Correlation of Acute Change in Left Ventricle End diastolic Pressure after Primary Percutaneous Coronary Intervention with cardiac biomarkers in Patients with ST Segment Elevation Myocardial Infarction

Thesis Submitted by

Osama Amin Abd EL-Hamid, MBBCh

In Partial Fulfillment of

Master Degree in Critical Care Medicine

Supervisors

Tarek EL-Gohary, MD.

Professor of Critical Care Medicine
Critical Care Department
Cairo University

Ahmed Battah, MD

Ass.Professor of Critical Care Medicine

Critical Care Department

Cairo University

Akram Abd EL.Bary, MD

Ass.Professor of Critical Care Medicine

Critical Care Department

Cairo University

ACKNOWLEDGMENT

Praise be to Allah, the creator and sustainer of the world, who has said in his holy Quran" We raise to degrees (of wisdom) whom we please, but overall endued with knowledge is one, the all-knowing" (Yusuf 76).

Thank to Prof. Dr. Sherif Mokhtar, our Master mind, I always owe him much. He offered us not only the idea and facilities to complete our researches but also the spirit of being eager to gain more experience and skills. Words are not sufficient to express my deep gratitude for him.

I would like to start by sending my deepest gratitude and sincere thanks to Dr. Alia Abd.EL.fattah, Chief of Critical Care Medicine Department, Cairo University, for her help and continuous support. I am extremely grateful for her advice and for her guidance and support throughout that work.

Special thanks to **Dr. Tarek EL.gohry**, Professor of Critical Care Medicine, Cairo University, for his help and continuous support. I am extremely grateful to him for his generous advice and for his guidance and assistance throughout the whole work.

I am deeply thankful to **Dr.Ahmed Battah**, Assistant professor of Critical Care Medicine, Cairo University, for his guidance. He teached me how to approach matters in a scientific and displined way, kindness and constructive advice and for treating me in a brotherly way.

I would like to express my deep sense of gratitude to **Dr. Akram Abd-El.Bary**, Assistant professor of Critical Care Medicine who had spared no effort in guiding me throughout the long and tiring task of writing this thesis.

Finally I am so thankful and honored to belong to the critical care medicine department, the land of imagination, innovation and fruitful research. Many thanks should go especially to the catheter laboratory team for their support.

Osama Amin

List of Tables

| Item | Page |
|---|------|
| Table (1): Non ischemic factors affecting BNP levels | 38 |
| Table (2): National Academy of Clinical Biochemistry Recommendations for Use of | 45 |
| Biochemical Markers for Risk Stratification in ACS | |
| Table (3): Distribution of the studied cases as regards clinical risk factors | 62 |
| Table(4):Distribution of the studied cases as regards | 63 |
| Killip class at presentation | |
| Table (5): Distribution of studied cases as regard TIMI flow before primary PCI | 64 |
| Table (6) Distribution of studied cases as regard TIMI flow | 64 |
| after primary PCI: | |
| Table (7) Distribution of the studied cases as regard Cuiprit vessel | 65 |
| Table (8): LVEDP data of the presnt study: | 66 |
| Table (9): Relationship between Pre-PCI LVEDP level at presentation and TIMI flow | 68 |
| pre-PCI | |
| Table (10): Relation between pre-LVEDP & post-LVEDP | 69 |
| Table (11): clinical outcome data of the study population . | |
| Table (12): Relation between Δ LVEDP & Inhospital clinical outcome | 71 |
| Table (13): Anterior versus inferior STEMI. | 72 |
| Table (14): BNP data of the present study. | 72 |
| Table (15): Correlation between BNP level at presentation and TIMI flow pre-PCI. | 74 |
| Table (16): Relation between change in BNP level and other clinical paremeters. | 77 |
| Table (17): Relation between presentation time & MACE: | 79 |

