \left(\frac{\rho V_m^2}{2} + p_m \right) V_N d\ell \quad (5.10)$$

Curve F = Curve E minus the rate of work done by shear on the wall:

$$F = E - \int V \cdot \tau d\ell = \int_W \left(\frac{\rho V^2}{2} + p \right) V_N d\ell + \int \int \left(\frac{\rho}{2} \frac{\partial V^2}{\partial t} \right) dx dy - \int_m \left(\frac{\rho V_m^2}{2} + p_m \right) V_N d\ell - \int V \cdot \tau d\ell \quad (5.11)$$

Curve G = Curve F + dissipation of energy; the result is left side of equation (5.5). The results for diastole are presented for curves A to G in figure 5.21. It should be notice that, because of low magnitudes for Curves A, E, F and G, they are presented in another diagram and can be seen on the right side of figure 5.21.

The same work has been done for figure 5.22, for rate of works during sys-

5. 2D Modeling of LV with Valve Leaflets

tole. Similarly, the above new plot will be employed for systolic flow from the LV chamber (wall, including the motion of mitral valve annulus and the closed mitral valve leaflet) to the aortic root. Because of the direction cosine, the above equation will be expressed as:

$$\begin{aligned}
 & - \int_W \left(\frac{\rho V^2}{2} + p \right) V_N d\ell + \int \int \left(\frac{\rho}{2} \frac{\partial V^2}{\partial t} \right) dx dy \\
 & + \int_m \left(\frac{\rho V_m^2}{2} + p_m \right) V_N d\ell - \int V \cdot \tau d\ell \\
 & + \int \int \mu \left(2 \left(\frac{\partial u}{\partial x} \right)^2 + 2 \left(\frac{\partial v}{\partial y} \right)^2 \right. \\
 & \left. + \left(\frac{\partial u}{\partial y} + \frac{\partial v}{\partial x} \right)^2 \right) dx dy = 0
 \end{aligned} \tag{5.12}$$

Based on the magnitude of these energy terms, the sign of the above equation is changed (moving the terms to the other side) for systolic flow and presented in equation (5.13). The new arrangement is to prevent confusion as each energy term is positive. The sign for each equation is to check the balance of the rate of energy with time.

$$\begin{aligned}
 & \int_W \left(\frac{\rho V^2}{2} + p \right) V_N d\ell - \int \int \left(\frac{\rho}{2} \frac{\partial V^2}{\partial t} \right) dx dy \\
 & - \int_m \left(\frac{\rho V_m^2}{2} + p_m \right) V_N d\ell + \int V \cdot \tau d\ell \\
 & - \int \int \mu \left(2 \left(\frac{\partial u}{\partial x} \right)^2 + 2 \left(\frac{\partial v}{\partial y} \right)^2 \right. \\
 & \left. + \left(\frac{\partial u}{\partial y} + \frac{\partial v}{\partial x} \right)^2 \right) dx dy = 0
 \end{aligned} \tag{5.13}$$

5. 2D Modeling of LV with Valve Leaflets

Figure 5.22: Rate of works during systole in a 2D modeling of LV.

In figure 5.22 the result for systole are presented for curves A to G based on equation (5.13) with the same procedure for diastole.

The total kinetic energy (KE) transfer from the wall to the blood during systole is

$$KE = \int \int_{WALL} \left(\frac{\rho V^2}{2} \right) V_N d\ell dt \quad (5.14)$$

It is equal to 0.1 mJ while kinetic energy delivered to the blood by LV dilation is 0.24 mJ. The total work done by pressure (WP) for the 2D analysis is

$$WP = \int \int_{WALL} (p) V_N d\ell dt \quad (5.15)$$

Table 5.1 compares the value of KE and WP for the wall motion and those across the mitral valve section for diastolic flow and across the aortic root during systole. In our knowledge, there is not any numerical study about the total energy transfer in LV and there are few non-invasive MRI measurement for obtaining the kinetic energy of LV. For verifying the magnitudes of rate of works, we have compared the magnitudes of kinetic energy for those which derived from non-invasive MRI

5. 2D Modeling of LV with Valve Leaflets

Table 5.1: Comparison between KE and WP during diastole and systole.

	Diastole		Systole	
	Wall	Mitral valve	Wall	Aortic valve
KE (mJ)	0.1	2.3	0.24	4.3
WP (mJ)	23	25	52	55

Figure 5.23: Rate of works for 3D modeling of LV during systole.

measurement by Carlsson *et al.* (2012) and our 3D CFD model (will be presented in Chapter 6). They divided the LV chamber to small cells and computed the kinetic energy for each of them. The sum of kinetic energy of small cells for each time step is the kinetic energy of blood flow in LV. They derived 6 mJ for maximum magnitude of kinetic energy for a 3D normal LV. With the same method, the maximum magnitudes for kinetic energy in present study and our 3D model are 1 mJ and 7 mJ, respectively. The formulation and related plots are presented in Chapter 6, section 6.3.4. The magnitude of 1 mJ for 2D modeling seems logical to be one seventh of 3D, because it is for one slice. In addition, the rates of works for our 3D model during systole are presented in figure 5.23.

5. 2D Modeling of LV with Valve Leaflets

Figure 5.24: Streamlines during rapid filling phase for LV without leaflets.

5.3.5 The effect of valve leaflets on fluid patterns

The diastolic flow is simulated by the prescribed LV dilatation obtained from the MRI data. The results discussed below are grouped into three phases: rapid filling, slow filling and late filling. Streamlines during rapid filling are shown in figure 5.24. At $Re_D = 1355$ a CW vortex is begun to form underneath of aortic valve. As flow rate is increased, the CW vortex became stronger and at the $Re_D = 2842$ a CCW below of mitral valve is formed. During slow filling phase the CW vortex is dominated in the LV and in the same time CCW extended itself in LV but it is weaker than CW vortex (see figure 5.25).

5. 2D Modeling of LV with Valve Leaflets

Figure 5.25: Streamline during slow filling, late filling and end-diastole for LV without leaflets.

With the start of atrial contraction during late filling the CCW vortex is moved toward apex at $Re_D = 1094$. Finally when two valves are closed CW and CCW vortices are dominated in LV and have an important role during ejection (figure 5.25f). As can be seen during systole approximately all streamlines are straight toward aortic root except for initial time steps of systole which CW and CCW vortices help to achieve more efficient ejection. Without the presence of aortic leaflets during systole, there are small vortices in the curvature of sinus of Valsalva. During iso-volumetric relaxation (IVR) two weak vortices are dominated in LV as shown in figure 5.26f.

The pressure difference between mitral and apex during diastole ($\Delta P_D(t^*)$) and

5. 2D Modeling of LV with Valve Leaflets

Figure 5.26: Streamline during systole for LV without leaflets.

pressure difference between apex and outlet ($\Delta P_O(t^*)$) are shown in figure 5.27. The maximum pressure difference occurs during systole.

Comparison between pressure difference between LV with and without valve leaflets shows the effect of leaflets on pressure difference. As shown in figure 5.28, the pressure differences are increased in presence of leaflets during diastole and systole. the peak of pressure drop for both of them occurs at the same time during diastole and systole.

Rate of works are computed for LV without valve leaflets, and the results are the same as rate of works in presence of leaflets (figures 5.21 and 5.22). The main reason is that the wall velocities, inlet and outlet velocities remains the same. Thus, leaflets have not affected the energy transfer during systole and diastole.

5. 2D Modeling of LV with Valve Leaflets

Figure 5.27: Pressure difference during diastole and systole for LV without valves.

Figure 5.28: Pressure difference during one LV cycle for with and without presence of leaflets.

5. 2D Modeling of LV with Valve Leaflets

5.4 Conclusion

Ventricular flow pattern are derived and analyzed for a 2D model of LV including the motion of leaflets. The momentum produced by valve leaflets transfers to the vortices at the tip of leaflets. The adverse pressure drop is observed during opening and closing of both AV and MV leaflets. The flow patterns are highly altered when the valve leaflets are included. However, the pressure differences between basal and apical is too close during diastole and systole with and without valve leaflets. Boundary layer is not developed during filling and ejection because of motion of leaflets. The magnitudes of shear stress on surface of leaflets are rather small to damage in a short time interval of accelerated flow during opening. The energy transfer analysis show that the most work done by LV is due to pressure produced by inflow, outflow, wall dilation and contraction. The rate of energy transfer by shear stress and energy dissipation is significantly lower than other terms in work-energy equation. The rate of works during diastole and systole is the same with and without valve leaflets. During filling and ejection the rate of energy transfers reflects an optimal filling and ejection.

Chapter 6

Three-Dimensional Modeling of LV for a Normal Case

6.1 Introduction

In this chapter, a normal LV dilation and contraction are modeled by using high resolution unstructured grids. The proximal left atrium and part of sinuses of Valsalva are also modeled with geometry based on MRI data to provide more realistic boundary conditions. The results are presented in different directions to capture vortices formation and movement in LV. The three dimensional flow patterns are qualitatively demonstrated first by spiral streamlines; they are further studied by using the Lagrange stream function on various longitudinal sections. This approach results in presenting the growth and decade of three-dimensional vortices. Different from all the previous studies, the pressure difference between basal and apical, and energy transfer into and from the LV are analyzed. The relationship between the time-dependent pressure drop and diastolic flow is dis-

6. Three-Dimensional Modeling of LV

Figure 6.1: Temporary LV volume ($vol(t^*)$) versus dimensionless time(t^*). $vol_{max}(t^*)$ is the volume at the end of diastole. $t^* = 0$ shows the beginning of diastole and $t^* = 1$ shows the end of systole.

cussed with the kinetic energy transfer associated with wall dilation, the rate of work done by pressure, and their balance with the rate change of kinetic energy. For the normal diastolic flow, the rate of work done by shear and the dissipation of energy are insignificant. Similar energy characteristics are also obtained for systolic blood flow in the ventricle.

6.2 Methodology

Methodology is the same as described in Chapter 3. In this section the boundary conditions related to this chapter are explained. A typical temporary volume versus time for 25 LV geometries for a normal case derived from MRI is shown in the figure 6.1 in which the cardiac period T is used to define the dimensionless time: $t^* = t/T$.

The reconstruction of LV geometry for CFD simulation was performed by using

6. Three-Dimensional Modeling of LV

Figure 6.2: (a) Reconstruction of geometries of LV, left atrium and ascending aorta at the end-diastolic and end-systolic phases. (b) Grid topology for the LV, left atrium and ascending aorta. Top right shows the left atrium and ascending aorta view, and the bottom right shows apex view.

a semi-automatic method with the high precision including the left atrium and the ascending aorta. Figure 6.2a shows the reconstruction geometry at the end-diastole and end-systole.

For each of segmented geometry, separate unstructured grids consisting of tetrahedral cells were generated. The final model contained 168,750 tetrahedral cells. The grid topology for the computational domain (LV, left atrium and ascending aorta) is shown in figure 6.2b.

Grid dependency study was done for five different grids and the number of grid cells were increased by the factor of 1.5 (50000, 75000, 112500, 168750 and 253125). The results showed that the global flow features (velocity component) did not change for the cell numbers above 168750. The changes of global flow features in the grid range of 112500 to 168750 were negligible, however, for more accurate solution 168750 cell numbers was chosen for 3D modeling. This grid resolution is more than two times of previous studies reported in the literature

6. Three-Dimensional Modeling of LV

Figure 6.3: Time variations of the inlet velocity ($V_D(t)$) and the outlet velocity ($V_O(t)$).

review chapter for more detailed flow simulation.

