Non Invasive Evaluation of Ventricular Filling Pressures Using Tissue Doppler Imaging

Thesis

Submitted for the fulfillment of master degree in Cardiology medicine

Presented by Ahmed Shehata Mohamed Ismail M.B, B.Ch.

Supervised by **Prof. Dr. Sherif El Tobgi**

Professor of Cardiology
Faculty of Medicine, Cairo University

Dr. Zeinab Ashour

Professor of Cardiology
Faculty of Medicine, Cairo University

Dr. Karim Said

Lecturer of Cardiology
Faculty of Medicine, Cairo University

Faculty of Medicine
Cairo University
2010

Acknowledgment

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

I would like to express my deepest gratitude and cardinal appreciation to Prof. Dr. **Sherif El Tobgi**, Professor of Cardiology, Faculty of medicine, Cairo University, who kindly supervised and motivated the performance of this work, for his kind guidance and constant encouragement throughout this work.

I am greatly honored to express my sincere appreciation to Dr. **Zeinab Ashour**, Professor of Cardiology, Faculty of medicine, Cairo University, for devoting part of her precious time to help me in the preparation of this work.

I am greatly honored to express my deep thankfulness to Dr. Karim Said, Lecturer of Cardiology, Faculty of medicine, Cairo University, for his patience and meticulous supervision throughout the course of conducting this research. The idea of undertaking this research was originally his and he also conducted most of the echocardiographic studies. He has enriched me with many ideas and has aided me in performing and interpreting the statistics by myself.

I am very grateful to all my staff members and my colleagues in the department of Cardiology, Faculty of medicine, Cairo University, for their help and support throughout the course of the work.

I am most grateful to my family for their patience and everlasting support.

Finally, I want to dedicate this work to the memory of my father who encouraged me in my first steps of life.

Ahmed Shehata

Non Invasive Evaluation Of Ventricular Filling Pressures Using Tissue Doppler Imaging

Ahmed Shehata, Sherif El Tobgi MD, Zeinab Ashour MD, Kareem Said MD Cardiovascular Department- Cairo University

Abstract:

Background: Left and right ventricular diastolic filling pressures are of clinical importance in both patient treatment and prognosis of cardiac and pulmonary diseases.

Purpose: To assess the ability of TDI - as a noninvasive method- to predict left ventricular filling pressures testing the validity of the current indices taking into consideration the controversies surrounding them and trying to drive new indices. In addition we assess its ability to predict right ventricular filling pressure especially in the presence of few data regarding this issue.

Patients and methods: The study population comprised two parallel groups of consecutive patients. The study groups were: Group A. In which conventional Doppler and TDI-derived echocardiographic variables were correlated with invasively measured LVEDP. Group B. In which conventional Doppler and TDI-derived echocardiographic variables were correlated with invasively measured RAP via a central venous catheterization.

Results: In group A. The median age of the patients was 55.5 years, 68% of them were males. Among all conventional and TDI indices measured, all conventional indices (E velocity, A velocity, E DT and E/A) together with both A' velocity and E'/A' showed the strongest correlation with LVEDP (r=0.59, P < 0.001) especially in patients with advanced diastolic dysfunction (r= 0.77, p < 0.01). Multiple regression analysis revealed that E'/A' is the best predictor of LVEDP having the greatest standardized coefficient B (0.4). Using linear regression analysis E'/A' showed the best model to predict the LVEDP (R=0.63, p < 0.001) especially in patients with advanced diastolic dysfunction (R=0.74, p< 0.001). Using ROC analysis, E-DT \leq 158 msec can predict elevated LVEDP (> 15 mm Hg) with 86.4% sensitivity and 84% specificity. In patient with advanced diastolic dysfunction A' velocity \leq 10.5 cm/sec and E'/A' \geq 1.29 can predict elevated LVEDP with specificity of 100% and sensitivity of 93.8% and 70% respectively. In group B. The median age of the patients was 50 years, 76% of them were males. Regression analysis showed that E' velocity, IVRT, S duration and A velocity were the most important predictors of RAP. ROC analysis revealed that $E/E' \geq 3$ is associated with 89% sensitivity and 100% specificity to detect RAP > 10 mmHg and IVRT \leq 66.5 msec was associated with 79% sensitivity and 100 % specificity to detect RAP > 10 mmHg.