List of figures

| 1 The relation between the cardiac cycle pressure and ECG. 2 The ischemic cascade. 3 Ventricular pressure tracing. 4 Proposed mechanisms of BNP release in coronary ischemic disease. 5 Incidence of death and new CHF at 30 d and 10 following ACS 6 Biomarker profile in acute coronary syndromes 7 Technique for retrograde crossing of an aortic valve using a pigtail catheter. 8 Age distribution of study cases 9 Gender distribution of study cases. 59 10 Hypertension distribution of study cases. 59 10 Hypertension distribution of study cases. 60 11 Distribution of dyslipidemic to non-dyslipidemic patients. 61 12 Distribution of diabetic to non-diabetic patients. 61 13 Relation of smokers to non-smokers. 61 14 Distribution of study population regarding location of MI. 63 15 Distribution of studied cases as regard TIMI flow after primary PCI 16 Distribution of the studied cases as regard Cuiprit vessel 65 primary PCI 16 Distribution of the presnt study 67 18 Relationship between Pre-PCI LVEDP level at presentation and TIMI flow pre-PCI 19 Relation between PRP-PCI LVEDP & post-LVEDP 20 BNP data of the presnt study 21 Correlation between BNP level at presentation and TIMI flow pre-PCI 22 Correlation between LVEDP pre-PCI and BNP on day 5 post-PCI 23 Correlation between LVEDP post-PCI and BNP on day 5 post-PCI 24 Correlation between LVEDP and Δ BNP 78 | Fig. No | Title | Page |
|---|---------|--|------|
| 3 Ventricular pressure tracing. 4 Proposed mechanisms of BNP release in coronary ischemic disease. 5 Incidence of death and new CHF at 30 d and 10 following ACS 6 Biomarker profile in acute coronary syndromes 45 7 Technique for retrograde crossing of an aortic valve using a pigtail catheter. 8 Age distribution of study cases 58 9 Gender distribution of study cases. 59 10 Hypertension distribution of study cases 60 11 Distribution of dyslipidemic to non-dyslipidemic patients. 60 12 Distribution of diabetic to non-diabetic patients. 61 13 Relation of smokers to non-smokers. 61 14 Distribution of study population regarding location of MI. 63 15 Distribution of study population regard TIMI flow after primary PCI 16 Distribution of the studied cases as regard Cuiprit vessel 66 17 LVEDP data of the presnt study 67 18 Relationship between Pre-PCI LVEDP level at presentation and TIMI flow pre-PCI 19 Relation between pre-LVEDP & post-LVEDP 69 20 BNP data of the presnt study 73 21 Correlation between BNP level at presentation and TIMI flow pre-PCI 20 Correlation between LVEDP pre-PCI and BNP on day 5 post-PCI 23 Correlation between LVEDP post-PCI and BNP on day 5 post-PCI 23 Correlation between LVEDP post-PCI and BNP on day 5 post-PCI 23 Correlation between LVEDP post-PCI and BNP on day 5 post-PCI | | The relation between the cardiac cycle pressure and ECG. | 9 |
| 4 Proposed mechanisms of BNP release in coronary ischemic disease. 5 Incidence of death and new CHF at 30 d and 10 following ACS 6 Biomarker profile in acute coronary syndromes 45 7 Technique for retrograde crossing of an aortic valve using a pigtail catheter. 8 Age distribution of study cases 58 9 Gender distribution of study cases. 59 10 Hypertension distribution of study cases. 60 11 Distribution of dyslipidemic to non-dyslipidemic patients. 60 12 Distribution of diabetic to non-diabetic patients. 61 13 Relation of smokers to non-smokers. 61 14 Distribution of study population regarding location of MI. 63 15 Distribution of studied cases as regard TIMI flow after primary PCI 65 16 Distribution of the studied cases as regard Cuiprit vessel 66 17 LVEDP data of the presnt study 67 18 Relationship between Pre-PCI LVEDP level at presentation and TIMI flow pre-PCI 9 19 Relation between pre-LVEDP & post-LVEDP 69 20 BNP data of the presnt study 73 21 Correlation between BNP level at presentation and TIMI flow pre-PCI Correlation between BNP level at presentation and TIMI flow pre-PCI Correlation between LVEDP pre-PCI and BNP on day 5 post-PCI Correlation between LVEDP post-PCI and BNP on day 5 post-PCI correlation between LVEDP post-PCI and BNP on day 5 post-PCI post-PCI | 2 | The ischemic cascade. | 14 |
