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Understanding Diastolic Heart Failure

José F. Guadalajara Boo*

National Institute of Cardiology Ignacio Chavez, Mexico City.

***Correspondence:**
José F. Guadalajara Boo, National Institute of Cardiology Ignacio Chavez, Mexico City.

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ABSTRACT

This paper analyzes the concepts of systolic function, diastolic function, heart failure, diastolic dysfunction, and diastolic heart failure. Reference is made to the historical evolution of the concept of heart failure and the origin of the term diastolic heart failure.

In light of current knowledge of the physiology of the heart and its pathophysiology, the inappropriate terms for heart failure with preserved systolic function (is there heart failure? or is ventricular function normal?).

The clinical picture of pulmonary and systemic venous congestion in patients with obliteration of the cardiac chambers by endocardial thrombi or cardiac tamponade and finally pulmonary edema, caused by tight mitral stenosis, are they in diastolic heart failure? or do they have heart failure with preserved systolic function? This confusion has originated in clinical practice, since numerous research papers have called it "diastolic heart failure". It is concluded that there is a growing and increasingly urgent need to clearly identify the concepts of heart failure, diastolic dysfunction or diastolic heart failure.

This paper attempts to conceptualize the aforementioned concepts by analyzing the pathology, pathophysiology, clinical picture, prognosis, and treatment in an attempt to conceptualize the clinical picture of the entities that can lead to heart failure as opposed to the diastolic dysfunction and diastolic heart failure.

Keywords
Systolic function, Diastolic function, Heart failure.

Introduction
In the year of 2001 Colonel R, Groot JR and Van Lieshout JJ. Cardiovasc Research [1] consulted 2,238 experts to define the concept of "heart failure", of which only 1,018 responded with 130 different definitions, which were based: on the symptoms or described the pathophysiology, or according to the etiology and others that did not give a concrete concept; Therefore, Coronel R. et al. They concluded that: “heart failure is a cardiovascular syndrome that does not have a uniform definition criteria” [1].

For this reason, in 2006, an analysis of all the works published in the 20th century in relation to the review of the pathophysiology, clinical picture, treatment and prognosis of heart failure was carried out and it was found that great researchers had provided all the elements to be able to construct a definition of heart failure, in which the definition **“It is a condition in which diffuse structural myofibrils damage (inflammation, hibernation, necrosis, apoptosis, toxic effect), or excessive hemodynamic overload, they cause a decrease in the contractile force of the heart (hence the ejection fraction) and consequently increase ventricular volumes with normal cardiac output (compensated) or decrease cardiac output with tissue hypoperfusion (decompensated)”**. Managed to clarify a pathophysiological concept of the definition of heart failure [2].

On the other hand, the normal diastolic function of the heart is to receive blood, it distends the myocardium to prepare for its expulsion and this relaxation depends especially on myocardial diastolic stress, which can be defined as the force that tends to separate the myofibrils from each other per cm2 according to with Laplace's Law [3], thus, that diastolic stress constitutes the preload of the intact heart and is directly influenced by the speed of myocardial relaxation [4,5], in fact, isovolumic relaxation

significantly influences ventricular filling and when this part of the cardiac cycle is incomplete or slow, as can happen in acute myocardial ischemia or inappropriate hypertrophy [5,6].

The ventricular distensibility is very important myocardial property since. Compliance can be defined as Δ of volume divided by Δ of pressure, when the ventricle is distensible, the filling volume increases, in a concordant way for the physiology of the heart; in contrast, the rigid ventricle, with same filling volume increases inappropriately the intraventricular pressure and it is becomes a pathological alteration of the heart [6].

In clinical terms, diastolic dysfunction can be seen in patients with acute myocardial infarction, in which it can be so severe as to reach acute pulmonary edema due an incomplete isovolumic relaxation (Figure 1 A - B).

In this patient with an acute myocardial infarction associated with pulmonary edema, in the current classifications he would have a very high mortality, however, as a functional alteration in isovolumic relaxation was recognized taking into account that the heart function is normal (Figure 1 – C), it was started anti-ischemic treatment, which resolved the severe acute symptoms, with low mortality.

On the other hand, when the diastolic function is altered due to intrinsic damage as occurs in acute transmural miocardial infarction with high diastolic pressure has higher mortality [7].

