Echocardiographic indices of Acute and Chronic Left Ventricular Filling Pressure and its' Relation to Ventricular Dilation After Acute Myocardial Infarction

Thesis

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LIST OF ABBREVIATIONS

DT: Deceleration time
LV: Left ventricle
LAVi: index of left atrial volume
MI: Myocardial infarction
Ca++: Calcium
ATP: Adenosine triphosphate
SR: Sarcoplasmic reticulum
PVd: Diastolic peak velocity
PVs: Systolic peak velocity
LVEDP: Left ventricular end diastolic pressure
LA: Left atrium
IC: Isometric contraction
IR: Isometric relaxation
TVI: Tissue velocity imaging
LVH: Left ventricular hypertrophy
HF: Heart failure
STEMI: ST-segment elevation myocardial infarction
EF: Ejection fraction
WMS: Wall motion score
IHD: Ischemic heart disease
CAD: Coronary artery disease
CD: conventional Doppler
TDI: Tissue Doppler imaging

Introduction

After acute myocardial infarction enlargement of the left ventricle may occur, even after successful percutaneous intervention ***(Bolognese et al., ٢٠٠٢).*** Indeed, significant LV dilation is observed in almost one third of patients despite patency of the infarct-related coronary artery ***(Bolognese et al., ٢٠٠٢).*** Such LV remodeling is an important determinant of outcome in this setting-predicting an increased risk of progressive heart failure and death ***(Bolognese et al., ٢٠٠٢ and St John Sutten et al., ١٩٩٤).***

The mechanism of LV remodeling is complex and remain to be fully defined. It is, however, recognized that the process is associated with important changes in LV diastolic properties, such as compliance and relaxation, secondary to scar formation and compensatory hypertrophy of non infracted tissue ***(Cerisano et al., ١٩٩٩).*** These changes may be associated with increased LV filling pressure that may, in turn, contribute to the remodeling process.

Certainly, prior studies have suggested that an abbreviated Doppler-derived deceleration time (DT) of early transmitral flow is associated with an increased risk of LV dilation after AMI ***(Temporelli et al., ٢٠٠٤).*** However, the correlation between DT and LV filling (or pulmonary capillary wedge) pressure depends on underling LV systolic function ***(Ommen et al., ٢٠٠٠).*** To more accurately identify patients

with elevated LV filling pressures transmitral flow velocities and DT have been combined with other relatively preload-independent indicators of diastolic function. Of these, Doppler tissue imaging of early mitral annulus velocity (é) appears to be of particular value ***(Ommen et al., ٢٠٠٠ and Dokainsh et al., ٢٠٠٤).*** The E/é ratio is well correlated with LV filling pressure ***(Ommen et al., ٢٠٠٠ and Dokainsh et al., ٢٠٠٤)*** and elevated levels are associated with increased mortality after AMI ***(Hillis et al., ٢٠٠٤).*** In addition the volume of LA indexed to body surface (LAVi), is thought to reflect chronic LV filling pressure and also predicts mortality after infarction ***(Moller et al., ٢٠٠٣ and Beinart et al., ٢٠٠٤).***

However, the relationship between indicators of acute and chronic LV filling pressure and remodeling after AMI has not been defined. The current study addresses this issue. Specifically, we aimed to define whether E/é or LAVi predicted LV dilation in patients with successful reperfusion after AMI.

Aim of the Work

To assess the relationship between echocardio-graphic indicators of acute and chronic LV filling pressure, and its relation to LV dilation.

Diastolic Function

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.

Definition of Diastole:

The tem diastole is derived from two Greek words, to send and apart. Physiological diastole commences as left ventricular pressure starts to fall after the peak of ejection phase and extends to the onset of isovolumic 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.

In contrast, cardiological diastole is demarcated by the heart sounds and extends from closure of the aortic valve (A_2) to the start of the first heart sound (M_1) with the term protodiastole being applied to the early phase of rapid filling, the time when third heard can be heart ***(Opie, ٢٠٠١).***

On an electrocardiogram, diastole is the period between the end of the T wave and the beginning of the QRS complex ***(Peterson, ٢٠٠٠).***

Phase of Ventricular Diastole:

Diastolic function of the heart is a complex sequence of numerous interrelated events, the pathophysiology of which is incompletely understood.

Haemodynamically, diastole can be divided into

Four distinct phases:

١. Isovolumic relaxation phase:

In extends from aortic valve closure until left ventricular pressure falls below left atrial pressure when the mitral valve opens and filling phase starts. During this phase, there is a steep exponential fall in ventricular pressure with a constant ventricular volume. This phase is energy-dependent, requiring ATP for Ca reuptake by the SR, which is an active process.