| disease. Incidence of death and new CHF at 30 d and 10 following ACS Biomarker profile in acute coronary syndromes Technique for retrograde crossing of an aortic valve using a pigtail catheter. Replace distribution of study cases Gender distribution of study cases. Hypertension distribution of study cases. Hypertension distribution of study cases. Distribution of dyslipidemic to non-dyslipidemic patients. Collaboration of smokers to non-smokers. Relation of smokers to non-smokers. In distribution of study population regarding location of MI. Distribution of study population regarding location of MI. Distribution of studied cases as regard TIMI flow after primary PCI Distribution of the studied cases as regard Cuiprit vessel LVEDP data of the presnt study Relationship between Pre-PCI LVEDP level at presentation and TIMI flow pre-PCI Relation between pre-LVEDP & post-LVEDP BNP data of the presnt study Relation between BNP level at presentation and TIMI flow pre-PCI Correlation between BNP level at presentation and TIMI flow pre-PCI Correlation between LVEDP pre-PCI and BNP on day 5 post-PCI Correlation between LVEDP post-PCI and BNP on day 5 post-PCI Correlation between LVEDP post-PCI and BNP on day 5 post-PCI Correlation between LVEDP post-PCI and BNP on day 5 post-PCI | 3 | | 20 |
| ACS 6 Biomarker profile in acute coronary syndromes 45 7 Technique for retrograde crossing of an aortic valve using a pigtail catheter. 8 Age distribution of study cases 58 9 Gender distribution of study cases. 59 10 Hypertension distribution of study cases 60 11 Distribution of dyslipidemic to non-dyslipidemic patients. 60 12 Distribution of diabetic to non-diabetic patients. 61 13 Relation of smokers to non-smokers. 61 14 Distribution of study population regarding location of MI. 63 15 Distribution of studied cases as regard TIMI flow after primary PCI 16 Distribution of the studied cases as regard Cuiprit vessel 66 17 LVEDP data of the presnt study 67 18 Relationship between Pre-PCI LVEDP level at presentation and TIMI flow pre-PCI 19 Relation between pre-LVEDP & post-LVEDP 69 20 BNP data of the presnt study 73 21 Correlation between BNP level at presentation and TIMI flow pre-PCI 22 Correlation between LVEDP pre-PCI and BNP on day 5 post-PCI 23 Correlation between LVEDP post-PCI and BNP on day 5 post-PCI 23 Correlation between LVEDP post-PCI and BNP on day 5 post-PCI 25 Correlation between LVEDP post-PCI and BNP on day 5 post-PCI 26 Correlation between LVEDP post-PCI and BNP on day 5 post-PCI 26 Correlation between LVEDP post-PCI and BNP on day 5 post-PCI 27 Correlation between LVEDP post-PCI and BNP on day 5 post-PCI 27 Correlation between LVEDP post-PCI and BNP on day 5 post-PCI 27 Correlation between LVEDP post-PCI and BNP on day 5 post-PCI 28 Correlation between LVEDP post-PCI and BNP on day 5 post-PCI 29 post-PCI | - | | 36 |
| 7 Technique for retrograde crossing of an aortic valve using a pigtail catheter. 8 Age distribution of study cases | 5 | ACS | 41 |
| pigtail catheter. 8 Age distribution of study cases | 6 | Biomarker profile in acute coronary syndromes | |
| 9 Gender distribution of study cases. 59 10 Hypertension distribution of study cases 60 11 Distribution of dyslipidemic to non-dyslipidemic patients. 60 12 Distribution of diabetic to non-diabetic patients. 61 13 Relation of smokers to non-smokers. 61 14 Distribution of study population regarding location of MI. 63 15 Distribution of studied cases as regard TIMI flow after primary PCI 16 Distribution of the studied cases as regard Cuiprit vessel 66 17 LVEDP data of the presnt study 67 18 Relationship between Pre-PCI LVEDP level at presentation 68 and TIMI flow pre-PCI 19 Relation between pre-LVEDP & post-LVEDP 69 20 BNP data of the presnt study 73 21 Correlation between BNP level at presentation and TIMI flow 74 pre-PCI 22 Correlation between LVEDP pre-PCI and BNP on day 5 post-PCI 23 Correlation between LVEDP post-PCI and BNP on day 5 | 7 | | 55 |