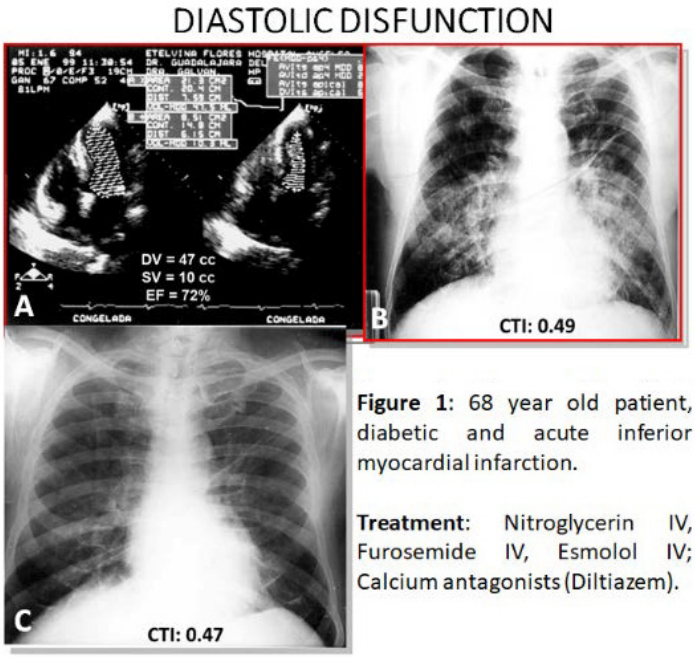

Figure 1: 68 year old patient, diabetic and acute inferior myocardial infarction.

Treatment: Nitroglycerin IV, Furosemide IV, Esmolol IV; Calcium antagonists (Diltiazem).

Restrictive cardiomyopathy

This group of diseases is conceptualized when endocardial thrombi obliterate the cardiac cavities and are part of a condition that obliterates diastolic function (diastolic dysfunction). It is when endocardial thrombi obliterate one or both cardiac cavities, as occurs in African endomyocardial fibrosis [8] in these cases the Symmetric obliteration produces a picture identical to that of congestive heart failure; when only left ventricle is obliterated, the picture similar to that of mitral diseases with disnea, pulmonary hypertension and right ventricular failure, when the right ventricle is obliterated, the picture simulates Ebstein's disease (Big cardiac enlargement, with pulmonary hiperclarity). This form of diastolic dysfunction must be treated surgically with an approximate mortality rate of 17% [9], so if it is not recognized clinically, it necessarily leads to death in 100% of cases. This form of diastolic dysfunction cannot be conceptualized as primary diastolic heart failure.

Diastolic Heart Failure

Ramachandran S. Vasan y Daniel Levy, diastolic heart failure was defined as patients with symptoms suggestive of heart failure who had an ejection fraction >50% [10].

Case I: An 83 year old female patient with progressive exertion dyspnea, edema and 4 months after progressed to dyspnea on minor exertion, orthopnea, and paroxysmal nocturnal dyspnea accompanied by progressive edema of the lower limbs up to anasarca with functional class III-IV, physical examination reveals rhythmic heart sounds and IV sound on auscultation, with a BP of 105/60 mmHg.

Chest X-ray PA shows cardiac enlargement (CTI: 0.58) with signs of pulmonary venocapillary hypertension in the lung fields (Figure 2A). Electrocardiogram: Sinus rhythm: 90 x min., P wave 0.22 mm; PR space 0.28, QT 0.01 sec.; âP + 60°, âQRS + 30°, Sokolof 29 mm, left atrial enlargement, 1st degree AV block, right ventricular dilation (Figure 2-B).

Figure 2: Diastolic Heart Failure (I).

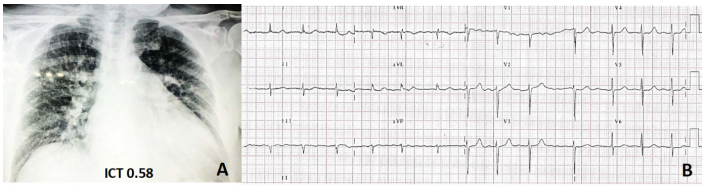

Figure 3-A demonstrates a very intense RESTRICTIVE PATTERN that contrasts with a dp/dt (3-B) and also high EF (3-C) that contrasts with the dilation of the IVC. Figure 3-D signs of DIASTOLIC HEART FAILURE.

The transmitral diastolic flow shows a restrictive pattern E/A ratio is 3.5, deceleration 130 mm/sec.; Likewise, the E/E' ratio 13.25 and the TAPSE at 22 mm and the inferior vena cava shows dilation without inspiratory collapse. Calculation of the pulmonary systolic pressure, through the tricuspid regurgitant flow 50 mmHg.