The velocity during diastole ($V_D(t)$) was applied at the inlet of LA along with no flow at the outlet of the aortic valve. During systole, the velocity at the outlet ($V_O(t)$) was applied with no flow at the inlet (see figure 6.3). The geometry for inlet (left atrium) and outlet (ascending aorta) can affect the results of simulation (Long *et al.*, 2003; Schenkel *et al.*, 2009).

6.3 Results and discussions

In this study, computational analysis was performed for a normal subject. The end-systolic volume and end diastolic volume were 48.8 ml and 162.5 ml, respectively, resulting in the stroke volume of 113.7 ml and ejection fraction of 70%. The period (T) of cardiac cycle was 0.88 s, wherein diastolic time was 0.55 s and systolic time 0.33 s (see figure 6.3). The simulation was initiated at the start of diastolic phase.

6. Three-Dimensional Modeling of LV

Figure 6.4: Four 2D longitudinal sections: P-P', Q-Q', R-R' and S-S'; and horizontal cross section H in two different views.

The flow patterns in 3D LV are very complex and the vortex motion could not be understood only with 3D spiral streamlines. Therefore, the flow patterns are further presented by 2D flow patterns as depicted in figure 6.4. Figure 6.4 shows four longitudinal sections: P-P', Q-Q', R-R' and S-S'; and horizontal cross section H. For some time steps additional cross section are used such as PQ-P'Q' which shows a cross section between PQ and P'Q' arches. The flow pattern on each longitudinal section is based on the Lagrange stream function which shows 2D-flow patterns (not the actual 3D streamlines).

6.3.1 3D diastolic flow

During the rapid filling, flow enters the LV with high inlet velocities relative to other phases. Figure 6.5 shows the flow patterns at $t = 0.04T$ (0.036 s). During 0.036 second, Reynolds number (Re_D) rises from zero to 901 and high accelerated

6. Three-Dimensional Modeling of LV

flow enters LV. The 3D spiral streamlines and streamlines on 2D longitudinal cross sections describe the movement and formation of vortices in LV due to wall dilation. Because of curve boundaries between the mitral valve and the aortic root the blood flow swirls toward the aorta and result in high velocities there. A small clockwise (CW) vortex begins as formation near the aorta and can be seen in P-P' and PQ-P'Q' cross sections. The flow patterns show a typical irrotational flow with boundary layer development from the wall.

The continued accelerated flow results in growth of CW vortex in Figure 6.6. The flow at this time can be indicated by an instantaneous $Re_D = 2329$. The 2D cross sections show the vortex on the P-P', Q-Q' and S-S' sections. This crescent-shaped vortex is longer than the curve QPS' but shorter than RQPS'R'. In present study the upward motion of closed aortic valve is modeled. During filling phase because of this motion some streamlines end on the outlet section. Figure 6.7 shows the flow patterns at the peak of inlet velocity at $t = 0.25T$. The CW crescent vortex becomes stronger at this time and the Re_D reaches to maximum magnitude of 3110. Further a counter clockwise (CCW) crescent vortex being developed on the other side of the mitral jet. The vortex is related to vanishing dilatation of the adjacent boundaries (see figure 6.7). It should be noticed that, as can be observed in 2D cross sections, the CW and CCW vortices are not connected at this time.

With the end of rapid filling at $t/T = 0.33$, the Re_D decreases to 1928 in figure 6.8. The 2D cross sections show the vortex in all direction (see P-P', PQ-P'Q', Q-Q', R-R' and S-S'). It becomes evident that the CW and CCW vortex are now connected and form a ring vortex during slow filling and late filling phases. The vortex length is longer than the arch of SP'Q'R' while the CW vortex exceeds

6. Three-Dimensional Modeling of LV

the arch of S'PQR. As the wall motion near the aortic root diminishes, the main flow moves toward the anterior wall. A decrease in strength of the CW crescent vortex on the Q-Q' section, and an increase on the R-R' section are noticeable in figure 6.8. The changes of vortices are due to stronger dilation of anterior wall and the associated momentum transfer.

Figure 6.5: Streamlines during early phase of diastole at $t/T = 0.04$.

6. Three-Dimensional Modeling of LV

Figure 6.6: Streamlines during diastolic acceleration at $t/T = 0.17$.

6. Three-Dimensional Modeling of LV

Figure 6.7: Streamlines at the peak of inlet velocity during diastole; $t/T = 0.25$.

6. Three-Dimensional Modeling of LV

Figure 6.8: Streamlines during decelerated early filling of diastole at $t/T = 0.33$.

6. Three-Dimensional Modeling of LV

The figure 6.9 shows the flow patterns during slow filling phase. The flow patterns shown in figure 6.9 portray rapidly changes of 3D flows in the LV chamber during the slow filling phase indicated by small Reynolds number, $Re_D = 23$. They are associated with insignificant wall motion of the LV chamber. By diminishing wall velocities, resulting in an immediate development of 3D circulatory flows with vortices. During slow filling, the core of ring vortex moves toward apex and occupies most part of the LV. The vortex is not only visible in all 2D cross sections in figure 6.9 but also in mid section between sections R-R' and S-S' (*i.e.* R'S'-RS), indicating that these vortices are connected to form a vortex ring. The main feature of slow filling phase is the growth of vortices in the LV. In the lack of powerful inlet momentum into LV, the maximum velocity still is large (*i.e.* 46 cm/s). This phenomenon shows the minimum energy dissipation in LV.

6. Three-Dimensional Modeling of LV

Figure 6.9: Streamlines during slow filling phase of diastole at $t/T = 0.46$.

6. Three-Dimensional Modeling of LV

The second inflow jet due to atrial contraction changes the flow pattern. The figure 6.10 shows the flow patterns at $t/T = 0.54$ for $Re_D = 1197$. Because of the strong vortex in the core region, the momentum of the main flow switches back to the posterior region, breaking up the ring vortex to form a large counter clockwise vortex in the core and anterior region. The main flow occupies the posterior LV chamber. The intensity of the vortex is increased by higher flow rate with Re_D increased from 23 to 1197 in 0.074 second. At $t/T = 0.58$, the Reynolds number reaches 2179, the velocity field and vortices become stronger (figure 6.11).

In the last time step of late filling phase, when the inlet velocity is close to zero and aortic valve is approximately closed, the main vortex however still grows and approximately affects all of stream lines from middle to apex. Figure 6.12 shows flow patterns at the end of diastole when the $Re_D = 0$. Also shown in figure 6.12 are the velocity vectors on cross sections H1 and H2. The positions of horizontal cross sections H1 and H2 are close to core of vortices. The flow deceleration leads to a growth of the clockwise vortex near the front part (below point R in figure 6.4). A rapid increase in the vortex zone and the splitting of a vortex into two associated with rapid flow deceleration are evident. They are somewhat similar to two dimensional vortices reported by Hung & Tsai (1996, 1997). Although the inflow vanishes, slight movements (local dilation or contraction) of the wall are visible in the 2D flow pattern. In the last time step of late filling phase, when the inlet velocity was close to zero and aortic valve was approximately closed, the main vortex however still grew and approximately affected all of stream lines from middle to apex.

6. Three-Dimensional Modeling of LV

Figure 6.10: Streamlines during late filling phase of diastole at $t/T = 0.54$.

6. Three-Dimensional Modeling of LV

Figure 6.11: Streamlines during late diastole at $t/T = 0.58$.

6. Three-Dimensional Modeling of LV

Figure 6.12: Streamlines and velocity vectors at the end of diastole; $t/T = 0.63$.

6. Three-Dimensional Modeling of LV

6.3.2 Systolic flow during ventricular contraction

With the start of wall contraction, the blood is being pushed into aorta because of LV wall contraction. The onset systolic flow patterns are depicted in figure 6.13 corresponds to $Re_O = 2041$. At the initial time steps of systole, the main vortex still remains in the LV and minimizes the energy dissipation. The numerical solutions of the Navier-Stokes equations show that blood near the LV chamber is moving toward the aortic root. The flow pattern indicates an effective transfer of momentum and energy from the LV contraction to the aortic flow, leaving the 3D vortices in the core region practically undisturbed or gradually dissipated. Figure 6.14 shows a continued increase in the aortic flow rate by $Re_O = 3687$ along with the decreases in vortices. At the peak of outlet jet velocity, the maximum velocity in the LV is 118 cm/s (figure 6.14). The converging streamlines to the aortic root from the wall reflect an optimal transfer of kinetic energy from the wall to the blood flow. The bundle of streamlines for the main flow encloses the 3D circulatory flows without much exchanges of momentum between them. Figure 6.14 demonstrates the aortic flow is primarily pushed by the contraction of the LV chamber as the velocities in the core region are small (refer to the blue areas). As the systolic flow continues, the momentum of fluids in the core region reduces and move towards the aortic root by the LV contraction. Figures 6.15 and 6.16 show the flow patterns during decelerated ejection. The Re_O decreases from 3687 to 3039 and 2221. The overall flow pattern in the LV chamber becomes simpler with the reduction in the LV volume. The most streamlines are straightened from wall to aortic root. Furthermore, there are some small and weak vortices in the LV as can be seen in 2D cross section flow patterns.

6. Three-Dimensional Modeling of LV

Figure 6.13: Streamlines during accelerated systole at $t/T = 0.66$.

6. Three-Dimensional Modeling of LV

Figure 6.14: Streamlines at the peak of outlet velocity during systole; $t/T = 0.75$.

6. Three-Dimensional Modeling of LV

Figure 6.15: Streamlines during systolic deceleration at $t/T = 0.83$.

6. Three-Dimensional Modeling of LV

Figure 6.16: Streamlines during decelerated ejection at $t/T = 0.91$.

6. Three-Dimensional Modeling of LV

Figure 6.17 shows the flow patterns at the last time steps of diastole ($t/T = 0.95$), when the outlet velocity is too low. During these time steps, the vortices increase in LV. The vortices are different from those produced by boundary layer separation; they are due to the diminished wall contraction.

Figure 6.17: Streamlines during systolic deceleration at $t/T = 0.95$.

6. Three-Dimensional Modeling of LV

Figure 6.18 shows the 3D flow in the LV at the end of systole. At this instant, the aortic valve is closed and LV chamber in the computational model is suddenly motionless. This deceleration of end systole creates 3D circulatory flows.

Figure 6.18: Streamlines at the end of systole; $t/T = 1$.

6. Three-Dimensional Modeling of LV

6.3.3 Pressure difference

Same as previous chapter, the time variation of pressure difference ($\Delta P_D(t^*)$) between basal and apical is presented. The pressure difference between apex and entrance of LV is indicated by $\Delta P_D(t^*)$ and shown in figure 6.19. Further, the pressure drop between apex and aortic root ($\Delta P_S(t^*)$) is depicted in figure 6.19. The pressure drop $\Delta P_D(t^*)$ is compared with the time variation of inflow velocity $V_D(t^*)$ and outflow velocity $V_O(t^*)$. The results show that the peak pressure drop is ahead of the peak velocity during diastole and systole due to inertial effects. The decreases in $V_D(t^*)$ and $V_O(t^*)$ indicate flow deceleration which is related to negative pressure drop. The maximum pressure difference occurs during systole.