Conclusions: Among conventional and tissue Doppler variables including the E/E' ratio, E'/A' ratio is the best index to estimate LVEDP especially in patients with advanced diastolic dysfunction (grade II and III). Both tricuspid E/E' ratio and IVRT are useful Doppler indices for non-invasive estimation of RAP.

Key words: Filling pressure, Tissue Doppler, Diastolic function

Contents

| Item List of abbreviations | Page I |
|--|-----------|
| List of tables | IV |
| List of figures | VI |
| Introduction | 1 |
| Aims of the work | 2 |
| Review of literature | 3 |
| Chapter 1: Physiology of filling pressure | 3 |
| • Chapter 2: Determinants of filling pressure | 25 |
| Chapter 3: Invasive estimation of left ventricular filling pressure | 47 |
| Chapter 4: Invasive estimation of right ventricular filling pressure | 56 |
| Chapter 5: Non invasive evaluation of ventricular filling pressure | 68 |
| patients and methods | 77 |
| Results | 85 |
| Discussion | 121 |
| conclusion | 134 |
| Study limitations | 135 |
| Recommendations | 136 |
| Summary | 137 |
| References | 139 |
| Arabic summary | 1-2 |

LIST OF ABBREVIATIONS

2-D TDI: Two dimensional tissue Doppler imaging

A: Late diastolic wave
A': Late annular motion

ACE: Angiotensin-converting enzyme

ACS: Acute coronary syndrome ANOVA: Analysis of variance Ar: Reverse pulmonary flow wave

AUC: Area under the curve

BP: Blod pressure

CAD: Coronary artery disease CHF: Congestive heart failure

CI: Confidence interval

CKD: Chronic kidney disease CVP: Central venous pressure D: Pulmonary vein diastolic wave DBP: Diastolic blood pressure

DM: Diabetes mellitus

dP/dt: Rate of pressure change

dP/dtmin: Minimum rate of pressure change

DR: diastolic flow reversal

E/A: Ratio between early diastolic and late diastolic inflow velocity

E/E': Ratio between early diastolic wave inflow velocity and early diastolic annular motion velocity

E/Pv: Ratio between early diastolic wave amplitude of transmitral flow and its propagation velocity into the left ventricle

E: early diastolic inflow velocity

E'/A': Ratio between early diastolic wave amplitude of annulus movement and late diastolic wave amplitude of annulus movement

E': early annular motion velocity

E'Acc rate: early annular wave acceleration rate
E'Acc Time: early annular wave acceleration time
E'Decel rate: early annular wave deceleration rate
E'Decel Time: early annular wave deceleration time

ECG: Electrocardiogram

E-DT: E wave deceleration time

EF: Ejection fraction

HCM: Hypertrophic cardiomyopathy

HVF: hepatic vein flow

IPPV: Intermittent positive pressure ventilation

IR: Impaired relaxation IVC: inferior vena cava

IVCT: Isovolumic contraction time

IVR: Isovolumic relaxation

IVRT: Isovolumic relaxation time

KPa: Kilo Pascal (standard unit of pressure)

LAP: Left atrial pressure

LV: Left ventricle

LVDP: Left ventricular diastolic pressure

LVEDD: Left ventricular end diastolic dimensions LVEDP: Left ventricular end diastolic pressure