Case II: A 22-year-old male patient who arrived with amiloidosis and congestive heart failure with fatigue, on exertion dyspnea, jugular plethora, and ascending lower limb edema. The diagnosis was established with a 2D echo and myocardial scan [11] (Figure

4- A – B).

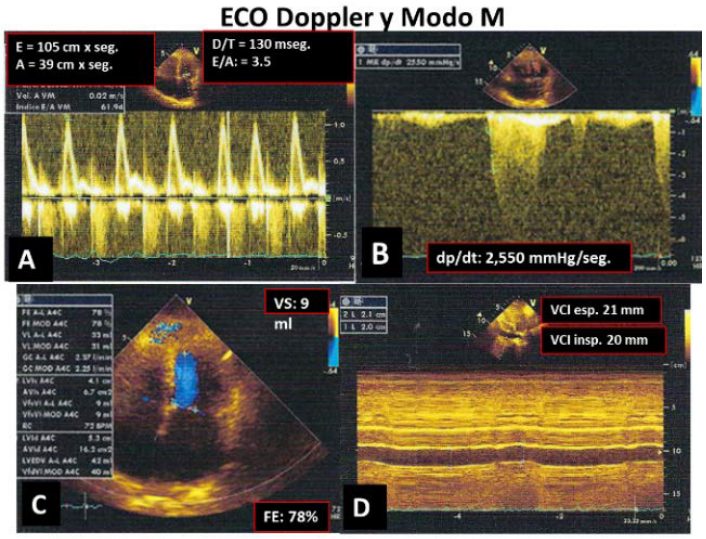

Figure 3: A) Pulsed Doppler, transmitral flow demonstrating a severely restrictive pattern with an E/A ratio of 3.5 and a very rapid deceleration of 130 msec. B) Continuous Doppler of 2,550 mmHg/sec. C) EF of 78%. D) Inferior vena cava expiration and inspiration demonstrates the presence of capillary and systemic venous hypertension due to extreme diastolic heart failure.

Simultaneous biventricular cardiac catheterization with high diastolic pressures with slow relaxation (Figure 4-C).

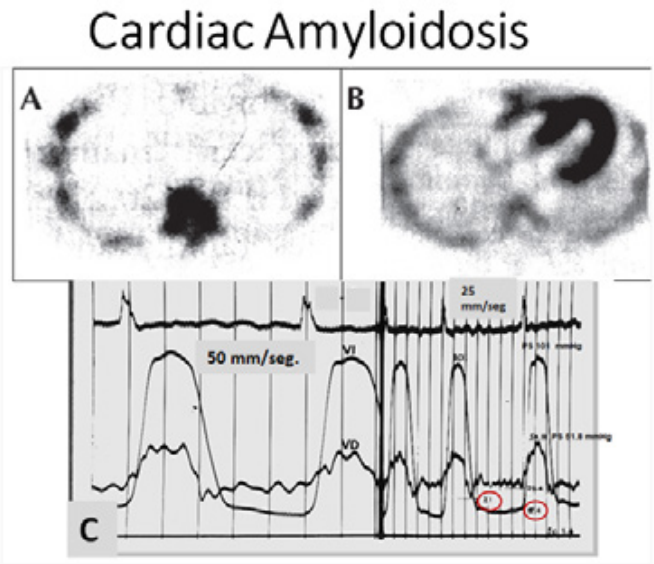

Figure 4: Cardiac scintigram with 99MTC- DPD. A) Negative cardiac scintigram (absence of radioisotope uptake). B) Cardiac scintigram that captures the radioisotope and demonstrates the diagnosis of cardiac amyloidosis. C) In cases of amyloidosis, the ventricular filling pattern rarely shows elevated filling pressures with a slow relaxation pattern.

Case III: A 60 year old female patient with a 6 month history of fatigue followed by progressive dyspnea until resting, jugular plethora, progressive lower limb edema, ascites and anasarca, functional class III-IV. With M mode Echo small ventricular cavites but the fractional shortening was very low. During the initial ventricular filling, the opening of the mitral valve (E/E') but the final opening of the valve was very brief (figure 5-A); that means that the early intraventricular pressure is low and the final pressure is very high that corresponds to diastolic pressures with morfology of dip and plateau (Figure 6-C).

