



شبكة المعلومات الجامعية

بِسْمِ اللَّهِ الرَّحْمَنِ الرَّحِيمِ





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# شبكة المعلومات الجامعية

## التوثيق الالكتروني والميكرو فيلم

# جامعة عين شمس

التوثيق الالكتروني والميكرو فيلم

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# بعض الوثائق الأصلية تالفة



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بالرسالة صفحات  
لم ترد بالأصل

**THE USE OF COLORED M-MODE  
DOPPLER ECHOCARDIOGRAPHY IN  
ASSESSMENT OF LEFT VENTRICULAR  
DIASTOLIC FUNCTION**

**Thesis**

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*By*

***Sherif Moussa Ibrahim***

(M.B.B.CH.)

Faculty of Medicine  
University of Alexandria

2000

B 7111

# **SUPERVISORS**

## **Professor Dr. Tarek Hussein El-Badawy**

Professor of internal Medicine,  
Faculty of Medicine,  
University of Alexandria.

## **Dr. Tarek Hussein El – Zawawy**

Assistant Professor of Cardiology  
Faculty of Medicine,  
University of Alexandria.

## **Dr. Mohamed Ayman Abdel Moneim Abdel Hai**

Assistant Professor of Cardiology  
Faculty of Medicine,  
University of Alexandria

## **Dr. Salah Mohamed El-Tahhan**

Lecturer of Cardiology,  
Faculty of Medicine,  
University of Alexandria

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# Introduction



## **Introduction**

### **Left ventricular diastolic performance**

#### **Definition**

Normal diastolic L.V. performance can be defined as the amount of filling of the L.V. necessary to produce a cardiac output commensurate with the body's needs at a mean pulmonary venous pressure of less than 12 mm Hg.

#### **Phases of diastole:**

**Diastole can be divided into Five phases:**

- Proto diastolic phase:
- Isovolumic relaxation
- Early rapid filling
- Diastasis
- Atrial systole.

#### **Proto diastolic phase:**

It is the period between the end of ventricular systole and the closure of semilunar valves. The ventricular pressure begins to drop rapidly and become lower than the aortic. The regurgitation of blood from the aorta into the ventricle is prevented by the sudden closure of the aortic valve at the end of this phase.

The sudden closure of the aortic valve behind the moving column of blood creates a sharp decrease in the aortic pressure tracing, which is called the diacrotic notch. The sudden closure of the aortic and pulmonary valves produces the second heart sound.

### **Isovolumic Relaxation:**

L.V ejection terminates with closure of the aortic valve. From the time of aortic valve closure until mitral valve opening, the L.V. is normally a closed chamber with little alteration in the volume. Myocardial relaxation, which begins in the latter part of systole, continues causing a steep fall in intraventricular pressure. Isovolumic relaxation ends when the L.V. pressure decreases below the left atrial pressure, the mitral valve opens, and rapid filling of the ventricle begins. Although no filling occurs during isovolumic relaxation, the processes that determine the rate of decline of the isovolumic pressure may influence ventricular filling following the mitral valve opening <sup>(1)</sup>.

### **Rapid filling phase:**

When L.V. pressure falls below left atrial pressure, the mitral valve opens. L.V. pressure continues to fall, due to myocardial relaxation and elastic recoil. This results in the development of a pressure gradient to between the left atrium and L.V. the magnitude of this pressure gradient in early diastole is determined primarily by the level of the left atrial pressure at the time of mitral valve opening and the rate of decline of the L.V. pressure <sup>(2)</sup>

In early diastole, the left atrium to L.V. pressure gradient produces a rapid flow of blood from the left atrium across the mitral valve. L.V. volume increases rapidly with 60% to 80% of the first stroke volume entering the L.V. during the first third of diastole. Much of this increase in L.V. volume occurs while the L.V. pressure continues to decrease. If mitral valve flow is prevented experimentally, the L.V. pressure will decrease to a subatmospheric level. Thus, the L.V. will fill, even when

the left atrial pressure is zero <sup>(1)</sup>. This ventricular suction may be due to the recoil of elastic elements that are compressed during systole. Early filling ends when the left atrial and L.V. pressures equilibrate. This occurs when L.V. relaxation nears completion and the flow of blood from the left atrium fills the L.V. raising the L.V. pressure while lowering the left atrial pressure <sup>(3)</sup>.