LVEF: Left ventricular ejection fraction LVFP: left ventricular filling pressure

MAP: Mean arterial pressure MFV: mitral flow velocity

MR: Mitral regurge NS: Non-significant

NYHA: New York Heart Association Functional Classification

P wave: Propagation wave P∞: pressure asymptote

Pa: Pulmonary alveolar pressure

PA: Pulmonary artery

PAC: pulmonary artery catheterization

PAO: Aortic pressure

PAOP: pulmonary artery occlusion pressure

PAP: Pulmonary artery pressure

PAWP: pulmonary artery wedge pressure Pcap: Pulmonary capillary filtration pressure PCWP: Pulmonary capillary wedge pressure

PEEP: Positive end expiratory pressure

PFR: peak filling rate

P_{IP}: Intrapleural pressure

P_{LV}: Left ventricular pressure **PN**: Psedonormalization normal

P_{PA}: Pulmonary artery pressure **P_{RV}:** Right ventricular pressure

PTCA: percutaneous transluminal coronary angioplasty

PV: Propagation velocity

Pv: Pulmonary venous pressure

PVF: pulmonary vein flow

PW-TDI: Pulsed wave - tissue Doppler imaging

QAO: Aortic flow

Q_{PA}: Pulmonary artery flow

RA: Right atrium

RAP: Right atrial pressure

RF: Restrictive filling

ROC: Receiver Operator Characteristics

RV: Right ventricle

RWMA: Regional wall motion abnormality

S wave: Systolic wave

SBP: Systolic blood pressure

SF: Systolic fraction

SGC: Swan Ganz catheter SR: systolic flow reversal

Std: Standard

Sv O2: Venous O2 saturation SVC: Superior vena cava

t: time

T: time constant of isovolumic relaxation

Tau: Time constant of left ventricular pressure decay

TD: Tissue Doppler

TDI: tissue Doppler imaging TDI: Tissue Doppler imaging TPFR: time to peak filling rate

TR: Tricuspid regurge V₀: Equilibrium volume α: elastic parameter β: elastic parameter

 γ : viscoelastic parameter

ε: Lagrangian strain

έ: strain rate

σ: stress

List of tables

| Table 1 | Disorders involving abnormalities in diastolic left ventricular filling | 15 |
|----------|---|--------|
| Table 2 | Lists of factors affecting the CVP | 59 |
| Table 3 | Parameters for identification of patients with an elevated left ventricular filling pressure. | 72 |
| Table 4 | Proposed formulas for the estimation of left ventricular filling pressure | 72 |
| Table 5 | Intraclass correlation coefficient | 84 |
| Table 6 | Baseline clinical and hemodynamic characteristics | 86 |
| Table 7 | Echocardiography: 2- dimensional and color flow imaging | 86 |
| Table 8 | Echocardiography: Conventional Doppler and TDI-derived measurements | 87 |
| Table 9 | Linear correlation between LVEDP and Doppler indices | 88 |
| Table 10 | Correlation between LVEDP and Doppler indices according to left ventricular systolic function | 91 |
| Table 11 | Correlation between LVEDP and Doppler indexes in patients with RWMA | 92 |
| Table 12 | Comparison between patients with and without elevated LVEDP | 93, 94 |
| Table 13 | Comparison between patients with grade I versus grades II-III LV diastolic dysfunction | 97, 98 |
| Table 14 | Relations between grades of LV diastolic dysfunction and Doppler indices by ANOVA | 100 |
| Table 15 | Multiple regression analysis | 102 |
| Table 16 | Linear regression analysis | 103 |
| Table 17 | ROC analysis to predict elevated LVEDP (> 15 mmHg) in all patients | 105 |

List of tables

| Table 18 | ROC analysis to predict elevated LVEDP (> 15 mmHg) in patients with grades II and III LV diastolic dysfunction | 106 |
|----------|--|----------|
| Table 19 | Baseline clinical and hemodynamic characteristics | 108 |
| Table 20 | Echocardiography: Conventional Doppler and TDI-derived measurements | 109 |
| Table 21 | Linear regression correlation between RAP and Doppler indices | 110 |
| Table 22 | Comparison between patients with and without elevated RAP | 112, 113 |
| Table 23 | Results of stepwise multiple regression analysis | 115 |
| Table 24 | Coefficients of model 1, 2 and 6 | 115 |
| Table 25 | Confidence interval of model 2 and 6 | 116 |
| Table 26 | List of equation | 116 |
| Table 27 | ROC analysis for prediction of elevated RAP | 119 |