| 10 Hypertension distribution of study cases 11 Distribution of dyslipidemic to non-dyslipidemic patients. 12 Distribution of diabetic to non-diabetic patients. 13 Relation of smokers to non-smokers. 14 Distribution of study population regarding location of MI. 15 Distribution of studied cases as regard TIMI flow after primary PCI 16 Distribution of the studied cases as regard Cuiprit vessel 17 LVEDP data of the presnt study 18 Relationship between Pre-PCI LVEDP level at presentation and TIMI flow pre-PCI 19 Relation between pre-LVEDP & post-LVEDP 20 BNP data of the presnt study 21 Correlation between BNP level at presentation and TIMI flow pre-PCI 22 Correlation between LVEDP pre-PCI and BNP on day 5 post-PCI 23 Correlation between LVEDP post-PCI and BNP on day 5 post-PCI 23 Correlation between LVEDP post-PCI and BNP on day 5 post-PCI | _ | Age distribution of study cases . | |
| 11 Distribution of dyslipidemic to non-dyslipidemic patients. 60 12 Distribution of diabetic to non-diabetic patients. 61 13 Relation of smokers to non-smokers. 61 14 Distribution of study population regarding location of MI. 63 15 Distribution of studied cases as regard TIMI flow after primary PCI 16 Distribution of the studied cases as regard Cuiprit vessel 66 17 LVEDP data of the presnt study 67 18 Relationship between Pre-PCI LVEDP level at presentation 68 and TIMI flow pre-PCI 19 Relation between pre-LVEDP & post-LVEDP 69 20 BNP data of the presnt study 73 21 Correlation between BNP level at presentation and TIMI flow pre-PCI 22 Correlation between LVEDP pre-PCI and BNP on day 5 post-PCI 23 Correlation between LVEDP post-PCI and BNP on day 5 | 9 | Gender distribution of study cases. | 59 |
| 12 Distribution of diabetic to non-diabetic patients. 13 Relation of smokers to non-smokers. 14 Distribution of study population regarding location of MI. 15 Distribution of studied cases as regard TIMI flow after primary PCI 16 Distribution of the studied cases as regard Cuiprit vessel 17 LVEDP data of the presnt study 18 Relationship between Pre-PCI LVEDP level at presentation and TIMI flow pre-PCI 19 Relation between pre-LVEDP & post-LVEDP 20 BNP data of the presnt study 21 Correlation between BNP level at presentation and TIMI flow pre-PCI 22 Correlation between LVEDP pre-PCI and BNP on day 5 post-PCI 23 Correlation between LVEDP post-PCI and BNP on day 5 post-PCI | 10 | Hypertension distribution of study cases | 60 |
| 13 Relation of smokers to non-smokers. 14 Distribution of study population regarding location of MI. 15 Distribution of studied cases as regard TIMI flow after 16 Distribution of the studied cases as regard Cuiprit vessel 16 LVEDP data of the presnt study 18 Relationship between Pre-PCI LVEDP level at presentation 20 And TIMI flow pre-PCI 20 BNP data of the presnt study 21 Correlation between BNP level at presentation and TIMI flow 22 Correlation between LVEDP pre-PCI and BNP on day 5 post-PCI 23 Correlation between LVEDP post-PCI and BNP on day 5 26 post-PCI 27 Correlation between LVEDP post-PCI and BNP on day 5 28 Correlation between LVEDP post-PCI and BNP on day 5 29 post-PCI | 11 | Distribution of dyslipidemic to non-dyslipidemic patients. | 60 |