٢. Rapid filling phase:

It extends from mitral valve opening to the onset of plateau of LV volume, during this phase, left ventricular pressure continues to decrease despite a substantial increase in left ventricular volume because of further active relaxation and a suction effect. It provides most of ventricular filling in early diastole.

٣. Slow filling phase (diastasis):

It extends from onset of plateau of LV volume to the start of atrial systole. During this phase, the pressures in the left atrium and left ventricle filling equalize, and filling of LV occurs during the mid-diastole by the inertial forces.

٤. Atrial systole pahse (final booster phase):

It accounts for about ١٥ percent of the total filling ***(Opie, ٢٠٠١).*** However, because of the complex interactions among

numerous interrelated events, including the influence of the preceding systolic contraction, it has been difficult to apply the above classic approach clinically. A proposed model that can be used clinically is to consider the cardiac cycle in terms of systolic contraction, relaxation and diastolic filling, a modification of the model proposed by Brusteart et al. ١٩٨٩ ***Nishimura and Tajik, ١٩٩٧).***

Contraction encompasses isovlumetric contraction and the first half of ejection with a transition into relaxation. Which consists of a large portion of the second half of ejection, isovolumetric relaxation and the rapid filing phase. Diastolic filling is the period in which the ventricle fills with blood from the left atrium (from the onset of mitral valve opening to mitral valve closure). The early phase of diastolic filling coincides with and is dependent on continued ventricular relaxation ***(Brustsaert and Sys, ١٩٨٩).***

Determinates of Ventricular Filling:

Numerous factors determine how the ventricle fills with blood during diastole. Each of these factors including ventricular relaxation, diastolic suction, erectile coronary effect.
Viscoelatic forces of the myocardium pericardial restraint, ventricular interaction and atrial contribution is interrelated to the others in a complex sequence of events ***(Grossman and McLaurin, ١٩٧٦).***

The most important determinants of left ventricular filling are:

Ventricular relaxation:

It is a complex energy-dependent process during which the contractile elements are inactivated and the myofibrils return to their original (pre-contraction) length. In a normal heart, it begins during midsystole and continues throughout the first third of diastolic filling, it does not normally effect diastasis or atrial filling ***(Little and Downes, ١٩٩٠).***

There is triple control of relaxation in the intact heart consisting of inactivation, the load on the left ventricle and the non-uniformity of relaxation. Delayed inactivation, diminished load dependence and increased non-uniformity impair relaxation and diminish the mechanical efficiency of the heart.
This result in a decrease in the ability of the left ventricle to fill in early diastole, that is the rapid filling phase. There is usually a compensatory increase in filling with atrial contraction ***(Brustsaert et al., ١٩٨٤).***

Factors that slow myocardial relaxation include advanced age, myocardial ischaemic, LV hypertrophy, hypothyroidism, dilated cardiomyopathy, and intracellular calcium overload ***(Nishimura et al., ١٩٨٩).***

Diastolic suction:

Whether the LV suction-effect of active relaxation-could increase the pressure gradient from left atrium to LV during the early phase of rapid filling remains controversial, although well supported by the data.

The proposed mechanism of sucking is a follow: when the end-systolic volume is less than the equilibrium volume, the

shortened muscle fibers and collagen matrix may act as a compressed spring to generate recoil forces in diastole.

Thus, LV relaxation depends on early diastolic release of elastic recoil that is accumulated during systole as well as on calcium reuptake by the SR during early diastole in myocytes ***(Gilbert and Glantz, ١٩٨٩).***

Effective LV chamber compliance or stiffness:

The "effective operating chamber compliance" describes the passive properties of the left ventricle during blood flow across the mitral valve. Several complex interactions occur during this period, including the continued effect of ventricular relaxation, diastolic suction, passive filling, pericardial restraint, ventricular interaction and visoelastic forces of the myocardium ***(Appleton and Hatle, ١٩٩٦).***

Following completeness of the active decay of systolic tension, the passive filling properties of LV and strength of atrial contraction influence late filling and determine the LV end-diastolic pressure and volume ***Grossman and McLaurin, ١٩٧٦).***

"Effective operating camber compliance", defined as the change in volume over the change in pressure during diastolic filling, is used to provide a clinical assessment of passive left ventricular properties. It is generally measured in the relaxed ventricle at end diastole ***(Ohno et al., ١٩٩٤).***

The compliance of the left ventricle, reflected in the end-diastolic pressure-volume relationship, is altered in a variety of cardiac disorders. A decrease in chamber compliance can be caused by: (١) a shift of the curve upward and to the left because