List of Figures

| Figure 2 Plots of LV pressure versus length in normal and ischemic myocardial segments before and after rapid cardiac pacing in patient with single vessel coronary disease LV time-activity curves and schematic representations of these curves in patient with CAD before and after coronary angioplasty. PFR improved from 1.1 to 2.3 EDV/sec after PTCA, and TPFR decreased from 186 to 166 msec, without change in baseline heart rate or ejection fraction. Figure 4 Representations of ventricular pressure-volume relations in different forms of heart failure. Figure 5 Several different factors affect the diastolic pressure during diastole, with different factors being important at different times Figure 6 The rate, but not the extent, of left ventricular relaxation is slowed during reoxygenation after fifteen minutes of hypoxia. Figure 7 Pressure-volume relations of the heart and pericardium, together and alone. Figure 8 Hemodynamic response to the application and removal of 12 cm H2O positive end-expiratory pressure. Two viscoelastic models used to describe muscle, the three-element model and the two-element (or Voigt) model (Panel A). Panel B shows the stress relaxation response of these two models, elicited by an imposed sudden, constant change in length (stress relaxation experiment). Panel C shows the corresponding results for creep experiments, when the change in length is measured in response to a sudden, constant change in externally imposed force Figure 10 Prigical waveform progression as the PAC floats through the cardiac chambers. Monitoring these waveforms tells the anesthesiologists where in the heart the catheter is as it advances Figure 11 Physiologic lung zones. For pulmonary capillary wedge pressure to be reliable, the catheter tip must lie in zone 3. | | | Page |
|---|-----------|--|------|
| Figure 2 segments before and after rapid cardiac pacing in patient with single vessel coronary disease LV time-activity curves and schematic representations of these curves in patient with CAD before and after coronary angioplasty. PFR improved from 1.1 to 2.3 EDV/sec after PTCA, and TPFR decreased from 186 to 166 msec, without change in baseline heart rate or ejection fraction. Figure 4 Representations of ventricular pressure-volume relations in different forms of heart failure. Figure 5 Several different factors affect the diastolic pressure during diastole, with different factors being important at different times Figure 6 The rate, but not the extent, of left ventricular relaxation is slowed during reoxygenation after fifteen minutes of hypoxia. Figure 7 Pressure-volume relations of the heart and pericardium, together and alone. Figure 8 Hemodynamic response to the application and removal of 12 cm H2O positive end-expiratory pressure. Two viscoelastic models used to describe muscle, the three-element model and the two-element (or Voigt) model (Panel A). Panel B shows the stress relaxation response of these two models, elicited by an imposed sudden, constant change in length (stress relaxation experiment). Panel C shows the corresponding results for creep experiments, when the change in length is measured in response to a sudden, constant change in externally imposed force Figure 10 Typical waveform progression as the PAC floats through the cardiac chambers. Monitoring these waveforms tells the anesthesiologists where in the heart the catheter is as it advances Figure 11 Physiologic lung zones. For pulmonary capillary wedge pressure to be reliable, the catheter tip must lie in zone 3. Figure 12 Central venous pressure waveform from a ventilated patient (bottom) with | Figure 1 | Wiggers diagram | 8 |