The results for pressure differences are compared to our 2D modeling (with and without valves leaflets) and are depicted in figure 6.20. The results show that the similarity of pressure differences during diastole and systole in 3D and 2D modeling. This is related to similar accelerated and decelerated blood flow

Figure 6.19: The time variation of the inflow velocity $V(t^*)$ and the pressure drop $\Delta P(t^*)$ in the 3D LV during diastole and systole.

6. Three-Dimensional Modeling of LV

during diastole and systole in 2D and 3D modeling of LV.

Figure 6.20: Comparison of pressure differences between 3D modeling and 2D modeling with and without valves leaflets.

6. Three-Dimensional Modeling of LV

6.3.4 Energy characteristics of diastolic and systolic flow

The 3D integral form of the work-energy equation was presented in section 5.3.4, equations (5.3) and (5.4) (see pages 78 and 79). The integral on the left hand side of equation (5.4) can be divided into three parts: the inlet and outlet sections and the wall of LV chamber. Thus the equation (5.4) (see page 79) can be expressed as

$$\begin{aligned} & \int \int_W \left(\frac{\rho V^2}{2} \right) V_N dS + \int \int_W (p) V_N dS + \int \int \int \left(\frac{\rho}{2} \frac{\partial V^2}{\partial t} \right) dx dy dz \\ & - \int \int_m (p_m) V_m dS - \int \int_m \left(\frac{\rho V_m^2}{2} \right) V_m dS - \int \int_W V \cdot \tau dS \\ & + \int \int \int \mu \left(2 \left(\frac{\partial u}{\partial x} \right)^2 + 2 \left(\frac{\partial v}{\partial y} \right)^2 + 2 \left(\frac{\partial w}{\partial z} \right)^2 \right. \\ & \left. + \left(\frac{\partial u}{\partial y} + \frac{\partial v}{\partial x} \right)^2 + \left(\frac{\partial u}{\partial z} + \frac{\partial w}{\partial x} \right)^2 + \left(\frac{\partial w}{\partial y} + \frac{\partial v}{\partial z} \right)^2 \right) dx dy dz = 0 \quad (6.1) \end{aligned}$$

As described in Chapter 5 for 2D form of work-energy equation, the results arranged in A to G graphs. The same procedure is done for 3D form of work-energy (equation (6.1)). The first term in equation (6.1) is the rate of kinetic energy transfer (kinetic energy flux) to blood from ventricle dilatation. The calculated results are presented as curve A in figure 6.21a and also in 6.21b for amplification.

$$A = \int \int_W \left(\frac{\rho V^2}{2} \right) V_N dS \quad (6.2)$$

The second integral is the rate of work done by pressure of the wall motion. Since the actual pressure in the ventricle cannot be obtained from the computational flow simulation, the magnitude of pressure is based on a reference pressure of 10

6. Three-Dimensional Modeling of LV

mmHg at the apex of the ventricle. The sum of the first two integral in equation (6.1) is shown by curve B, making the gap between curves A and B the energy produced by wall pressures.

$$B = A + \int \int_W (p) V_N dS = \int \int_W \left(\frac{\rho V^2}{2} + p \right) V_N dS \quad (6.3)$$

The third term in the equation (6.1) is the rate of change of kinetic energy and is indicated by the gap between curves C and B. It is positive when C is higher than B, otherwise negative.

$$C = B + \int \int \int \left(\frac{\rho}{2} \frac{\partial V^2}{\partial t} \right) dx dy dz \quad (6.4)$$

The fourth and fifth integrals in equation (6.1) are respectively the rate of work done by pressure and the kinetic energy flux across the mitral valve section. The negative signs for these two terms are associated with energy balance of the equation. The rate of work done by pressure is shown by the gap between curves C and D. It is higher than the work done by pressure on the wall during systolic acceleration but smaller during most of the deceleration. The latter is related to adverse pressure gradient. The gap between curves D and E (see figure 6.21b) is the kinetic energy influx at the mitral valve section.

$$D = C - \int \int_m (p_m) V_N dS \quad (6.5)$$

$$E = D - \int \int_m \left(\frac{\rho V_m^2}{2} \right) V_N dS \quad (6.6)$$

6. Three-Dimensional Modeling of LV

The last two integrals in equation (6.1) are, respectively, the rate of work done by shear and the rate of dissipation of energy; they are shown in figure 6.21*b* as the gap between curves E and F, and that between curves F and G. The salient results indicate that the viscous effect is very small because the streamlines of diastolic flow are primarily produced by wall dilation. Curve G in figure 6.21*b* is the sum of all the energy terms of equation (6.1). It should be zero; the small numerical residues indicate a good balance of numerical results of equation (6.1). It also reflects accurate numerical solutions of the Navier-Stokes equations for the 3D model of diastolic flow in the left ventricle.

$$F = E - \int \int V \cdot \tau dS \quad (6.7)$$

$$G = F + \int \int \int \mu \left(2 \left(\frac{\partial u}{\partial x} \right)^2 + 2 \left(\frac{\partial v}{\partial y} \right)^2 + 2 \left(\frac{\partial w}{\partial z} \right)^2 + \left(\frac{\partial u}{\partial y} + \frac{\partial v}{\partial x} \right)^2 + \left(\frac{\partial u}{\partial z} + \frac{\partial w}{\partial x} \right)^2 + \left(\frac{\partial w}{\partial y} + \frac{\partial v}{\partial z} \right)^2 \right) dx dy dz = 0 \quad (6.8)$$

6. Three-Dimensional Modeling of LV

Figure 6.21: Rate of works during diastole for 3D modeling of LV.

Similarly, equation (6.1) can be applied for systolic flow when V_m and p_m are replaced by V_o and p_o . Because of the direction cosine, the equation (6.1) will be expressed as

$$\begin{aligned}
 & - \int \int_W \left(\frac{\rho V^2}{2} + p \right) V_N dS + \int \int \int \left(\frac{\rho}{2} \frac{\partial V^2}{\partial t} \right) dx dy dz \\
 & + \int \int_o \left(p_o + \frac{\rho V_o^2}{2} \right) V_N dS - \int \int V \cdot \tau dS \\
 & + \int \int \int \mu \left(2 \left(\frac{\partial u}{\partial x} \right)^2 + 2 \left(\frac{\partial v}{\partial y} \right)^2 + 2 \left(\frac{\partial w}{\partial z} \right)^2 \right. \\
 & \left. + \left(\frac{\partial u}{\partial y} + \frac{\partial v}{\partial x} \right)^2 + \left(\frac{\partial u}{\partial z} + \frac{\partial w}{\partial x} \right)^2 + \left(\frac{\partial w}{\partial y} + \frac{\partial v}{\partial z} \right)^2 \right) dx dy dz = 0 \quad (6.9)
 \end{aligned}$$

Based on the magnitude of these energy terms, the sign of the above equation is changed (moving the terms to the other side) for systolic flow and presented in equation (6.10). The new arrangement is to prevent confusion as each energy term is positive. The sign for each equation is to check the balance of the rate of

6. Three-Dimensional Modeling of LV

Figure 6.22: Rate of works during systole for 3D modeling of LV.

energy with time.

$$\begin{aligned}
 & \int \int_W \left(\frac{\rho V^2}{2} + p \right) V_N dS - \int \int \int \left(\frac{\rho}{2} \frac{\partial V^2}{\partial t} \right) dx dy dz \\
 & - \int \int_0 \left(p_o + \frac{\rho V_o^2}{2} \right) V_N dS + \int \int V \cdot \tau dS \\
 & - \int \int \int \mu \left(2 \left(\frac{\partial u}{\partial x} \right)^2 + 2 \left(\frac{\partial v}{\partial y} \right)^2 + 2 \left(\frac{\partial w}{\partial z} \right)^2 \right. \\
 & \left. + \left(\frac{\partial u}{\partial y} + \frac{\partial v}{\partial x} \right)^2 + \left(\frac{\partial u}{\partial z} + \frac{\partial w}{\partial x} \right)^2 + \left(\frac{\partial w}{\partial y} + \frac{\partial v}{\partial z} \right)^2 \right) dx dy dz = 0 \quad (6.10)
 \end{aligned}$$

Figure 6.22 shows the characteristics of energy terms in equation (6.10) for systolic flow in the left ventricle. Because of rapid contraction of the LV chamber, the kinetic energy flux transfer from the wall to the blood flow is much higher than that of the diastolic flow.

As mentioned in Chapter 5, there is not any numerical study about the total energy transfer in LV and there are few non-invasive MRI measurement for obtaining the kinetic energy of LV. For verifying the magnitudes of rate of works, we have compared the magnitudes of kinetic energy for those which derived from

6. Three-Dimensional Modeling of LV

non-invasive MRI measurement by Carlsson *et al.* (2012). They divided the LV chamber to small cells and computed the kinetic energy for each of them. In present study, to derive the kinetic energy for blood flow inside LV (KE_{LV}), the KE_{LV} expressed as

$$KE_{LV} = \sum_1^{168750} \left(\int \int \int \left(\frac{1}{2} \rho V^2 \right) dx dy dz \right) \quad (6.11)$$

In which 168750 is the number of cells in computational grid. The equation (6.11) is computed for each time step. The maximum magnitude for KE_{LV} in present study is 7 mJ and occurs during systole. Carlsson *et al.* (2012) derived maximum magnitude of 6 mJ for KE_{LV} during systole for a 3D normal LV by using non-invasive MRI measurement. Further, the results of rate of works in our 3D modeling could be compared with our 2D modeling as presented in Chapter 5. The rate of works for 2D modeling with valve leaflets during diastole and systole, respectively, can be seen in figures 5.21 (see page 81) and 5.22 (see page 83). Our 2D modeling results with and without valve leaflets show that the presence of valve leaflets could not affect the energy transfer results. Therefore, the presence of leaflets for 3D modeling could not change the rate of energy transfer.

6.4 Conclusion

In this study the, the flow patterns are presented by using 3D spiral streamlines and 2D cross sections. During rapid filling, accelerated flow forms a CW vortex at the junction of mitral and aortic orifices. At the peak of inlet velocity, a CCW vortex begin to form at posterior side of mitral valve while the CW vortex still

6. Three-Dimensional Modeling of LV

grows. At the end of diastole these two vortices become connected and forming a ring vortex in LV. During slow filling phase and end of diastole, because of diminishing main entrance flow, the CCW vortex occupies most part of the LV. During re-filling phase, the accelerated flow pushes the core of main CCW vortex toward apex. At the first time steps of systole, the main CCW vortex remains and minimize the energy dissipation. With the decreasing of LV volume due to wall contraction, the systolic flow can be characterized by a more direct momentum transport from the wall to the aortic root with vanishing vortices. However, during decelerated ejection, some weak vortices are generated in LV. The pressure drop during diastole and systole results show that the peak of pressure drop is ahead of peak of velocity due to inertial effects. Similar to 2D normal case in Chapter 5, the rate of works reflect an optimal filling and ejection of LV.