To the 2D echo in 4 chambers view reveals small ventricles and the atrias very dilated (Figure 5B), and this conditions that in diastole the blood returns from the ventricle to the atrium. (Diastolic mitral regurgitation) (Figure 6A-B) [12] and that in systole there is no valvular regurgitation and this is a consequence of the enormous elevation of left ventricular diastolic pressure that in the pressure curves demonstrates the image of dip and plateau the recording of intraventricular pressures show an image of "square root" at different levels (Figure 6C) and that in this case the anatomical and clinical hemodynamic picture is caused by massive interstitial myocardial fibrosis (Figure 7).

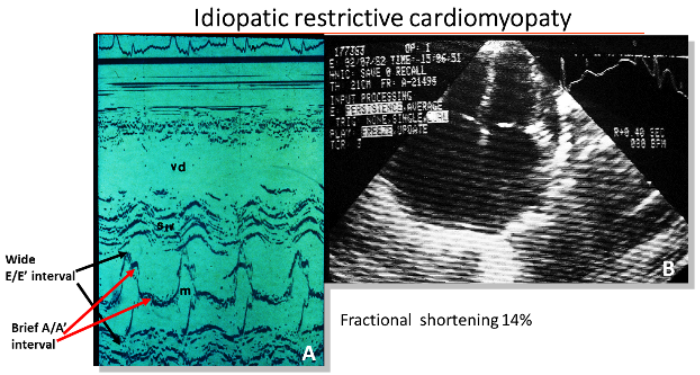

Figura 5: A) The interval behavior E/E' and A/A' translate D1 wide and D2 the same short interval = Pressure 1< and Pressure 2 > +++. **B)** The small ventricular cavities with great dilation of both atria with a clinical picture of capillary and systemic venous hypertension are shown.

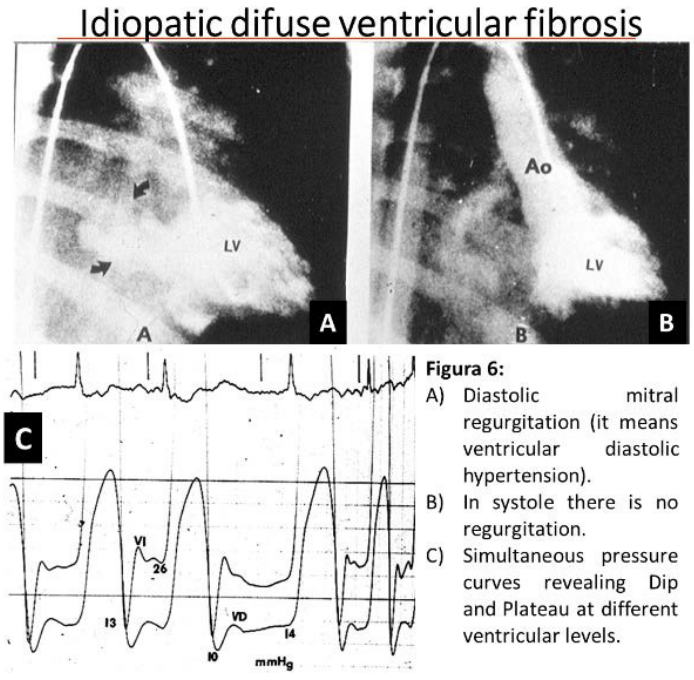

Figura 6:
A) Diastolic mitral regurgitation (it means ventricular diastolic hypertension).
B) In systole there is no regurgitation.
C) Simultaneous pressure curves revealing Dip and Plateau at different ventricular levels.

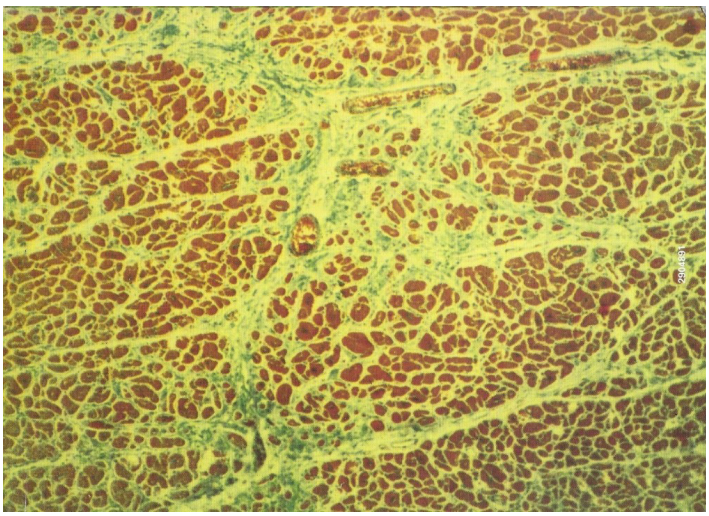

Figure 7: Diffuse Interstitial Fibrosis.