| Figure 3 patient with CAD before and after coronary angioplasty. PFR improved from 1.1 to 2.3 EDV/sec after PTCA, and TPFR decreased from 186 to 166 msec, without change in baseline heart rate or ejection fraction. Figure 4 Representations of ventricular pressure-volume relations in different forms of heart failure. Figure 5 Several different factors affect the diastolic pressure during diastole, with different factors being important at different times Figure 6 The rate, but not the extent, of left ventricular relaxation is slowed during reoxygenation after fifteen minutes of hypoxia. Figure 7 Pressure-volume relations of the heart and pericardium, together and alone. Figure 8 Hemodynamic response to the application and removal of 12 cm H2O positive end-expiratory pressure. Two viscoelastic models used to describe muscle, the three-element model and the two-element (or Voigt) model (Panel A). Panel B shows the stress relaxation response of these two models, elicited by an imposed sudden, constant change in length (stress relaxation experiment). Panel C shows the corresponding results for creep experiments, when the change in length is measured in response to a sudden, constant change in externally imposed force Figure 10 Typical waveform progression as the PAC floats through the cardiac chambers. Monitoring these waveforms tells the anesthesiologists where in the heart the catheter is as it advances Figure 11 Physiologic lung zones. For pulmonary capillary wedge pressure to be reliable, the catheter tip must lie in zone 3. | Figure 2 | segments before and after rapid cardiac pacing in patient with single vessel | 10 |
| heart failure. Figure 5 Several different factors affect the diastolic pressure during diastole, with different factors being important at different times Figure 6 The rate, but not the extent, of left ventricular relaxation is slowed during reoxygenation after fifteen minutes of hypoxia. Figure 7 Pressure-volume relations of the heart and pericardium, together and alone. Figure 8 Hemodynamic response to the application and removal of 12 cm H2O positive end-expiratory pressure. Two viscoelastic models used to describe muscle, the three-element model and the two-element (or Voigt) model (Panel A). Panel B shows the stress relaxation response of these two models, elicited by an imposed sudden, constant change in length (stress relaxation experiment). Panel C shows the corresponding results for creep experiments, when the change in length is measured in response to a sudden, constant change in externally imposed force Figure 10 Typical waveform progression as the PAC floats through the cardiac chambers. Monitoring these waveforms tells the anesthesiologists where in the heart the catheter is as it advances Figure 11 Physiologic lung zones. For pulmonary capillary wedge pressure to be reliable, the catheter tip must lie in zone 3. Figure 12 Central venous pressure waveform from a ventilated patient (bottom) with | Figure 3 | patient with CAD before and after coronary angioplasty. PFR improved from 1.1 to 2.3 EDV/sec after PTCA, and TPFR decreased from 186 to 166 msec, | 12 |
| different factors being important at different times The rate, but not the extent, of left ventricular relaxation is slowed during reoxygenation after fifteen minutes of hypoxia. Figure 7 Pressure-volume relations of the heart and pericardium, together and alone. Figure 8 Hemodynamic response to the application and removal of 12 cm H2O positive end-expiratory pressure. Two viscoelastic models used to describe muscle, the three-element model and the two-element (or Voigt) model (Panel A). Panel B shows the stress relaxation response of these two models, elicited by an imposed sudden, constant change in length (stress relaxation experiment). Panel C shows the corresponding results for creep experiments, when the change in length is measured in response to a sudden, constant change in externally imposed force Figure 10 Typical waveform progression as the PAC floats through the cardiac chambers. Monitoring these waveforms tells the anesthesiologists where in the heart the catheter is as it advances Figure 11 Physiologic lung zones. For pulmonary capillary wedge pressure to be reliable, the catheter tip must lie in zone 3. Figure 12 Central venous pressure waveform from a ventilated patient (bottom) with | Figure 4 | | 13 |