Chapter 7

Three-dimensional Modeling of Blood flow in a Heart Failure Case Before and After Surgery

7.1 Introduction

In this chapter, a patient before and after surgery is studied. Combined CFD/MRI for patients before and after surgery with different heart diseases such as myocardial infarction could facilitate better understanding of flow pattern and research into ways to optimize and refine surgical treatment approaches in the future.

As mentioned in Chapter 1, the main reason of heart failure is myocardial infarction. Surgical ventricular restoration (SVR) and Coronary artery bypass graft (CABG) are two options for surgeons. SVR is a surgical procedure that restores enlarged LV for patients with heart failure. In this procedure the shape and size of LV changes to provide efficient blood delivery for body. The CABG performs

7. Heart failure Case Before and After Surgery

on the patients with problem of supply sufficient blood flow into coronary arteries. The coronary arteries supply the blood flow to myocardial muscles. The lack of blood for muscles will cause the dead muscles in myocardial and result in low ejection fraction.

SVR is designed to restore the left ventricle to its normal shape and size in patients with akinetic segments of the heart (Athanasuleas *et al.*, 2004). It is performed usually with coronary artery bypass grafting (CABG)(Prucz *et al.*, 2008). In present study, CABG is performed with SVR (CABG+SVR). The improvement of left ventricular ejection fraction (EF) has been well documented (Athanasuleas *et al.*, 2004; Mickleborough *et al.*, 2004; Sartipy *et al.*, 2005, 2006; Zhong *et al.*, 2011). However, mechanism and basis of this improvement remains uncompleted understood (Bolooki *et al.*, 2003). Doenst *et al.* (2009) have shown that the combined CFD/MRI is an effective tools to asses the fluid dynamics of blood flow before and after surgery. They investigated the changes of blood flow patterns in a case before and after SVR.

Generally the heart surgery changes the ventricle shape and causes the change of blood flow characteristics. The objective of current chapter is to assess the kinematic, dynamics and energy characteristics of blood flow in a LV before and after SVR+CABG. The methodology for modeling is exactly the same as previous normal case. Thus only changes in boundary conditions such as inlet and outlet velocities are presented.

7. Heart failure Case Before and After Surgery

7.2 Patient

In this section, Biodata which derived directly from MRI are described. These data are the main data which clinicians diagnose about the conditions (ability or inability) of LV. The MRI data obtained from a patient before surgery and 4 months after surgery. The table 7.1 shows Biodata for a 48 year old man, before and 4 months after CABG+SVR. As can be seen in the table 7.1, the EDV and ESV before surgery are 244.7 ml and 160.8 ml, respectively, result in the SV of 83.9 ml and EF of 34%. The EDV and ESV after surgery are 219 ml and 114 ml, respectively, which give the stroke volume of 105 ml and EF of 48%.

Table 7.1: Biodata before and after CABG+SVR (Male, 48 years old).

	Blood pressure	Heart rates (beats/min)	EDV (ml)	ESV (ml)	EF (%)
Pre-surgery	107/69	51	244.7	160.8	34
Post-surgery	106/67	72	219	114	48

7.3 Results

The Changes of LV volumes before and after surgery versus dimensionless time are depicted in figure 7.1. The cardiac cycle periods before and after surgery are 1.15 s and 0.80 s, respectively. The simulation is initiated at the begin of diastolic phase.

7. Heart failure Case Before and After Surgery

Figure 7.1: Temporary volume versus dimensionless time for 25 before and after surgery LV geometries which derived from MRI; $t^* = 0$ and $t^* = 1$ show the first-diastole and end-systole, respectively.

7.3.1 Pre-surgery results

The mean velocities at inlet ($V_D(t)$) and outlet ($V_O(t)$) of the computational model are depicted in figure 7.2. They are uniformly applied at the inlet during diastolic flow and at the outlet for systolic flow. In addition, the Reynolds number ($Re(t)$) versus time is shown in figure 7.2 during diastole ($Re_D(t)$) and systole ($Re_O(t)$).

As mentioned, the methodology is same as the normal 3D case in previous chapter. With the same procedure the geometry is reconstructed and meshes are produced. The geometry meshes at the end of diastole and at the end of systole are shown in figure 7.3.

7. Heart failure Case Before and After Surgery

Figure 7.2: Temporary velocity versus time for pre-surgery LV.

Figure 7.3: Pre-surgery end-diastole and end-systole geometries.

7. Heart failure Case Before and After Surgery

7.3.1.1 Flow patterns during diastole

The 3D flow patterns (spiral streamlines) associated with 2D flow patterns on longitudinal cross sections are presented for some selected time steps. The longitudinal cross sections (P-P', Q-Q', R-R' and S-S') are the same as those presented in Chapter 6 for 3D normal case (see figure 6.4, page 94).

The inlet velocity profile in figure 7.2 shows that in the case before surgery, there is not late filling phase. The diastolic phase for the before surgery LV can be divided into: accelerated filling and decelerated filling phase, because there is only one velocity peak during diastole.

The simulation is begun at the first of diastolic phase. Figure 7.4 shows the streamlines at the initial time steps of diastole for $Re_D = 361$. The location of maximum velocity (9 cm s^{-1}) in the LV, during initial time steps of diastole, is around the junction between LA and aortic root. The reason is that the change of the LV volume, below of the aortic valve is more than other parts of LV.

7. Heart failure Case Before and After Surgery

Figure 7.4: Flow patterns during initial time steps of diastole at $t = 0.048$ s for pre-surgery LV.

7. Heart failure Case Before and After Surgery

As wall dilatation is continued, more blood flow crosses through the inlet section and enters the LV. The flow swirls toward the aortic root because of more wall movement. This phenomenon causes the formation of a CW vortex at the underneath of the aortic valve. Figure 7.5 shows the streamlines for $Re_D = 799$. The CW vortex can be watched in the P-P' cross section and 3D view of LV in figure 7.5.

Figure 7.5: Flow patterns during accelerated diastole at $t = 0.192$ s for pre-surgery LV.

7. Heart failure Case Before and After Surgery

With the increasing of inlet velocity the CW vortex becomes stronger. As can be seen in figure 7.6, the length of the vortex is increased and in the Q-Q' plan and S-S' plan the vortex can be observed.

Figure 7.6: Flow patterns during accelerated diastole at $t = 0.337$ s for pre-surgery LV.

7. Heart failure Case Before and After Surgery

At the peak of inlet velocity, a crescent vortex is completely formed. The crescent vortex is powerful in the aortic side and weak in the other sides. The 2D cross sections in figure 7.7 show the diameter of the ring vortex on all sides. At the peak of inlet velocity, Reynolds number reaches the maximum magnitude of 1146 during diastole.

Figure 7.7: Flow patterns at the peak of diastole for pre-surgery LV.

7. Heart failure Case Before and After Surgery

As mentioned, for the case before surgery there are not late filling phase. After the peak of inlet velocity, the momentum of inlet flow is decreased. With the decrease of inlet momentum, the crescent vortex increases in size specially on the posterior side of mitral orifice. However, the core of crescent vortex does not move toward apex. Figure 7.8 shows the flow patterns for $Re_D = 784$.

Figure 7.8: Flow patterns during decelerated diastole at $t = 0.626$ s for pre-surgery LV.

7. Heart failure Case Before and After Surgery

Figure 7.9 shows the fluid pattern during isovolumetric contraction (IVC), when two valve orifices are closed. As can be seen, the core of crescent vortex is close to the basal and the vortex is dominated in the LV chamber. The maximum velocity in the LV is 19 cm s^{-1} and the pair of crescent vortex is too weak compared to a normal case.

Figure 7.9: Flow patterns during IVC at $t = 0.722 \text{ s}$ for pre-surgery LV.

7. Heart failure Case Before and After Surgery

7.3.1.2 Flow patterns during systole

With the start of wall contraction, fluid flow is pushed into the aortic root. Figure 7.10 shows the streamlines at the onset of systole for $Re_O = 350$. The weak crescent vortex during IVC after few time steps is disappearing and does not help the LV for efficient ejection.

Figure 7.10: Flow patterns at the initial time steps of systole at $t = 0.730$ s for pre-surgery LV.

7. Heart failure Case Before and After Surgery

After 0.041 s, Reynolds number is increased from 350 to 1170. The flow patterns at $t = 0.771$ s are shown in figure 7.11. During systole majority of streamlines are straight forward toward aortic root.

Figure 7.11: Flow patterns at the initial time steps of systole at $t = 0.771$ s for pre-surgery LV.

7. Heart failure Case Before and After Surgery

Figure 7.12 shows the flow patterns at the peak of outlet velocity. The Reynolds number reaches to 2358 which is the maximum magnitude of Re_O during systole. During ejection, because of curvature in the sinus of valsalva, there is a small vortex in the aortic root (see P-P' plan).

Figure 7.12: Flow patterns at the peak of systole at $t = 0.915$ s for pre-surgery LV.

7. Heart failure Case Before and After Surgery

Figure 7.13 shows the flow patterns for $Re_0=0$ at the end of systole. As can be observed, there is two vortices in the aortic root and some weak vortices in LV.

Figure 7.13: Flow patterns at the end of systole at $t = 1.155$ s for pre-surgery LV.

7. Heart failure Case Before and After Surgery

7.3.1.3 Pressure difference

The difference between pressure (p_a) at the apex of LV and the instantaneous mean pressure (p_m) at the entrance of the left atrium and is indicated by $\Delta P_D(t^*) = \Delta p_a(t^*) - \Delta p_m(t^*)$. The difference between pressure at the apex and pressure at the aortic root is indicated by $\Delta P_S(t^*)$. The numerical solution of the Navier-Stokes equations yields $\Delta P(t^*)$ which is well correlated with the acceleration and deceleration of the diastolic flow. The pressure drop is compared with $V_D(t^*)$ and $V_O(t^*)$ in figure 7.14.

Figure 7.14: The pressure difference $\Delta P_D(t^*)$ during diastole and $\Delta P_S(t^*)$ during systole for pre-surgery LV.

7. Heart failure Case Before and After Surgery

7.3.1.4 Energy characteristics

The results of work-energy equation are computed in the same way which described in Chapter 6 for the 3D normal case. Figure 7.15 and figure 7.16 show the results during diastole and systole for before surgery, respectively. The results are also shown in figure 7.15*b* and figure 7.16*b* for amplification of A, E, F and G curves.

Figure 7.15: Rate of works during diastole for pre-surgery LV.

Figure 7.16: Rate of works during systole for pre-surgery LV.

7. Heart failure Case Before and After Surgery

7.3.2 Post-surgery results

The velocities at the inlet and outlet of LV are shown in figure 7.17. The duration of diastolic and systolic phases after surgery are 0.468 s and 0.335 s, respectively. Further, the changes of Reynolds number versus time have been depicted in figure 7.17.

The reconstructed LV geometry from MRI data with geometry grid at the end of diastole and end of systole are shown in figure 7.18. For better understanding of surgery effects on the shape of LV, the figures 7.18 and 7.3 can be compared. The comparison show that the shape of LV after SVR+CABG is more spherical than elongated and there are sharp edges near the apex for after surgery LV.

7. Heart failure Case Before and After Surgery

Figure 7.17: Temporary velocity versus time for post-surgery LV.

Figure 7.18: Post-surgery end-diastole and end-systole geometries.