| 14 Distribution of study population regarding location of MI. 15 Distribution of studied cases as regard TIMI flow after 65 primary PCI 16 Distribution of the studied cases as regard Cuiprit vessel 66 17 LVEDP data of the presnt study 67 18 Relationship between Pre-PCI LVEDP level at presentation and TIMI flow pre-PCI 19 Relation between pre-LVEDP & post-LVEDP 69 20 BNP data of the presnt study 73 21 Correlation between BNP level at presentation and TIMI flow pre-PCI 22 Correlation between LVEDP pre-PCI and BNP on day 5 post-PCI 23 Correlation between LVEDP post-PCI and BNP on day 5 post-PCI 26 Correlation between LVEDP post-PCI and BNP on day 5 post-PCI | 12 | Distribution of diabetic to non-diabetic patients. | 61 |
| 15 Distribution of studied cases as regard TIMI flow after primary PCI 16 Distribution of the studied cases as regard Cuiprit vessel 66 17 LVEDP data of the presnt study 67 18 Relationship between Pre-PCI LVEDP level at presentation and TIMI flow pre-PCI 19 Relation between pre-LVEDP & post-LVEDP 69 20 BNP data of the presnt study 73 21 Correlation between BNP level at presentation and TIMI flow pre-PCI 22 Correlation between LVEDP pre-PCI and BNP on day 5 post-PCI 23 Correlation between LVEDP post-PCI and BNP on day 5 post-PCI 26 Correlation between LVEDP post-PCI and BNP on day 5 post-PCI | 13 | Relation of smokers to non-smokers. | 61 |
| primary PCI 16 Distribution of the studied cases as regard Cuiprit vessel 66 17 LVEDP data of the presnt study 67 18 Relationship between Pre-PCI LVEDP level at presentation 68 and TIMI flow pre-PCI 19 Relation between pre-LVEDP & post-LVEDP 69 20 BNP data of the presnt study 73 21 Correlation between BNP level at presentation and TIMI flow pre-PCI 22 Correlation between LVEDP pre-PCI and BNP on day 5 post-PCI 23 Correlation between LVEDP post-PCI and BNP on day 5 post-PCI 26 Correlation between LVEDP post-PCI and BNP on day 5 post-PCI | 14 | Distribution of study population regarding location of MI. | 63 |
| 17 LVEDP data of the presnt study 18 Relationship between Pre-PCI LVEDP level at presentation 20 Relation between pre-LVEDP & post-LVEDP 20 BNP data of the presnt study 21 Correlation between BNP level at presentation and TIMI flow 22 pre-PCI 23 Correlation between LVEDP pre-PCI and BNP on day 5 post- PCI 23 Correlation between LVEDP post-PCI and BNP on day 5 post-PCI | 15 | Distribution of studied cases as regard TIMI flow after | 65 |
| 18 Relationship between Pre-PCI LVEDP level at presentation and TIMI flow pre-PCI 19 Relation between pre-LVEDP & post-LVEDP 20 BNP data of the presnt study 73 21 Correlation between BNP level at presentation and TIMI flow pre-PCI 22 Correlation between LVEDP pre-PCI and BNP on day 5 post-PCI 23 Correlation between LVEDP post-PCI and BNP on day 5 post-PCI | 16 | Distribution of the studied cases as regard Cuiprit vessel | 66 |
| and TIMI flow pre-PCI 19 Relation between pre-LVEDP & post-LVEDP 20 BNP data of the presnt study 73 21 Correlation between BNP level at presentation and TIMI flow pre-PCI 22 Correlation between LVEDP pre-PCI and BNP on day 5 post-PCI 23 Correlation between LVEDP post-PCI and BNP on day 5 post-PCI | 17 | LVEDP data of the presnt study | 67 |
| 20 BNP data of the presnt study 73 21 Correlation between BNP level at presentation and TIMI flow pre-PCI 22 Correlation between LVEDP pre-PCI and BNP on day 5 post-PCI 23 Correlation between LVEDP post-PCI and BNP on day 5 post-PCI 26 post-PCI | 18 | | 68 |
| 21 Correlation between BNP level at presentation and TIMI flow pre-PCI 22 Correlation between LVEDP pre-PCI and BNP on day 5 post-PCI 23 Correlation between LVEDP post-PCI and BNP on day 5 post-PCI | 19 | Relation between pre-LVEDP & post-LVEDP | 69 |
| pre-PCI 22 Correlation between LVEDP pre-PCI and BNP on day 5 post-PCI 23 Correlation between LVEDP post-PCI and BNP on day 5 post-PCI post-PCI | 20 | | |
| PCI 23 Correlation between LVEDP post-PCI and BNP on day 5 76 post-PCI | 21 | | 74 |
| post-PCI | 22 | | 75 |
| 24 Correlation between Δ LVEDP and Δ BNP 78 | 23 | post-PCI | 76 |
| | 24 | Correlation between Δ LVEDP and Δ BNP | 78 |