| Figure 7 Pressure-volume relations of the heart and pericardium, together and alone. Figure 8 Hemodynamic response to the application and removal of 12 cm H2O positive end-expiratory pressure. Two viscoelastic models used to describe muscle, the three-element model and the two-element (or Voigt) model (Panel A). Panel B shows the stress relaxation response of these two models, elicited by an imposed sudden, constant change in length (stress relaxation experiment). Panel C shows the corresponding results for creep experiments, when the change in length is measured in response to a sudden, constant change in externally imposed force Figure 10 Typical waveform progression as the PAC floats through the cardiac chambers. Monitoring these waveforms tells the anesthesiologists where in the heart the catheter is as it advances Figure 11 Physiologic lung zones. For pulmonary capillary wedge pressure to be reliable, the catheter tip must lie in zone 3. Central venous pressure waveform from a ventilated patient (bottom) with | Figure 5 | | 25 |
| Figure 8 Hemodynamic response to the application and removal of 12 cm H2O positive end-expiratory pressure. Two viscoelastic models used to describe muscle, the three-element model and the two-element (or Voigt) model (Panel A). Panel B shows the stress relaxation response of these two models, elicited by an imposed sudden, constant change in length (stress relaxation experiment). Panel C shows the corresponding results for creep experiments, when the change in length is measured in response to a sudden, constant change in externally imposed force Figure 10 Typical waveform progression as the PAC floats through the cardiac chambers. Monitoring these waveforms tells the anesthesiologists where in the heart the catheter is as it advances Figure 11 Physiologic lung zones. For pulmonary capillary wedge pressure to be reliable, the catheter tip must lie in zone 3. | Figure 6 | | 30 |
| Figure 9 A & Two viscoelastic models used to describe muscle, the three-element model and the two-element (or Voigt) model (Panel A). Panel B shows the stress relaxation response of these two models, elicited by an imposed sudden, constant change in length (stress relaxation experiment). Panel C shows the corresponding results for creep experiments, when the change in length is measured in response to a sudden, constant change in externally imposed force Typical waveform progression as the PAC floats through the cardiac chambers. Monitoring these waveforms tells the anesthesiologists where in the heart the catheter is as it advances Figure 11 Physiologic lung zones. For pulmonary capillary wedge pressure to be reliable, the catheter tip must lie in zone 3. Central venous pressure waveform from a ventilated patient (bottom) with | Figure 7 | Pressure-volume relations of the heart and pericardium, together and alone. | 38 |
| Figure 9 A & relaxation response of these two models, elicited by an imposed sudden, constant change in length (stress relaxation experiment). Panel C shows the corresponding results for creep experiments, when the change in length is measured in response to a sudden, constant change in externally imposed force Figure 10 Typical waveform progression as the PAC floats through the cardiac chambers. Monitoring these waveforms tells the anesthesiologists where in the heart the catheter is as it advances Figure 11 Physiologic lung zones. For pulmonary capillary wedge pressure to be reliable, the catheter tip must lie in zone 3. | Figure 8 | | 40 |
| Figure 10 chambers. Monitoring these waveforms tells the anesthesiologists where in the heart the catheter is as it advances Figure 11 Physiologic lung zones. For pulmonary capillary wedge pressure to be reliable, the catheter tip must lie in zone 3. Figure 12 Central venous pressure waveform from a ventilated patient (bottom) with | _ | the two-element (or Voigt) model (Panel A). Panel B shows the stress relaxation response of these two models, elicited by an imposed sudden, constant change in length (stress relaxation experiment). Panel C shows the corresponding results for creep experiments, when the change in length is | 43 |
| reliable, the catheter tip must lie in zone 3. Figure 12 Central venous pressure waveform from a ventilated patient (bottom) with | Figure 10 | chambers. Monitoring these waveforms tells the anesthesiologists where in the | 50 |
| | Figure 11 | | 51 |
| | Figure 12 | | 57 |