7. Heart failure Case Before and After Surgery

7.3.2.1 Flow patterns during diastole

The flow patterns for post-surgery case has been presented in a similar way as pre-surgery case for better understanding of surgery effects. Simulation is begun with the start of diastole. Figure 7.19 shows the flow patterns at the initial time steps of diastole for $Re_D = 357$.

Figure 7.19: Flow patterns during initial time steps of diastole at $t = 0.033$ s for post-surgery LV.

7. Heart failure Case Before and After Surgery

At $t = 0.1$ s, the Reynolds number increases to 976 (see figure 7.20). At this time, a small CW vortex forms close to the junction of aortic root and LA. The small CW vortex can be observed in P-P' cross section.

Figure 7.20: Flow patterns during accelerated diastole at $t = 0.100$ s for post-surgery LV.

7. Heart failure Case Before and After Surgery

With reaching the Reynolds number to 1626, the small CW vortex becomes larger. Figure 7.21 shows that the vortex is extended between P side of P-P' cross section and S' side of S-S' cross section.

Figure 7.21: Flow patterns during accelerated filling at $t = 0.200$ s for post-surgery LV.

7. Heart failure Case Before and After Surgery

At $t = 0.301$ s, the inflow reaches the maximum velocity. The Re_D increases to 1833 which is the maximum magnitude of Re_D during diastole. The vortex becomes larger and can be observed in P side of P-P' cross section, S' side of S-S' and Q side of Q-Q' cross section (see figure 7.22).

Figure 7.22: Flow patterns at the peak of diastole for post-surgery LV.

7. Heart failure Case Before and After Surgery

During deceleration, the momentum of inflow is decreased and the vortices become larger and weaker. At $t = 0.401$ s, a crescent vortex forms in LV. The Re_D decreases to 1312 and the aortic side of crescent vortex travels slowly toward apex. The crescent vortex can be seen on the all sides of four 2D cross sections (see figure 7.23). However, the diameter and strength of vortex are larger and higher on the aortic side than other sides.

Figure 7.23: Flow patterns during decelerated diastole at $t = 0.401$ s for post-surgery LV.

7. Heart failure Case Before and After Surgery

Figure 7.24 shows the flow patterns for $Re_D = 965$. The aortic side of crescent vortex becomes larger. The evidences of this phenomenon are Q side of Q-Q' cross section and S' side of S-S' cross section.

Figure 7.24: Flow patterns during last time steps of diastole at $t = 0.435$ s for post-surgery LV.

7. Heart failure Case Before and After Surgery

The flow patterns during IVC are shown in figure 7.25. The crescent vortex is dominated on the LV and recirculating the blood flow. The strength of crescent vortex during IVC is a criteria for assessing the momentum of blood flow for efficient ejection. The difference of the flow patterns during IVC will be further discussed in the next sections.

Figure 7.25: Flow patterns during IVC at $t = 0.468$ s for post-surgery LV.

7. Heart failure Case Before and After Surgery

7.3.2.2 Flow patterns during systole

During the initial time steps of systolic phase, the flow patterns are complicated because of vortices during IVC. The vortices in normal cases help to ensure efficient ejection of blood flow. The flow patterns during the initial time steps of systole are shown in four figures. Figure 7.26 shows the flow patterns at $t = 0.476$ s and $Re_O = 370$.

Figure 7.26: Flow patterns at the first time steps of systole at $t = 0.476$ s for post-surgery LV.

7. Heart failure Case Before and After Surgery

At $t = 0.487$ s and $Re_O = 630$, the crescent vortex becomes smaller but it is still dominated in LV. The vortex recirculating the blood flow in CW direction (see figure 7.27).

Figures 7.28 and 7.29 show the flow patterns for $Re_O = 995$ and $Re_O = 1303$,

Figure 7.27: Flow patterns at the first time steps of systole at $t = 0.487$ s for post-surgery LV.

respectively. After $t = 0.502$ s, the vortex is disappeared completely by wall contraction.

7. Heart failure Case Before and After Surgery

Figure 7.28: Flow patterns at the first time steps of systole at $t = 0.495$ s for post-surgery LV.

Figure 7.29: Flow patterns at the first time steps of systole at $t = 0.502$ s for post-surgery LV.

7. Heart failure Case Before and After Surgery

As wall contraction continued, at $t = 0.636$ s the Re_O reaches the maximum magnitude of 3076. The streamlines are directly toward aortic root. However, in the lack of valve leaflets, there is a small vortex in the sinus of Valsalva part due to wall curvature during ejection. The flow patterns at the peak of outlet velocity are shown in the figure 7.30.

Figure 7.30: Flow patterns at the peak of systole at $t = 0.636$ s for post-surgery LV.

7. Heart failure Case Before and After Surgery

At the end of systole the outlet velocity is close to zero. In the lack of wall contraction and momentum, there are some weak vortices in LV and aortic root (figure 7.31). After this time step the aortic valve will be closed.

Figure 7.31: Flow patterns at the end of systole at $t = 0.803$ s for post-surgery LV.

7. Heart failure Case Before and After Surgery

7.3.2.3 Pressure difference

Figure 7.32 show the pressure difference for after surgery. Further, the pressure drop is compared with $V_D(t^*)$ and $V_O(t^*)$. The difference between pressure (p_a) at the apex of LV and the instantaneous mean pressure (p_m) at the entrance of the left atrium and is indicated by $\Delta P_D(t^*)$. The difference between pressure at the apex and pressure at the aortic root is indicated by $\Delta P_S(t^*)$.

Figure 7.32: The pressure difference $\Delta P_D(t^*)$ during diastole and $\Delta P_S(t^*)$ during systole for after surgery.

7.3.2.4 Energy characteristics

Figure 7.33 and figure 7.34 show the results of work-energy equation during diastole and systole for after surgery, respectively. The results are also shown in

7. Heart failure Case Before and After Surgery

figure 7.33b and figure 7.34b for amplification of curves A, E, F and G.

Figure 7.33: Rate of works during diastole for after surgery LV.

Figure 7.34: Rate of works during systole for after surgery LV.

7.4 Discussion

In this section, the results of CFD modeling for before and after surgery are compared and discussed. The medical data such as EDV, ESV and LVEF are shown in table 7.1 for before and after surgery LV. In addition, comparison has

7. Heart failure Case Before and After Surgery

been performed between pre-surgery and post-surgery LV with our results from 3D modeling of a normal subject in chapter 6, as presented in table 7.2. Duration of cardiac cycle (i.e., 0.8 second) after surgery is shorter than before surgery (i.e., 1.15 second). As shown in figure 7.2 and figure 7.17, the inlet and outlet velocities and Reynolds number for pre-surgery LV are lower than post-surgery LV. The Maximum Re_O for pre-surgery, post-surgery and normal case are 2358, 3076 and 3700, respectively.

In the post-surgery LV, vortices are more powerful than the pre-surgery LV. However, they are not powerful as well as normal case which discussed in Chapter 6. During diastole and systole, the blood velocity in the field for after surgery LV is higher than before surgery LV. As shown in table 7.1, the volume is decreased after surgery. The decreased volume associated with more powerful vortices in after surgery LV help to better recirculation of blood flow in LV during diastole. The crescent vortex during diastole does not travel toward apex in pre-surgery LV. Although, in post surgery LV the crescent vortex does not move significantly toward apex, it is however more than before surgery. The shape of LV is changed after surgery from elongated shape to spherical shape. The surgeons should go toward to keep the elongated shape of LV because in elongated shape the crescent vortex could asymmetrically grows and travels toward the apex. However, the elongated shape must be associated with powerful inlet momentum into LV.

At the first time steps of systole, the vortices disappear in pre-surgery LV, but in after surgery LV they sustain longer. The direction of the main vortex during initial time steps of systole in both before and after surgery LV does not help to better ejection of LV. The vortices could be compared with the normal case in the Chapter 6.

7. Heart failure Case Before and After Surgery

The pressure difference between apex and apical are compared for before and after surgery in figure 7.35. The results show that the pressure difference in after surgery case is increased and provides better LV filling and ejection. Also shown in table 7.2, the maximum magnitudes of ΔP_D and ΔP_S for pre-surgery, post-surgery and normal LV. The results show that the maximum pressure differences between apex and basal during diastole and systole for normal LV are higher than both before and after surgery LV.

In our recent study (Khalafvand *et al.*, 2012b) as discussed in Chapter 4, we

Figure 7.35: Comparison between pressure difference before and after surgery.

have shown that the pressure differences for normal subjects are higher than heart failure patients. The 2D simulation was performed for three normal and three abnormal subjects. Further, the results have shown that numbers, size and

7. Heart failure Case Before and After Surgery

location of vortices are different for normal and heart failure cases. The maximum magnitudes of pressure differences during diastole and systole ($Max(\Delta P_D)$ and $Max(\Delta P_S)$) could reflect the efficiency of LV as a cardiac pump. The higher magnitudes $Max(\Delta P_D)$ demonstrate higher wall dilation and thus higher inlet flow rate into LV. The higher magnitudes of $Max(\Delta P_S)$ demonstrate higher wall contraction and thus higher outlet flow rate from LV into aortic root.

The effects of all clinical and fluid dynamics parameters appear in energy transfer

Table 7.2: Comparison between pre-surgery and post-surgery LV with normal LV.

Variables	Pre-surgery	Post-surgery	Normal
$T(sec)$	1.15	0.8	0.88
$t_D(sec)$	0.72	0.47	0.55
$t_S(sec)$	0.43	0.33	0.33
$Max(Re_O)$	2358	3076	3700
$Max(\Delta P_D)(Pa)$	180	234	282
$Max(\Delta P_S)(Pa)$	400	516	620

into and out of LV. The rate of works for pre-surgery and post-surgery LV during diastole and systole are shown in figures 7.15, 7.16, 7.33 and 7.34, respectively. The results suggest that after surgery, the rate of works done by pressure and kinetic energy which produced by walls, inlet and outlet flows are increased. The rate of work done by shear stress and energy dissipation for after surgery LV are decreased. However, the rate of works done by shear stress and energy dissipation in both the pre-surgery and post-surgery LV are relatively low.

The current modeling ignores the effects of the mitral and aortic valves to the fluid and the valves are presented as an orifice only. Due to the clinical constraint and computational complexity, there is not any attempt to model valves leaflets.

7. Heart failure Case Before and After Surgery

As mentioned in Chapter 5, the leaflets could not affect the rate of energy transfer in LV. However, they have important effects on the formation of vortices.

7.5 Conclusion

It can be concluded that during systolic and diastolic phase in post-surgery LV, velocity magnitudes in LV chamber, pressure difference between basal and apical and net energy transfer are increased. The ejection fractions before and after surgery were 34% and 48%, respectively. The duration of one cycle decreased with 30% after surgery. In post-surgery LV vortices were stronger than pre-surgery but weaker than a typical normal case. However, the direction of vortices at the first time steps of systole in post-surgery LV does not help in efficient ejection of LV. The main reason of this phenomenon is that the crescent vortex does not move deep toward the apex. The maximum Pressure differences between basal and apical during diastole in pre-surgery and post-surgery LV, respectively, were 180 Pa and 234 Pa, while during systole were 400 Pa and 516 Pa. The maximum pressure differences might be an index beside of EF for assessment of LV. The work done by shear stress and energy dissipation are decreased in post-surgery LV. Modeling of the LV for the cases before and after surgery with different LV conditions and valve diseases may provide useful clinical applications.