| 25 | Relation between presentation time & MACE | <i>7</i> 9 |
|----|---|------------|
| | | |

List of Abbreviations

| ACC | American College of Cardiology |
|------------------|---------------------------------------|
| ACS | Acute coronary syndrome |
| AHA | American Heart Association |
| AMI | Acute myocardial infarction |
| ANP | Atrial natriuretic peptide |
| ASE | American Society of Echocardiography |
| ATP | Adenosine Triphosphate. |
| BNP | Brain natriuretic peptide |
| ВР | Blood pressure |
| Ca ²⁺ | Calcium ion |
| CABG | coronary artery bypass grafting |
| CAD | Coronary artery disease |
| CHF | Congestive heart failure |
| СК | Creatine Kinase |
| СОР | cardiac output |
| CRP | C-Reactive protein |
| CTFC | The Corrected TIMI Frame Count |
| cTnI | Troponin I |
| CVS | Cerebrovascular stroke |
| ECG | electrocardiography |
| ESC | European Society of Cardiology |
| EDV | End diastolic volume |
| EF | Ejection fraction |
| ESV | End systolic volume |
| FBS | Fasting Blood Sugar |
| FH | Family history |
| GP | Glycoprotein |
| HDL | High-density lipoprotein |
| HF | Heart failure |
| HTN | Hypertension |
| IHD | Ischemic heart disease |
| LA | Left atrium |
| LAD | Left Anterior descending |
| LBBB | left bundle branch block |
| LCX | Left circumflex artery. |
| LDL | Low density lipoprotein |
| LVEDP | left ventricle end-diastolic pressure |
| LV | Left Ventricle |
| LVEF | left ventricular ejection fraction |
| LVSD | left ventricular systolic dysfunction |
| LVSF | left ventricular systolic function |

| w | |
|---------|--|
| MACE | Major Adverse Cardiovascular end points. |
| MBG | Myocardial Blush Grade |
| MRI | magnetic resonance imaging |
| MVD | Multivessel Disease. |
| PCI | Primary percutaneous intervention |
| PPCI | primary percutaneous coronary intervention |
| PT | Prothrombin Time |
| PTCA | Percutaneous transluminal coronary angioplasty |
| P value | Probability value |
| RBS | Random Blood Sugar. |
| RCA | Right Coronary Artery |
| RR | Relative risk |
| RV | Right ventricle |
| SPECT | single-photon emission computed tomography |
| STEMI | ST segment elevation myocardial infarction |
| TDI | tissue Doppler imaging |
| TFGs | TIMI Flow Grades |
| TIMI | Thrombosis In Myocardial Infarction |
| TL | thrombolysis |
| TMPG | TIMI Myocardial Perfusion Grade |
| UA | Unstable angina |
| Vp | velocity propagation |
| | |