List of figures

| Figure 13 | Ventricular function and venous return curves | 60 |
|--------------------------------------|---|-----|
| Figure 14 | Ventricular diastolic pressure volume curve | 62 |
| Figure 15 A &15B | A. A sample volume was placed 2-3 cm from inferior vena cava B. Pulsed wave Doppler recording of hepatic vein flow velocities in a normal subject. Systolic velocity (S) is usually graeter than diastolic velocity (D), with no prominent reversal velocities (SR and DR). Both velocities are graeter with inspiration than with expiration | 75 |
| Figure 16 A &16 B & 16 C &16 D | Hepatic vein Doppler recording from patients with restriction, pulmonary hypertension and severe tricuspid regurge A. restrictive cardiomyopathy. Systolic (S) forward flow velocity is smaller than diastolic (D) fprward flow velocity. Inspiratoy (Insp) diastolic flow reversal is larger than expiratory (Exp) diastolic flow reversal B. the recording of constriction is similar to that of restriction except that Exp diastolic flow reversal is larger than Insp diastolic reversal C. pulmonary hypertyension. Diastolic flow reversal (arrows) does not change much with respiration D. Severe tricuspid regurge with late systolic flow reversal (arrow) | 75 |
| Figure 17 | Example of Pulsed wave Doppler of the mitral flow | 79 |
| Figure 18 | Example of PW-TDI of the lateral tricuspid annulus | 80 |
| Figure 19 | Scatter plot showing the correlation between LVEDP and the following E velocity, A velocity, E-DT and E/A | 89 |
| Figure 20 | Scatter plot showing the correlation between LVEDP both A' velocity and E'/A' | 90 |
| Figure 21 | Correlation between E'/A' and LVEDP in patients with grade II-III diastolic dysfunction | 92 |
| Figure 22 | E-DT in patients with and without elevated LVEDP | 95 |
| Figure 23 | E'/A' in patients with and without elevated LVEDP | 95 |
| Figure 24 | E/E' ratio in patients with grade I versus grades II-III LV diastolic dysfunction | 99 |
| Figure 25 | E'/A' in patients with grade I versus grades II-III LV diastolic dysfunction | 99 |
| Figure 26 | Relation between LVEDP and different grades of LV diastolic dysfunction by ANOVA | 101 |
| Figure 27 | Relation between E/E' velocity and different grades of LV diastolic dysfunction by ANOVA | 101 |

| Figure 28 | Relation between E'/A' velocity and different grades of diastolic dysfunction by ANOVA | 102 |
|-----------|---|-----|
| Figure 29 | Linear regression between E'/A' and LVEDP in all patients | 103 |
| Figure 30 | Linear regression between E'/A' and LVEDP in patient with grade II and III diastolic dysfunction | 104 |
| Figure 31 | Example of PW-TSDI of the lateral mitral annulus | 104 |
| Figure 32 | ROC curve analysis of E-DT for prediction of LVEDP > 15 mmHg in all patients | 105 |
| Figure 33 | ROC curve analysis of A' wave for the prediction of LVEDP >15 mmHg in patient with grade II and III diastolic dysfunction | 106 |
| Figure 34 | LVEDP in patients with E/E' > 12 versus those with E/E' < 8 | 107 |
| Figure 35 | Linear regression between RAP and E/E' | 111 |
| Figure 36 | Linear regression between RAP and IVRT | 111 |
| Figure 37 | IVRT in patients with and without elevated RAP | 114 |
| Figure 38 | E/E' in patients with and without elevated RAP | 114 |
| Figure 39 | Bland-Altman plot of Doppler derived RAP pressure (using E/E') versus catheter derived RAP | 117 |
| Figure 40 | Bland-Altman plot of Doppler derived RAP pressure (using E/E' & IVRT) | 117 |
| Figure 41 | Example of PW-TDI of the lateral tricuspid annulus | 118 |
| Figure 42 | ROC curve analysis for the prediction of RAP > 10 mmHg for IVRT | 120 |
| Figure 43 | ROC curve analysis for the prediction of RAP > 10 mmHg for E/E' | 120 |

Introduction

Estimation of ventricular filling pressure is important in the assessment and management of cardiac patients especially under critical situations. Importantly, determination of ventricular filling pressure helps in the early initiation of appropriate therapy towards precise homodynamic goals.