Diastolic Heart Failure

The systolic function of the heart has different characteristics than the diastolic function [2]; Indeed, there are conditions that can alter diastolic function in a reversible manner, which is why it can be treated with pharmacological measures that correct the functional alterations, as happens when ventricular isovolumic relaxation is altered [6] (Figure 1). Ventricular compliance can almost always be altered due to pathological conditions with higher forms to correct, which causes diastolic dysfunction that can have clinical consequences [7]. When compliance is corrected by irreversible pathological factors, it is almost always the cause of the physiological impossibility of maintaining its function and in these cases diastolic mitral regurgitation appears (Figure 6-A) [12].

Indeed, there are occasions when diastolic function may be altered not due to physiological or pathophysiological conditions, but rather, extreme ventricular stiffness does not allow systolic function to be insufficient to maintain adequate cardiac output and thus, we find that systolic function that is normal or even excessive (dp/dt 2550 and ejection fraction 78%) (Figure 3 – B, C) do not achieve cardiac output reaching sufficient tissue perfusion to maintain the hemodynamic needs of the heart that the patient needs for life, **under these conditions, with fair pathophysiological reason should be called diastolic heart failure;** in the cases that we exemplify in which idiopathic diffuse interstitial fibrosis or fibrosis of aging or in younger people of idiopathic etiology, lead to the patient with altered heart failure, due to extreme myocardial rigidity: thus, in the elderly we find that heart failure cardiac arrest despite the fact that normal mechanisms that increase the inotropic force of the heart can lead to death.

Recently Popovic D, et al. studied 621 HFpEF patients and found that the greater the patients' ventricular stiffness (pressure/volume), the greater their EF, which confirms that ventricular stiffness stimulates compensation mechanisms to restore ventricular function and this is what ultimately influences directly for the appearance of diastolic heart failure [13].

It has been found that the combination of gliflozin and spironolactone can improve the restrictive process, but when fibrosis, is extreme (like case III), this may not be enough, so in these cases prospective studies are required with these treatments to determine their efficacy when diffuse interstitial fibrosis is present it is extreme (Figure7).

In this paper we analyze the conditions that can cause reversible or medically or surgically treatable diastolic dysfunction; in fact, there are occasions in which diastolic function may be altered not by physiological or pathophysiological conditions, but rather by extreme ventricular stiffness that does not allow sufficient systolic function to maintain adequate cardiac output, and thus, despite the fact that diastolic function systolic increases to the maximum it is not possible to achieve a cardiac output that is sufficient to maintain the normal physiology of the heart, and in these cases the diastolic alteration is the cause of the disease and is what should be called DIASTOLIC HEART FAILURE.

We first analyze patients who have reversible diastolic dysfunction and by medically treating patients we can bring them back to normal heart function (Figure 1).

The other group is in those cases in which the diastolic function is impeded by endocardial thrombi and even when the intrinsic function of the heart is normal, they cause severe symptoms, but in these cases when ventricular obliteration is treated, improvement and life can be achieved of the patients, and finally when the patient falls into heart failure and despite increasing the systolic capacity to the maximum it is not possible to achieve a sufficient cardiac output for these patients and it is in these cases that true diastolic heart failure appears. In the first case that we present, the patient has a systolic function that is normal and even excessive (dp/dt 2,550 and ejection fraction 78%), they do not achieve a cardiac output reaching sufficient tissue perfusion to maintain the hemodynamic needs of the heart that the patient it needs for life, under these conditions, with just pathophysiological reason it should be called diastolic heart failure.

In the second case in which the patient suffers from amyloidosis, he falls into heart failure with a normal ejection fraction with a very important alteration in ventricular relaxation that is excessively slow and the diastolic pressures are high and finally the third case that we show shows Extreme ventricular stiffness that is caused by excessive myocardial interstitial fibrosis and diastolic heart failure led to the death of this patient.

In these cases that we exemplify in which idiopathic diffuse interstitial fibrosis or aging fibrosis or in younger people with amyloidosis, lead the patient to altered heart failure, due to extreme myocardial rigidity: thus, in the elderly we find. It has been found that the combination of gliflozin and spironolactone can improve the restrictive process, but when the fibrosis is extreme as the one shown in Figure 7, therefore, in these cases, prospective studies are required with new therapeutic schemes that could reduce or improve diffuse interstitial fibrosis, which for now when it appears is the irreversible cause of diastolic heart failure.

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