Contents

| Introduction | | 1 | | | | | |
|----------------------|-----------------------------------|-----|--|--|--|--|--|
| Aim of The Wor | k | 5 | | | | | |
| Review of Literature | | | | | | | |
| Chapter I: | Diastolic dysfunction in ACS | 6 | | | | | |
| Chapter II: | Reperfusion After Primary PCI | 21 | | | | | |
| Chapter III: | BNP and Cardiac Biomarkers | 35 | | | | | |
| | | | | | | | |
| Patients & Methods | | | | | | | |
| Results | | 58 | | | | | |
| Discussion | | 80 | | | | | |
| Summary | | 90 | | | | | |
| Limitations of The | study | 91 | | | | | |
| conclusion | | 93 | | | | | |
| References | | 94 | | | | | |
| Arabic Summar | V | 3-1 | | | | | |

Introduction

ST segment elevation myocardial infarction (STEMI) constitutes 40% of all acute myocardial infarctions (AMI), which continues to be a significant public health problem in both developed and developing counties (1).

Primary percutaneous intervention (PCI) is now classified as class I indication in STEMI in the Guidelines of the European Society of Cardiology (ESC) (2).

Reperfusion therapy is the cornerstone of the treatment of patients with acute ST elevation myocardial infarction (STEMI) (3). Many randomized clinical trials have shown that primary percutaneous coronary intervention (PCI) is superior to thrombolytic therapy in the treatment of patients with STEMI (4).

The aim of reperfusion therapy for many years has focused on achieving epicardial artery patency at the site of the occlusive thrombus. It is now possible, through advances in interventional techniques and adjunctive pharmacological treatment, to achieve TIMI (Thrombosis In Myocardial

Infarction) grade 3 epicardial flow (normal) in 95% of patients_(5,6).

Despite this achievement, mortality, although declining, still remains high. This is possibly because despite restoration of TIMI grade 3 flow, 40% of patients do not achieve microvascular flow, which should be the goal of reperfusion therapy (7).

Successful primary PCI within 3–24 hours of the onset of chest pain has been associated with improved LV systolic function at a mean follow-up period of 22 months (8).. Other studies of primary PCI have also reported improved LV systolic function compared to thrombolysis (9).

In acute myocardial infarction (MI), decreasing compliance of the left ventricle is directly associated with prognosis(10). In patients with ST segment elevation MI (STEMI), left ventricular filling pressure increases(11,12).

Early improvement of perfusion after MI will improve left ventricle function and decrease the infarction area, thus decreasing mortality(13,14). The efficacy of reperfusion treatment may be shown indirectly with electrocardiography (ECG), by regression of ST elevation, but there is a need for methods to

demonstrate left ventricle and microvascular function improvement₍₁₅₎.

Primary percutaneous coronary intervention (PCI) is regarded as the best reperfusion model in STEMI. PCI may be used to show hemodynamic changes in the left ventricle or to measure left ventricle end-diastolic pressure (LVEDP) for evaluation of reperfusion efficacy and success.

studies have now demonstrated a robust association between BNP or NT-proBNP and the short- and long-term risk of death across the spectrum of non-ST-elevation ACS,(16-18). including patients without myocardial necrosis or clinical evidence of heart failure. In some patients with ACS, elevated levels of BNP directly reflect the degree of left ventricular dysfunction resulting from acute myocardial infarction. However, the strong association between levels of BNP/NT-proBNP and mortality among patients without measurable myocyte necrosis (i.e., release of cardiac troponin) indicate that the level of BNP may reflect the extent or severity of the ischemic insult, even when irreversible injury has not occurred.(18)

These findings suggest that transient ischemia may induce BNP synthesis and release in proportion to the severity of myocardial ischemia. As such, BNP adds a new dimension to our ability to quantify the consequences of acute myocardial ischemia.