Central venous pressure (CVP) provides an estimate of the right atrial pressure and is used as a marker of cardiac preload.^{1,2} Classically, CVP is measured invasively at the junction of superior vena cava and the right atrium.³ Similarly, invasive methods are the golden standard to measure left ventricular filling pressure This is performed using pulmonary artery catheter (in the wedge position) or via left ventricular catheterization.^{1,2,3} It is obvious that these invasive procedures are expensive, need experienced hands, and are not without complications (e.g., pneumothorax, nerve injury, air-embolism, thromboembolism, infection).^{1,2,3}

Various echocardiographic indices of transmitral and pulmonary vein flow have been combined to predict left atrial pressure noninvasively and, recently, tissue Doppler imaging (TDI) has showed promising role in this regard. TDI is a relatively new image modality that is based on quantification of myocardial velocities by consecutive phase shifts of the ultrasound signals reflected from the moving myocardium. ^{4,5} By combining transmitral E wave velocities with the velocity of motion of the lateral mitral annulus (Ea, which is a preload independent parameter), a reasonable estimate of left atrial pressure can be obtained. Naguel et al⁶ found that an E/E' ratio of greater than 10 correlated well with the mean pulmonary capillary wedge pressure (PCWP) of greater than 15 mmHg. Omnen et al⁷ confirmed this observation in another study. In this study, a ratio of (E/Ea) greater than 15 was identified in patients with elevated left ventricular end-diastolic pressure (LVEDP) greater than 12 mmHg. On the other hand an E/Ea ratio less than 8 accurately predicted normal LVEDP.

Although there are currently few data available, the previously mentioned early and well conducted studies are highly suggestive that the combination of TDI and mitral inflow velocities can be used for non invasive assessment of the LV filling pressure. On the other hand, and to the best of our knowledge, there is sparse data regarding the accuracy of TDI and tricuspid inflow velocities for the noninvasive estimation of right ventricular filling pressure.

Aim of the work

1-To assess the utility of several conventional Doppler and TDI parameters in the estimation of left ventricular filling pressures, trying to derive a new index that may be useful for the non-invasive estimation of left ventricular filling pressure.

2-To derive an echocardiographic index for estimating RAP using tricuspid annular velocity and inflow as assessed by conventional and tissue Doppler echocardiography.

Chapter 1

Physiology of filling pressure

Diastole, that portion of the cardiac cycle that begins with isovolumic relaxation and ends with mitral valve closure, results in ventricular filling and involves both active (energy dependent) and passive processes. The interactions between active processes (myocardial relaxation) that primarily influence early ventricular filling and passive processes, such as loading conditions, myocardial compliance, and valvular disease, are complex.⁸

Clinical methods to assess ventricular filling include cardiac catheterization, radionuclide angiography, and echocardiography. Any measurements of diastolic function must be made with an understanding of the determinants of ventricular filling and the limitations of the diagnostic test.

Many cardiac disorders are characterized by elevated pulmonary venous pressures in the face of normal systolic ventricular function, which suggests a primary abnormality of diastolic function .Abnormalities in diastolic function have been observed in coronary artery disease, congestive heart failure (with and without systolic dysfunction), hypertrophic cardiomyopathy, hypertension, and in healthy elderly subjects. Identification of these abnormalities may be useful clinically, particularly in patients with symptoms of heart failure and normal systolic function .Data are not available to determine the optimal therapy for such patients, although evidence suggests that calcium channel blockers, beta blockers, and agents that reverse myocardial hypertrophy may be useful.⁸

Physiology of Diastole

Diastole, a term derived from two Greek words meaning "to send apart," can be defined as the portion of the cardiac cycle that begins with isovolumic ventricular relaxation and ends with cessation of mitral inflow.

It is useful to define "normal" and "abnormal" diastolic function. In as much as normal heart function is concerned with the generation of adequate cardiac output at acceptable venous and arterial pressures, normal diastolic function may be defined as the level of ventricular filling needed to generate a cardiac output that is commensurate with