Chapter 8

Conclusions and Recommendations for Future Work

In this chapter, the key conclusions which are derived from the present research work are highlighted. A brief overview of potential research work to be undertaken in the future is also presented to motivate further research on LV modeling.

8.1 Concluding remarks

The present study contained both the 2D and 3D modeling of LV for different cases. The study began to model normal and heart failure cases by using 2D modeling of LV (see Chapter 4). The results of the 2D modeling for three normal and three heart failure cases without valve leaflets show that the number of vortices and pressure difference between basal and apical for normal cases is

8. Conclusions and Future Works

higher than abnormal cases. In the normal subjects, the mean EDV and ESV are 122.3 ± 16.04 ml (mean $\pm$ SD) and 37.3 ± 6.6 ml, respectively. While for the patients with MI, the mean EDV and ESV are increased to 276.7 ± 45.7 ml and 220.3 ± 45.6 ml. The EF is $69 \pm 6\%$ for normal cases and $20.33 \pm 10\%$ for patient cases. Ventricular flow patterns in the MI patients are significantly altered. At end-diastole, three main vortices and several small vortices are observed in normal subjects. Because of lower momentum in an enlarged ventricle, only two weak vortices are seen in the MI patients. The maximum pressure differences between basal and apical are substantially larger in normal subjects than the MI patients (4.34 ± 0.89 mmHg vs. 1.77 ± 0.58 mmHg). The time variation of pressure drop is well correlated with the acceleration and deceleration of diastolic and systolic blood flows in the LV. The results of this investigation appeared promising to provide useful information with proper classification on intra-LV flow velocity and pressure drops with diverse heart diseases.

The kinematic, dynamic and energy characteristics of blood flow were discussed in a complete 2D modeling of LV for a normal case including the motion of valves leaflets (see Chapter 5). The vortices associated with the movements of valve leaflets, and flow acceleration and deceleration are discussed along with pressure drops, pressures on the leaflets and shear stresses. The cardiac pumping characteristics are further studied by the rate of energy transfer from the ventricle to the blood flow, the rate of work done by pressure and shearing stresses, the rate change of kinetic energy and dissipation of energy. All these kinematic, dynamic and energy characteristics reflect an optimal contraction and dilation for normal cardiac function. The results show that the presence of valves leaflets could not change the energy characteristics of blood flow in LV.

8. Conclusions and Future Works

The 3D modeling of LV for a normal case including proximal LA and ascending aorta is performed in Chapter 6. The complexity of transient spiral flow produced by the LV dilation is difficult to analyze and graphically presented. The 3D flow processes are analyzed by calculating the Lagrange stream function on a sequence of longitudinal planes. These 2D flow patterns demonstrate flow produced by ventricle motion and the formation and decade of 3D vortices. The work-energy equation is well used to quantify the energy transfer from the contraction and dilation of the ventricle to the pulsating flow processes. The net rate of energy transfer from the wall motion to the blood flow in the ventricle is primarily contributed by the rate of kinetic energy. For the normal subject, the work done by shear stresses and the dissipation of energy are rather small. The results of energy characteristics of diastolic flow are compared with those of a two-dimensional model with opening and closing of the mitral leaflets obtained from MRI data.

Finally, the 3D modeling of LV for a case before and after surgery is described and discussed (see Chapter 7). A heart failure case before and after CABG+SVR is studied. The results show that all kinematic, dynamic and energy characteristics of blood flow improved after surgery. Though, they are not same as a normal case. The shape of LV is changed after surgery from elongated shape to spherical shape. The direction of crescent vortex after surgery does not help to better ejection of blood into ascending aorta. The surgeons should go toward to keep the elongated shape of LV, because in elongated shape the crescent vortex could asymmetrically grows and travels toward apex.

8. Conclusions and Future Works

8.2 Contribution of this thesis

In this study, we have demonstrated a quantitative assessment of LV conditions in normal and patient cases. For the first time, energy transfer from the LV is computed and analyzed for normal and abnormal cases. The effects of leaflet motions for both valves on kinematic, dynamic and energy characteristics of blood flow in LV are assessed. The details of fluid dynamics such as pressure difference, shear stress and net pressure are presented which help to better understanding of ventricular blood flow. The quantity of each term in work-energy equation is determined to assess the significant of each term in 2D and 3D modeling of LV for normal and abnormal cases. A case before and after CABG+SVR, with the details of fluid dynamics is modeled. In this study, we have presented the details of kinematic, dynamic and energy characteristics of blood flow in LV using 2D and 3D modeling for normal and patient cases.

8.3 Recommendations for future work

The future work could be the modeling of 3D LV with valve leaflets during diastole and systole. However, because of medical and computational constraint it is very difficult and challenging.

In future work, perhaps it would be good to simulate some cases that are in the early stage of abnormal, such as the flow pattern is overlapping between the normal and abnormal cases. This may give some idea on how good the CFD works particularly when FSI is included.

Modeling the LV before and after surgery for other type of surgeries could help

8. Conclusions and Future Works

the surgeons to improve their operation and create a new idea for them.

Including the other cardiac functions such as structure, tissue and cell metabolism in a multiphysics frame work could present a realistic model of LV. Hunter *et al.* (2003) have proposed such framework but, until now, there is not any progress about it. The modeling of heart with all functions should be the final goal.

In order to validate the numerical models, the experimental data is needed. Design a suitable platform for LV as an *in vitro* study and then use the experiment measurement for detailed quantitative comparison. For more accurate validation, *in vivo* study is obviously needed but challenging.

List of Publications Due to This Thesis

1. S.S. Khalafvand, E.Y.K. Ng, and L. Zhong. CFD simulation of flow through heart: a perspective review. *Computer Methods in Biomechanics and Biomedical Engineering*, 2011. 14(1): pp. 113 - 132.
2. S.S. Khalafvand, E.Y.K. Ng, L. Zhong, and T.K. Hung. Fluid-dynamics modeling of the human left ventricle with dynamic mesh for normal and myocardial infarction: Preliminary study. *Computers in Biology and Medicine*, 2012. 42(8): pp. 863-870.
3. S.S. Khalafvand, E.Y.-K. Ng, and L. Zhong. Three-Dimensional MRI-based Computational Fluid Modeling of the Left Ventricle for Patient before and after Surgical Ventricular Restoration. Proceeding *International Conference on Biomedical Engineering and Biotechnology (iCBEB)*, IEE Macau, 2012. pp. 637-640. DOI: 10.1109/iCBEB.2012.481
4. S.S. Khalafvand, E.Y.K. Ng, L. Zhong. Quantification of left ventricular kinetic energy using three-dimensional CFD/MRI flow modeling. *Medical Engineering & Physics*, 2013 (under review).
5. S.S. Khalafvand, L. Zhong, E.Y.K. Ng. Surgical ventricular restoration im-

proves blood flow in the left ventricle from three-dimensional CFD/MRI modeling. *International Journal for Numerical Methods in Biomedical Engineering* (under review, MS ID:CNM-Mar-13-0050).

6. S.S. Khalafvand, T.K. Hung, E.Y.K. Ng, and L. Zhong. Kinematic, dynamic and energy characteristics of diastolic flow in the left ventricle. *Journal of Fluid Mechanics*, 2013 (under review, MS ID:JFM-13-S-0725).
7. T.K. Hung, S.S. Khalafvand and E.Y.K. Ng. Fluid mechanics characteristics of systolic flow in the left ventricle. *Journal of Fluid Mechanics*, 2013 (under review, MS ID:JFM-13-S-0726).

Bibliography

- ALURI, S. & CHANDRAN, K. B. 2001 Numerical simulation of mechanical mitral heart valve closure. *Annals of Biomedical Engineering* **29** (8), 665–676. 48
- ATHANASULEAS, C. L., BUCKBERG, G. D., STANLEY, A. W. H., SILER, W., DOR, V., DI DONATO, M., MENICANTI, L., ALMEIDA DE OLIVEIRA, S., BEYERSDORF, F., KRON, I. L., SUMA, H., KOUCHOUKOS, N. T., MOORE, W., MCCARTHY, P. M., OZ, M. C., FONTAN, F., SCOTT, M. L. & ACCOLA, K. A. 2004 Surgical ventricular restoration in the treatment of congestive heart failure due to post-infarction ventricular dilation. *Journal of the American College of Cardiology* **44** (7), 1439–1445. 123
- AUTHORS/TASK FORCE, M., DICKSTEIN, K., COHEN-SOLAL, A., FILIPATOS, G., MCMURRAY, J. J. V., PONIKOWSKI, P., POOLE-WILSON, P. A., STRMBERG, A., VAN VELDHUISEN, D. J., ATAR, D., HOES, A. W., KEREN, A., MEBAZAA, A., NIEMINEN, M., PRIORI, S. G., SWEDBERG, K., GUIDELINES, E. S. C. C. F. P., VAHANIAN, A., CAMM, J., DE CATERINA, R., DEAN, V., FUNCK-BRENTANO, C., HELLEMANS, I., KRISTENSEN, S. D., MCGREGOR, K., SECHTEM, U., SILBER, S., TENDERA, M., WIDIMSKY, P., ZAMORANO, J. L., DOCUMENT, R., AURICCHIO, A., BAX, J., BHM, M., CORR, U., DELLA BELLA, P., ELLIOTT, P. M., FOLLATH, F.,

BIBLIOGRAPHY

- GHEORGHIAD, M., HASIN, Y., HERNBORG, A., JAARSMA, T., KOMAJDA, M., KORNOWSKI, R., PIEPOLI, M., PRENDERGAST, B., TAVAZZI, L., VACHIER, J.-L., VERHEUGT, F. W. A. & ZANNAD, F. 2008 Esc guidelines for the diagnosis and treatment of acute and chronic heart failure 2008. *European Heart Journal* **29** (19), 2388–2442. 1
- BACCANI, B., DOMENICHINI, F. & PEDRIZZETTI, G. 2002a Vortex dynamics in a model left ventricle during filling. *European Journal of Mechanics - B/Fluids* **21** (5), 527–543. 11, 12, 44, 48
- BACCANI, B., DOMENICHINI, F. & PEDRIZZETTI, G. 2003 Model and influence of mitral valve opening during the left ventricular filling. *Journal of Biomechanics* **36** (3), 355–361. 11, 12, 48
- BACCANI, B., FEDERICO, D., GIANNI, P. & GIOVANNI, T. 2002b Fluid dynamics of the left ventricular filling in dilated cardiomyopathy. *Journal of Biomechanics* **35** (5), 665–671. 11, 48
- BELLHOUSE, B. J. 1969 Velocity and pressure distributions in the aortic valve. *Journal of Fluid Mechanics* **37** (03), 587–600. 47
- BELLHOUSE, B. J. & TALBOT, L. 1969 The fluid mechanics of the aortic valve. *Journal of Fluid Mechanics* **35** (04), 721–735. 47, 66
- BOLOOKI, H., DEMARCHENA, E., MALLON, S. M., KATARIYA, K., BARRON, M., BOLOOKI, H. M., THURER, R. J., NOVAK, S. & DUNCAN, R. C. 2003 Factors affecting late survival after surgical remodeling of left ventricular aneurysms. *Journal of Thoracic and Cardiovascular Surgery* **126** (2), 374–385. 123