Aim of the work

The aim of this study is to assess the value of LVEDP measurement **before and after restoration of coronary artery patency** in the cath lab, in the setting of primary PCI, in predicting the success of reperfusion among patients with STEMI undergoing primary PCI and correlating the LVEDP change with the peaking of cardiac enzymes,ST segment resolution,chest pain relief, LV dysfunction and with the change in the level of BNP before and after primary PCI.

Chapter 1 Diastolic dysfunctionin Acute Coronary Syndrome

[A] HISTORY:

Left ventricular diastolic function plays an important role in myocardial performance[19]. Events that occur during diastole are crucial to effective systolic function; defects in lusitropy (the ability of the myocytes to relax) have been implicated as an early sign of the development of congestive heart failure[20]. The lusitropic state is influenced by both biochemical and biomechanical events (active relaxation) in addition to the biophysical properties of the heart (passive stiffness)[21].

To demonstrate the importance of assessment of left ventricular end-diastolic pressure (LVEDP) in patients presenting with acute myocardial infarction (STEMI), we will try to focus on the diastolic side of the hemodynamics of the heart.

[B] INTRODUCTION:

As pointed out by Henderson in 1923, diastolic relaxation of the heart is a vital factor and not merely the passive stretching of a rubber bag (22).

Normal LV diastolic function refers to the capacity to fill and maintain stroke volume without a compensatory increase of atrial filling pressure, either in rest or during exercise.

Events that precipitate diastole include both active (intramyocardial) and passive (extramyocardial) mechanisms [21].

Active events that occur within cardiac myocytes include:

- ♦ Ca2+ regulation.
- ♦ myofilament structure and function alteration.
- ♦ neurohormonal activation.

Passive events that influence relaxation include: Changes in early diastolic load and afterload [23].

[C] DEFINITIONS OF DIASTOLE:

1. Physiological diastole:

The term Diastole is derived from two Greek words, to send apart "expand". Physiological diastole commences as left ventricular pressure starts to fall after the peak of ejection phase and extends to the onset of isovolumetric contraction with the term protodiastole being applied to the early part of the relaxation phase-from when aortic flow begins to fall until aortic valve shuts (24).

2. <u>Cellular diastole:</u>

At the cellular level diastole can be considered as beginning when ATP hydrolyses and actin-myosin cross bridges become unlinked allowing for sarcomeric relaxation. This is integrally related to decreasing intracellular concentrations of calcium owing to enhanced sarcoplasmic reuptake of calcium.

Like systole, diastole is an active process. Dysfunction at the cellular level is mediated principally viadecreased ATP hydrolysis and/or impaired uptake of the intracellular calcium. Additionally, in the case of regional ischemia, there may be regional cellular impairment such that the heart ceases to function as a syncytium (24).

3. Mechanical diastole:

Diastole is considered to begin when the pressurewithin the left ventricle begins to fall duringthe isovolumic relaxation phase. This would occur after a significantnumber of myocardial cells had entered cellular diastole andin a metabolically active process. The left ventricular pressurewill continue to fall rapidly, with opening of the mitral valveoccurring when the left ventricular pressure falls below the left atrial pressure (24).

4. Clinical diastole:

In contrast, cardiological diastole is demarcated by the heart sounds and extends from closure to the aortic valve (A2) to the start of the first heart sound (M1) with the term protodiastole being applied to the early phase of rapid filling, the time when third heart sound can be heard (25).

5. Electrocardiographic diastole:

On an electrocardiogram, diastole is the period between the end of the T wave and the beginning of the QRS complex (26).