

**Relationship between left ventricular torsion
early after myocardial infarction and
remodeling in patients with ST segment
elevation myocardial infarction treated by
percutaneous coronary intervention**

Thesis

Submitted for Partial Fulfillment of MD Degree in
Cardiology

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2015

Acknowledgment

*First and foremost, I feel always indebted to **ALLAH**, the Most Kind and Most Merciful.*

*I'd like to express my respectful thanks and profound gratitude to **Prof.\ Mohamed Ayman Mostafa A.Saleh**, Professor of Cardiology - Faculty of Medicine- Ain Shams University for his keen guidance, kind supervision, valuable advice and continuous encouragement, which made possible the completion of this work.*

*I am also delighted to express my deepest gratitude and thanks to **Dr\ Hany Mohamed Ahmed Awad Allah**, Assistant Professor of Cardiology, Faculty of Medicine, Ain Shams University, for his kind care, continuous supervision, valuable instructions, constant help and great assistance throughout this work.*

*I wish to introduce my deep respect and thanks to **Dr\ Mohamed Abd el Kader Abd el Reheem**, Lecturer of Cardiology, Faculty of Medicine, Ain Shams University, for her kindness, supervision and cooperation in this work.*

I would like to express my hearty thanks to all my family for their support till this work was completed.

Last but not least my sincere thanks and appreciation to all patients participated in this study.

Amr Mansour Mohamed Zaky

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List of Abbreviations

Abb.	Full term
2D.....	Two dimensional
ABP.....	Arterial blood pressure
ACEI.....	Angiotensin converting enzyme inhibitors
AMI.....	Acute myocardial infarction
AVC.....	Aortic valve closure
BB.....	Beta-blocker
BMI.....	Body mass index
BP.....	Blood pressure
CAD.....	Coronary artery disease
CK.....	Creatine kinase
CK-MB.....	Creatine Kinase-myocardial band
CRT.....	Cardiac re-synchronization therapy
DALYs.....	Disability-adjusted life years
DD.....	Diastolic dysfunction
DSE.....	Dobutamine stress echocardiography
ECG.....	Electrocardiogram
ECM.....	Extra-cellular matrix
EDD.....	End diastole dimension
EDV.....	End diastolic volume
EDVI.....	End diastolic volume index
EF.....	Ejection fraction
ESD.....	End systole dimension
ESV.....	End systolic volume
ESVI.....	End systolic volume index
LA.....	Left atrium
LAD.....	Left anterior descending artery
LAVI.....	Left atrial volume index
LCX.....	Left circumflex artery
LV.....	Left ventricle

List of Abbreviations (cont...)

Abb.	Full term
LVEDD	Left ventricular end diastolic diameter
LVEF.....	Left ventricle ejection fraction
LVESD.....	Left ventricle end systolic diameters
MACE	Major adverse cardiac events
MBG.....	Myocardial blush grade
MI.....	Myocardial infarction
MO	Microvascular obstruction
MR.....	Mitral regurgitation
MRI	Magnetic resonance imaging
NSTEMI	Non ST segment elevation myocardial infarction
PCI	Per cutaneous coronary intervention
PPCI.....	Primary per cutaneous coronary intervention
PTCA.....	Percutaneous trans-luminal coronary angioplasty
RCA.....	Right coronary artery
ROC.....	Receiver operating characteristic curve
ROI.....	Region of interest
RV	Right ventricle
STE	Speckle tracking echocardiography
STEMI	ST segment elevation myocardial infarction
TIMI.....	Thrombolysis in myocardial infarction
WMSI.....	Wall motion score index

INTRODUCTION

After successful reperfusion therapy in the hyper-acute stage of acute myocardial infarction (MI), dysfunctional myocardial segments subtended by the infarct-related artery can follow two different natural courses: functional recovery or irreversible remodeling. Predicting functional recovery or remodeling remains an elusive goal of echocardiography (*Soloman et al., 2001*).

The clinical importance of LV (left ventricular) remodeling was emphasized by White et al., who demonstrated that patients who died during follow-up after myocardial infarction had significantly larger LV volumes and lower left ventricular ejection fraction (LVEF) than survivors. As a consequence, early identification of patients with LV remodeling after myocardial infarction is of vital importance (*White et al., 1987*).

Several variables have been identified to predict an increase in LV volume and a decrease in LV ejection fraction after an acute MI. These include infarct size (*Guardon et al., 1993, Chareon Thaitwae et al., 1995*), anterior location (*Warren et al., 1988*), cardiac enzyme index (*Rao et al., 1998*), trans-mural extent of infarction (*Bolognese et al., 1997*), patency of infarct-related artery (*Jermey et al., 1987*), end-systolic volume (ESV) (*White et al., 1987*), microvascular obstruction (*Wu et al., 1998*) and mitral deceleration time

(*Temporelli et al., 2004*). However, these are interrelated risk factors and each measure reflects a different aspect of the disease state and none can currently be considered as definitive (*Zhang et al., 2005*).

The systolic twisting of the LV along its longitudinal axis resulting from opposite rotation of the LV apex compared with the base is emerging as an important sensitive parameter of LV systolic function (*Sengupta et al., 2008*).

Recently, echocardiographic assessment of LV torsion mechanics based on 2 D speckle-tracking has been introduced and validated (*Notomi et al., 2005*).

In the clinical setting, however, not much data on changes in LV torsion after AMI are available and no specific data exist concerning the role of LV torsion in predicting post-infarction LV remodeling.

AIM OF THE WORK

To assess the relationship between left ventricular torsion measured by 2 D speckle tracking early after myocardial infarction and remodeling in patient with ST elevation myocardial infarction treated by primary PCI (PPCI).

*Chapter One***CARDIAC ARCHITECTURE AND
MYOCARDIAL MECHANICS****Cardiac architecture:**

The normal heart presents an architectural design that allows the contractile apparatus to empty and fill with optimal mechanical efficiency, determined by integration of vectors of force generated by sarcomeres that can only shorten by active contraction (*Gerald et al., 2008*).

Keith, in 1918, presented a currently unfulfilled challenge by stating, “We cannot claim to have mastered the mechanism of the human heart until we have a fundamental explanation of its architecture” (*Gerald et al., 2008*).

Ever since Danish anatomist, Nicolaus Steno (1638—1686), settled the muscular nature of the heart, in 1663, the architecture of the ventricular myocardium became a fascination for the generations of investigators (*Mladen et al., 2006*).

In addition to many other features, almost all historical predecessors in the field, from Richard Lower (1631—1691) onward, have recognized helical, trans-mural, overlapping pattern of the ventricular myocardial fibers. Unresolved problem was to reveal unique, rule-based assignment which, as