BIBLIOGRAPHY

- BOLZON, G., ZOVATTO, L. & PEDRIZZETTI, G. 2003 Birth of three-dimensionality in a pulsed jet through a circular orifice. *Journal of Fluid Mechanics* **493** (1), 209–218. 11, 12, 48
- BROWN, T. D. & HUNG, T.-K. 1977 Computational and experimental investigations of two-dimensional nonlinear peristaltic flows. *Journal of Fluid Mechanics* **83** (02), 249–272. 78
- CARLSSON, M., HEIBERG, E., TOGER, J. & ARHEDEN, H. 2012 Quantification of left and right ventricular kinetic energy using four-dimensional intracardiac magnetic resonance imaging flow measurements. *American Journal of Physiology - Heart and Circulatory Physiology* **302** (4), H893–H900. 84, 120
- CHENG, Y., OERTEL, H. & SCHENKEL, T. 2005 Fluid-structure coupled cfd simulation of the left ventricular flow during filling phase. *Annals of Biomedical Engineering* **33** (5), 567–576. 10, 42, 49
- COCHRAN, R. P. & KUNZELMAN, K. S. 1998 Effect of papillary muscle position on mitral valve function: relationship to homografts. *The Annals of Thoracic Surgery* **66** (6, Supplement 1), S155–S161. 48
- DE GONZLEZ, A. B. & DARBY, S. 2004 Risk of cancer from diagnostic x-rays: estimates for the uk and 14 other countries. *The Lancet* **363** (9406), 345–351. 16
- DE HART, J., PETERS, G. W. M., SCHREURS, P. J. G. & BAAIJENS, F. P. T. 2000 A two-dimensional fluidstructure interaction model of the aortic valve. *Journal of Biomechanics* **33** (9), 1079–1088. 47

BIBLIOGRAPHY

- DE HART, J., PETERS, G. W. M., SCHREURS, P. J. G. & BAAIJENS, F. P. T. 2003 A computational fluid-structure interaction analysis in the aortic valve. *J. Biomech.* **36**, 103. 47
- DOENST, T., SPIEGEL, K., REIK, M., MARKL, M., HENNIG, J., NITZSCHE, S., BEYERSDORF, F. & OERTEL, H. 2009 Fluid-dynamic modeling of the human left ventricle: Methodology and application to surgical ventricular reconstruction. *The Annals of Thoracic Surgery* **87** (4), 1187–1195. 123
- DOMENICHINI, F. & PEDRIZZETTI, G. 2011 Intraventricular vortex flow changes in the infarcted left ventricle: numerical results in an idealised 3d shape. *Computer Methods in Biomechanics and Biomedical Engineering* **14** (1), 95 – 101. 11, 12
- DOMENICHINI, F., PEDRIZZETTI, G. & BACCANI, B. 2005 Three-dimensional filling flow into a model left ventricle. *Journal of Fluid Mechanics* **539**, 179–198. 11, 12, 43, 44, 48
- DOMENICHINI, F., QUERZOLI, G., CENEDESE, A. & PEDRIZZETTI, G. 2007 Combined experimental and numerical analysis of the flow structure into the left ventricle. *Journal of Biomechanics* **40** (9), 1988–1994. 12
- DUMONT, K., STIJNEN, J. M. A., VIERENDEELS, J., VAN DE VOSSE, F. N. & VERDONCK, P. R. 2004 Validation of a fluidstructure interaction model of a heart valve using the dynamic mesh method in fluent. *Computer Methods in Biomechanics and Biomedical Engineering* **7** (3), 139 – 146. 47
- EBBERS, T., WIGSTROM, L., BOLGER, A. F., WRANNE, B. & KARLSSON, M. 2002 Noninvasive measurement of time-varying three-dimensional relative

BIBLIOGRAPHY

- pressure fields within the human heart. *Journal of Biomechanical Engineering* **124** (3), 288–293. 9, 42
- EINSTEIN, D., KUNZELMAN, K., REINHALL, P., NICOSIA, M. & COCHRAN, R. 2004 Haemodynamic determinants of the mitral valve closure sound: A finite element study. *Medical and Biological Engineering and Computing* **42** (6), 832–846. 48
- EINSTEIN, D. R., KUNZELMAN, K. S., REINHALL, P. G., NICOSIA, M. A. & COCHRAN, R. P. 2005 Non-linear fluid-coupled computational model of the mitral valve. *The Journal of heart valve disease* **14** (3), 376–85. 48
- EINSTEIN, D. R., PIN, F. D., JIAO, X., KUPRAT, A. P., CARSON, J. P., KUNZELMAN, K. S., COCHRAN, R. P., GUCCIONE, J. M. & RATCLIFFE, M. B. 2010 Fluid-structure interactions of the mitral valve and left heart: Comprehensive strategies, past, present and future. *International Journal for Numerical Methods in Biomedical Engineering* **26** (3-4), 348–380. 48
- FERZIGER, J. H. & PERIC, M. 1997 *Computational Methods for Fluid Dynamics*. Berlin: Springer. 21, 22
- GHARIB, M., RAMBOD, E., KHERADVAR, A., SAHN, D. J. & DABIRI, J. O. 2006 Optimal vortex formation as an index of cardiac health. *Proceedings of the National Academy of Sciences* **103** (16), 6305–6308. 44
- GILLANI, N. V. & SWANSON, W. M. 1976 Time-dependent laminar incompressible flow through a spherical cavity. *Journal of Fluid Mechanics* **78** (01), 99–127. 47

BIBLIOGRAPHY

- HIRT, C. W., AMSDEN, A. A. & COOK, J. L. 1974 An arbitrary lagrangian-eulerian computing method for all flow speeds. *Journal of Computational Physics* **14** (3), 227–253. 21, 22
- HUNG, T. K., BALASUBRAMANIAN, S. R., SIMON, M. A., SUFFOLETTO, M. S., BOROVETZ, H. S. & GORCSAN, J. 2008 Hemodynamic information and analysis of cardiac pumping. *Journal of Mechanics in Medicine and Biology* **08** (01), 87–95. 78, 79
- HUNG, T.-K. & SCHUESSLER, G. B. 1977 An analysis of the hemodynamics of the opening of aortic valves. *Journal of Biomechanics* **10** (9), 597–606. 47, 78
- HUNG, T. K., SCHUESSLER, G. B., ENERSON, D. M., SHIH, H. H., JARAMILLO, R. J. & BOROVETZ, H. S. 1978 Pressure flow relationship and dynamic behavior of prosthetic heart valves. *The South African Mechanical Engineering* **28**, 101–107. 66
- HUNG, T.-K. & TSAI, T. M.-C. 1996 Pulsatile blood flows in stenotic artery. *Journal of Engineering Mechanics* **122**. 102
- HUNG, T.-K. & TSAI, T. M.-C. 1997 Kinematic and dynamic characteristics of pulsatile flows in stenotic vessels. *Journal of Engineering Mechanics* **123**. 34, 37, 42, 54, 102
- HUNTER, P. J., PULLAN, A. J. & SMAILL, B. H. 2003 Modeling total heart function. *Annual Review of Biomedical Engineering* **5** (1), 147–177. 8, 164
- ISSA, R. I. 1986 Solution of the implicitly discretised fluid flow equation by operators-splitting. *J Comput. Phys.* **62**, 40–65. 25

BIBLIOGRAPHY

- KHALAFVAND, S. S., NG, E. Y. K. & ZHONG, L. 2011 Cfd simulation of flow through heart: a perspective review. *Computer Methods in Biomechanics and Biomedical Engineering* **14** (1), 113 – 132. 7, 49
- KHALAFVAND, S. S., NG, E. Y.-K. & ZHONG, L. 2012*a* Three-dimensional mri-based computational fluid modeling of the left ventricle for patient before and after surgical ventricular restoration. In *International Conference on Biomedical Engineering and Biotechnology (iCBEB), IEE Macau*, pp. 637–640. 7
- KHALAFVAND, S. S., NG, E. Y. K., ZHONG, L. & HUNG, T. K. 2012*b* Fluid-dynamics modelling of the human left ventricle with dynamic mesh for normal and myocardial infarction: Preliminary study. *Computers in Biology and Medicine* **42** (8), 863–870. 7, 157
- KILNER, P. J., YANG, G.-Z., WILKES, A. J., MOHIADDIN, R. H., FIRMIN, D. N. & YACoub, M. H. 2000 Asymmetric redirection of flow through the heart. *Nature* **404** (6779), 759–761. 9, 23, 34, 43, 44
- KIM, W. Y., WALKER, P. G., PEDERSEN, E. M., POULSEN, J. K., OYRE, S., HOULIND, K. & YOGANATHAN, A. P. 1995 Left ventricular blood flow patterns in normal subjects: A quantitative analysis by three-dimensional magnetic resonance velocity mapping. *Journal of the American College of Cardiology* **26** (1), 224–238. 9
- KING, M. J., CORDEN, J., DAVID, T. & FISHER, J. 1996 A three-dimensional, time-dependent analysis of flow through a bileaflet mechanical heart valve:

BIBLIOGRAPHY

- Comparison of experimental and numerical results. *Journal of Biomechanics* **29** (5), 609–618. 48
- KING, M. J., DAVID, T. & FISHER, J. 1994 An initial parametric study on fluid flow through bileaflet mechanical heart valves using computational fluid dynamics. *Proceedings of the Institution of Mechanical Engineers, Part H: Journal of Engineering in Medicine* **208** (2), 63–72. 48
- KLABUNDE, R. E. 2011 *Cardiovascular physiology concepts*, 2nd edn. Lippincott Williams and Wilkins. ix, 1, 2, 3, 4
- KRITTIAN, S., JANOSKE, U., OERTEL, H. & BHLKE, T. 2010a Partitioned fluidsolid coupling for cardiovascular blood flow. *Annals of Biomedical Engineering* **38** (4), 1426–1441. 10
- KRITTIAN, S., SCHENKEL, T., JANOSKE, U. & OERTEL, H. 2010b Partitioned fluidsolid coupling for cardiovascular blood flow: Validation study of pressure-driven fluid-domain deformation. *Annals of Biomedical Engineering* **38** (8), 2676–2689. 49
- KUNZELMAN, K. S., COCHRAN, R. P., CHUONG, C., RING, W. S., VERRIER, E. D. & EBERHART, R. D. 1993 Finite element analysis of the mitral valve. *The Journal of heart valve disease* **2** (3), 326–40. 48
- KUNZELMAN, K. S., EINSTEIN, D. R. & COCHRAN, R. P. 2007 Fluidstructure interaction models of the mitral valve: function in normal and pathological states. *Philosophical Transactions of the Royal Society B: Biological Sciences* **362** (1484), 1393–1406. 48