### **Diastasis:**

In the absence of tachycardia, rapid filling ends after approximately one third of diastole. Since the pressure in the left atrium and L.V. are nearly equilibrated, L.V. filling is slow during the mid portion of diastole (termed diastases). Only the blood returning from the lungs flows through the left atrium into the L.V.

### **Atrial systole:**

Atrial contraction increases atrial pressure. This produces a left atrial to L.V. pressure gradient that propels blood into the L.V. Atrial contraction and the configuration of the venous-atrial junction normally minimize retrograde flow into the pulmonary veins during atrial contraction. Atrial contraction can augment L.V. filling, increasing the L.V. end-diastolic pressure and volume, while only producing a small increase in the mean pulmonary venous pressure, this occurs because left atrial pressure is only transiently increased during atrial systole <sup>(2)</sup>.

Thus a properly timed vigorous atrial contraction can serve as a booster pump of the L.V. maximizing L.V. filling and protecting the lungs from an elevation of L.V. and diastolic pressure. Normally this atrial booster pump is not necessary since most of L.V. filling occurs

early in diastole, and only 25% or less of L.V. stroke volume enters the ventricle during atrial systole. Therefore, the absence of effective atrial contraction in patients who have cardiac output at rest, if the heart rate is slow, however at rapid heart rates during exercise, following standing, or when L.V. diastolic properties are depressed by myocardial infarction or other conditions the atrial booster pump reserve is used to maintain diastolic filling at a normal mean pulmonary venous pressure <sup>(4)</sup>. Following atrial systole, left atrial pressure may decrease below L.V. pressure, causing the mitral valve to begin closing. The beginning of ventricular systole produces a rapid increase in L.V. pressure that seals the mitral valve and end diastole.

## **Determinates of L.V. Diastolic performance**

### **Myocardial Relaxation:**

Active myocardial relaxation starts before aortic valve closure and lasts throughout early diastolic filling it is the major process that occurs during isovolumic relaxation and influences the rate of early L.V. filling. Myocardial relaxation is usually complete or almost complete by the middle of diastole and does not normally influence diastases or atrial filling pathologic processes such as ischaemia or hypertrophy may slow relaxation in some cases causing contractile activity to persist throughout diastole myocardial relaxation results from the dissociation of actin-myosin cross bridges and the recoil of elastic structures that were compressed during systole. The formation of the cross bridge during systole is an active process. Similarly, the reversal of tension generation is also an energy requiring process, in which calcium is actively taken up by the sarcoplasmic reticulum, the interaction between actin and myosin <sup>(1)</sup> the rate of myocardial relaxation can be influenced by the inotropic

state of the ventricle, the load the ventricle faced during the preceding ejection, heart rate and the synchrony of L.V. contraction and relaxation. Increasing the heart rate accelerates myocardial relaxation. Increased aortic pressure reduce active myocardial relaxation <sup>(1)</sup>. Inotropic agents or stimuli have a variable response. Catecholamines accelerate active myocardial relaxation, whereas increased extra cellular calcium ions, digitalis, and paired stimulation have little effect disease states also can affect active myocardial relaxation. Ischemia slows relaxation, intracellular calcium overload also slows relaxation in some patients who have L.V. hypertrophy <sup>(5)</sup>.

## II) Passive filling characteristics of the L.V. diastolic pressure-volume relaxation:

After the decay of systolic tension is complete, diastolic filling of the L.V. is influenced predominantly by the passive filling characteristics of the L.V. these characteristics are determined by the properties of the myocardium, the configuration and thickness of the L.V visco elastic effects, and external constraints four factors can be quantified as the passive L.V. diastolic pressure volume relation. Optimally, this curve should be constructed from points obtained after relaxation is complete and L.V. filling is slow, so that it represents only the passive characteristics of the L.V. The slope of the pressure-volume curve is the chamber stiffness. The normal passive pressure-volume relation of the L.V. is exponential in shape, therefore, the chamber stiffness is not a constant, but increases becomes stiffer at higher diastolic pressures <sup>(6)</sup> the position of the pressure-volume curve may be more important than the slope in determining the passive diastolic properties of the L.V. A shift upward and to the left, regardless of the chamber stiffness, indicates that a higher pressure is necessary to fill the L.V. to the same volume if the