BIBLIOGRAPHY

- KUNZELMAN, K. S., QUICK, D. W. & COCHRAN, R. P. 1998 Altered collagen concentration in mitral valve leaflets: biochemical and finite element analysis. *The Annals of Thoracic Surgery* **66** (6, Supplement 1), S198–S205. 48
- KUNZELMAN, K. S., REIMINK, M. S. & COCHRAN, R. P. 1997 Annular dilatation increases stress in the mitral valve and delays coaptation: a finite element computer model. *Cardiovascular Surgery* **5** (4), 427–434. 48
- LAI, Y. G., CHANDRAN, K. B. & LEMMON, J. 2002 A numerical simulation of mechanical heart valve closure fluid dynamics. *J. Biomech.* **35**, 881. 48
- LAU, K. D., DIAZ, V., SCAMBLER, P. & BURRIESCI, G. 2010 Mitral valve dynamics in structural and fluidstructure interaction models. *Medical Engineering and Physics* **32** (9), 1057–1064. 48
- LEE, C. S. F. & TALBOT, L. 1979 A fluid-mechanical study of the closure of heart valves. *Journal of Fluid Mechanics* **91** (01), 41–63. 47
- LIM, K. H., YEO, J. H. & DURAN, C. M. 2005 Three-dimensional asymmetrical modeling of the mitral valve: a finite element study with dynamic boundaries. *The Journal of heart valve disease* **14** (3), 386–92. 48
- LONG, Q., MERRIFIELD, R., XU, X. Y., KILNER, P., FIRMIN, D. N. & YANG, G. Z. 2008 Subject-specific computational simulation of left ventricular flow based on magnetic resonance imaging. *Proceedings of the Institution of Mechanical Engineers, Part H: Journal of Engineering in Medicine* **222** (4), 475–485. 14, 15, 48

BIBLIOGRAPHY

- LONG, Q., MERRIFIELD, R., YANG, G. Z., XU, X. Y., KILNER, P. J. & FIRMIN, D. N. 2003 The influence of inflow boundary conditions on intra left ventricle flow predictions. *Journal of Biomechanical Engineering* **125** (6), 922–927. 13, 14, 42, 48, 93
- MACAGNO, E. O. & HUNG, T.-K. 1967 Computational and experimental study of a captive annular eddy. *Journal of Fluid Mechanics* **28** (01), 43–64. 78
- MAISANO, F., REDAELLI, A., SONCINI, M., VOTTA, E., ARCOBASSO, L. & ALFIERI, O. 2005 An annular prosthesis for the treatment of functional mitral regurgitation: Finite element model analysis of a dog boneshaped ring prosthesis. *The Annals of Thoracic Surgery* **79** (4), 1268–1275. 48
- MCMURRAY, J. J. V. & PFEFFER, M. A. 2005 Heart failure. *The Lancet* **365** (9474), 1877–1889. 1
- MCQUEEN, D. M. & PESKIN, C. S. 1985 Computer-assisted design of butterfly bileaflet valves for the mitral position. *Scandinavian Cardiovascular Journal* **19** (2), 139–148. 10, 48, 49
- MCQUEEN, D. M. & PESKIN, C. S. 1989 A three-dimensional computational method for blood flow in the heart. ii. contractile fibers. *Journal of Computational Physics* **82** (2), 289–297. 10, 49
- MCQUEEN, D. M. & PESKIN, C. S. 1997 Shared-memory parallel vector implementation of the immersed boundary method for the computation of blood flow in the beating mammalian heart. *The Journal of Supercomputing* **11** (3), 213–236. 10, 49

BIBLIOGRAPHY

- MCQUEEN, D. M. & PESKIN, C. S. 2000 A three-dimensional computer model of the human heart for studying cardiac fluid dynamics. *Comput Graph* **34**, 56–60. 3, 10, 49
- MCQUEEN, D. M. & PESKIN, C. S. 2002 *Heart Simulation by an Immersed Boundary Method with Formal Second-order Accuracy and Reduced Numerical Viscosity Mechanics for a New Mellennium*. Springer Netherlands. 10, 49
- MICKLEBOROUGH, L. L., MERCHANT, N., IVANOV, J., RAO, V. & CAR-SON, S. 2004 Left ventricular reconstruction: Early and late results. *Journal of Thoracic and Cardiovascular Surgery* **128** (1), 27–37. 123
- NAKAMURA, M., WADA, S. & YAMAGUCHI, T. 2006 Influence of the opening mode of the mitral valve orifice on intraventricular hemodynamics. *Annals of Biomedical Engineering* **34** (6), 927–935. 14, 49
- PEDRIZZETTI, G. & DOMENICHINI, F. 2005 Nature optimizes the swirling flow in the human left ventricle. *Physical Review Letters* **95** (10), 108101. 11, 12, 48
- PESKIN, C. S. 1972 Flow patterns around heart valves: a numerical method. *J. Comput. Phys.* **10**, 252. 10, 49
- PESKIN, C. S. 1977 Numerical analysis of blood flow in the heart. *J. Comput. Phys.* **25**, 220. 49
- PROT, V., HAAVERSTAD, R. & SKALLERUD, B. 2009 Finite element analysis of the mitral apparatus: annulus shape effect and chordal force distribution. *Biomechanics and Modeling in Mechanobiology* **8** (1), 43–55. 48

BIBLIOGRAPHY

- PROT, V. & SKALLERUD, B. 2009 Nonlinear solid finite element analysis of mitral valves with heterogeneous leaflet layers. *Computational Mechanics* **43** (3), 353–368. 48
- PRUCZ, R. B., WEISS, E. S., PATEL, N. D., NWAKANMA, L. U., BAUMGARTNER, W. A. & CONTE, J. V. 2008 Coronary artery bypass grafting with or without surgical ventricular restoration: A comparison. *The Annals of Thoracic Surgery* **86** (3), 806–814. 123
- QUAINI, A., CANIC, S., GUIDOBONI, G., GLOWINSKI, R., IGO, S., HARTLEY, C., ZOGHBI, W. & LITTLE, S. 2011 A three-dimensional computational fluid dynamics model of regurgitant mitral valve flow: Validation against in vitro standards and 3d color doppler methods. *Cardiovascular Engineering and Technology* **2** (2), 77–89. 48
- ROACHE, P. J. 1998 *Verification and validation in computational science and engineering*. Hermosa Publishers, Albuquerque, New Mexico. 30
- SABER, N., GOSMAN, A. D., WOOD, N., KILNER, P., CHARRIER, C. & FIRMIN, D. 2001 Computational flow modeling of the left ventricle based on in vivo mri data: initial experience. *Annals of Biomedical Engineering* **29** (4), 275–283. 13, 24, 48
- SABER, N. R., WOOD, N. B., GOSMAN, A. D., MERRIFIELD, R. D., YANG, G.-Z., CHARRIER, C. L., GATEHOUSE, P. D. & FIRMIN, D. N. 2003 Progress towards patient-specific computational flow modeling of the left heart via combination of magnetic resonance imaging with computational fluid dynamics. *Annals of Biomedical Engineering* **31** (1), 42–52. 13, 48

BIBLIOGRAPHY

- SARTIPY, U., ALBAGE, A., STRAAT, E., INSULANDER, P. & LINDBLOM, D. 2006 Surgery for ventricular tachycardia in patients undergoing left ventricular reconstruction by the dor procedure. *Annals of Thoracic Surgery* **81** (1), 65–71. 123
- SARTIPY, U., ALBGE, A. & LINDBLOM, D. 2005 The dor procedure for left ventricular reconstruction. ten-year clinical experience. *European Journal of Cardio-Thoracic Surgery* **27** (6), 1005–1010. 123
- SCHENCK, J. F. 2000 Safety of strong, static magnetic fields. *Journal of Magnetic Resonance Imaging* **12** (1), 2–19. 16
- SCHENKEL, T., MALVE, M., REIK, M., MARKL, M., JUNG, B. & OERTEL, H. 2009 Mri-based cfd analysis of flow in a human left ventricle: Methodology and application to a healthy heart. *Annals of Biomedical Engineering* **37** (3), 503–515. 14, 15, 25, 42, 48, 49, 93
- STIJNEN, J. M. A., DE HART, J., BOVENDEERD, P. H. M. & VAN DE VOSSE, F. N. 2004 Evaluation of a fictitious domain method for predicting dynamic response of mechanical heart valves. *Journal of Fluids and Structures* **19** (6), 835–850. 47
- VAN STEENHOVEN, A. A. & VAN DONGEN, M. E. H. 1979 Model studies of the closing behaviour of the aortic valve. *Journal of Fluid Mechanics* **90** (01), 21–32. 47
- VIERENDEELS, J. A., RIEMSLAGH, K., DICK, E. & VERDONCK, P. R. 2000 Computer simulation of intraventricular flow and pressure gradients during diastole. *Journal of Biomechanical Engineering* **122** (6), 667–674. 11, 42, 48

BIBLIOGRAPHY

- VOTTA, E., CAIANI, E., VERONESI, F., SONCINI, M., MONTEVECCHI, F. M. & REDAELLI, A. 2008 Mitral valve finite-element modelling from ultrasound data: a pilot study for a new approach to understand mitral function and clinical scenarios. *Philosophical Transactions of the Royal Society A: Mathematical, Physical and Engineering Sciences* **366** (1879), 3411–3434. 48
- VOTTA, E., MAISANO, F., BOLLING, S. F., ALFIERI, O., MONTEVECCHI, F. M. & REDAELLI, A. 2007 The geoform disease-specific annuloplasty system: A finite element study. *The Annals of Thoracic Surgery* **84** (1), 92–101. 48
- WATANABE, H., SUGIURA, S., KAFUKU, H. & HISADA, T. 2004 Multiphysics simulation of left ventricular filling dynamics using fluidstructure interaction finite element method. *Biophys J* **87**, 2074–2085. 10, 49
- WATTON, P. N., LUO, X. Y., WANG, X., BERNACCA, G. M., MOLLOY, P. & WHEATLEY, D. J. 2007 Dynamic modelling of prosthetic chorded mitral valves using the immersed boundary method. *Journal of Biomechanics* **40** (3), 613–626. 48
- WATTON, P. N., LUO, X. Y., YIN, M., BERNACCA, G. M. & WHEATLEY, D. J. 2008 Effect of ventricle motion on the dynamic behaviour of chorded mitral valves. *Journal of Fluids and Structures* **24** (1), 58–74. 48
- YEO, S., ZHONG, L., SU, Y., TAN, R. & GHISTA, D. 2009 A curvature-based approach for left ventricular shape analysis from cardiac magnetic resonance imaging. *Medical and Biological Engineering and Computing* **47** (3), 313–322. 17

BIBLIOGRAPHY

- YOGANATHAN, A. P., CHANDRAN, K. & SOTIROPOULOS, F. 2005 Flow in prosthetic heart valves: State-of-the-art and future directions. *Annals of Biomedical Engineering* **33** (12), 1689–1694. 1, 46
- ZHONG, L., SU, Y., GOBEAWAN, L., SOLA, S., TAN, R.-S., NAVIA, J. L., GHISTA, D. N., CHUA, T., GUCCIONE, J. & KASSAB, G. S. 2011 Impact of surgical ventricular restoration on ventricular shape, wall stress, and function in heart failure patients. *American Journal of Physiology - Heart and Circulatory Physiology* **300** (5), H1653–H1660. 17, 44, 123
- ZHONG, L., SU, Y., YEO, S.-Y., TAN, R.-S., GHISTA, D. N. & KASSAB, G. 2009 Left ventricular regional wall curvedness and wall stress in patients with ischemic dilated cardiomyopathy. *American Journal of Physiology - Heart and Circulatory Physiology* **296** (3), H573–H584. 17